01 - motivation -
everything grows!

… what we do …

kinematic equations for finite growth

$$\boldsymbol{F} = \boldsymbol{F}_{\rm e} \cdot \boldsymbol{F}_{\rm g}$$

balance equations for open systems

$$\begin{aligned} \mathrm{D}_t \rho_0 &= \mathrm{Div}(\boldsymbol{R}) + \mathcal{R}_0 \\ \rho_0 \, \mathrm{D}_t \boldsymbol{v} &= \mathrm{Div}(\boldsymbol{P}) + \boldsymbol{b}_0 \end{aligned}$$

constitutive equations for living tissues

$$\boldsymbol{P} = \boldsymbol{P}(\rho_0, \boldsymbol{F}, \boldsymbol{F}_{\rm g})$$

fe analyses for biological structures

continuum- & computational biomechanics

… why we do what we do …

kinematic equations for finite growth

$$\boldsymbol{F} = \boldsymbol{F}_{\rm e} \cdot \boldsymbol{F}_{\rm g}$$

balance equations for open systems

$$\begin{aligned} \mathrm{D}_t \rho_0 &= \mathrm{Div}(\boldsymbol{R}) + \mathcal{R}_0 \\ \rho_0 \, \mathrm{D}_t \boldsymbol{v} &= \mathrm{Div}(\boldsymbol{P}) + \boldsymbol{b}_0 \end{aligned}$$

constitutive equations for living tissues

$$\boldsymbol{P} = \boldsymbol{P}(\rho_0, \boldsymbol{F}, \boldsymbol{F}_{\rm g})$$

fe analyses for biological structures

… because biological structures are …

… why we do what we do …

highly deformable

kinematic equations for finite growth

$$\boldsymbol{F} = \boldsymbol{F}_{\rm e} \cdot \boldsymbol{F}_{\rm g}$$

balance equations for open systems

$$\begin{aligned} \mathrm{D}_t \rho_0 &= \mathrm{Div}(\boldsymbol{R}) + \mathcal{R}_0 \\ \rho_0 \, \mathrm{D}_t \boldsymbol{v} &= \mathrm{Div}(\boldsymbol{P}) + \boldsymbol{b}_0 \end{aligned}$$

constitutive equations for living tissues

$$\boldsymbol{P} = \boldsymbol{P}(\rho_0, \boldsymbol{F}, \boldsymbol{F}_{\rm g})$$

fe analyses for biological structures

… because biological structures are …

… why we do what we do …

kinematic equations for finite growth

$$\boldsymbol{F} = \boldsymbol{F}_{\mathrm{e}} \cdot \boldsymbol{F}_{\mathrm{g}}$$

balance equations for open systems

$$\mathrm{D}_t \rho_0 = \mathrm{Div}(\boldsymbol{R}) + \mathcal{R}_0$$
$$\rho_0 \, \mathrm{D}_t \boldsymbol{v} = \mathrm{Div}(\boldsymbol{P}) + \boldsymbol{b}_0$$

constitutive equations for living tissues

$$\boldsymbol{P} = \boldsymbol{P}(\rho_0, \boldsymbol{F}, \boldsymbol{F}_{\mathrm{g}})$$

fe analyses for biological structures

… because biological structures are …

highly deformable

living

… why we do what we do …

kinematic equations for finite growth

$$\boldsymbol{F} = \boldsymbol{F}_{\mathrm{e}} \cdot \boldsymbol{F}_{\mathrm{g}}$$

balance equations for open systems

$$\mathrm{D}_t \rho_0 = \mathrm{Div}(\boldsymbol{R}) + \mathcal{R}_0$$
$$\rho_0 \, \mathrm{D}_t \boldsymbol{v} = \mathrm{Div}(\boldsymbol{P}) + \boldsymbol{b}_0$$

constitutive equations for living tissues

$$\boldsymbol{P} = \boldsymbol{P}(\rho_0, \boldsymbol{F}, \boldsymbol{F}_{\mathrm{g}})$$

fe analyses for biological structures

… because biological structures are …

highly deformable

living

nonlinear

… why we do what we do …

kinematic equations for finite growth

$$\boldsymbol{F} = \boldsymbol{F}_{\mathrm{e}} \cdot \boldsymbol{F}_{\mathrm{g}}$$

balance equations for open systems

$$\mathrm{D}_t \rho_0 = \mathrm{Div}(\boldsymbol{R}) + \mathcal{R}_0$$
$$\rho_0 \, \mathrm{D}_t \boldsymbol{v} = \mathrm{Div}(\boldsymbol{P}) + \boldsymbol{b}_0$$

constitutive equations for living tissues

$$\boldsymbol{P} = \boldsymbol{P}(\rho_0, \boldsymbol{F}, \boldsymbol{F}_{\mathrm{g}})$$

fe analyses for biological structures

… because biological structures are …

highly deformable

living

nonlinear

inelastic

… why we do what we do …

kinematic equations for finite growth

$$\boldsymbol{F} = \boldsymbol{F}_{\mathrm{e}} \cdot \boldsymbol{F}_{\mathrm{g}}$$

balance equations for open systems

$$\mathrm{D}_t \rho_0 = \mathrm{Div}(\boldsymbol{R}) + \mathcal{R}_0$$
$$\rho_0 \, \mathrm{D}_t \boldsymbol{v} = \mathrm{Div}(\boldsymbol{P}) + \boldsymbol{b}_0$$

constitutive equations for living tissues

$$\boldsymbol{P} = \boldsymbol{P}(\rho_0, \boldsymbol{F}, \boldsymbol{F}_{\mathrm{g}})$$

fe analyses for biological structures

… because biological structures are …

highly deformable

living

nonlinear

inelastic

anisotropic

… why we do what we do …

- kinematic equations for finite growth

$$\boldsymbol{F} = \boldsymbol{F}_{\mathrm{e}} \cdot \boldsymbol{F}_{\mathrm{g}}$$

- balance equations for open systems

$$\mathrm{D}_t \rho_0 = \mathrm{Div}(\boldsymbol{R}) + \mathcal{R}_0$$

$$\rho_0 \, \mathrm{D}_t \boldsymbol{v} = \ldots \boldsymbol{b}_0$$

- constitutive equations

$$\boldsymbol{P} = \boldsymbol{P}(\rho_0, \boldsymbol{F}, \ldots)$$

- fe analyses for biological structures

highly deformable

living

anisotropic

nonlinear

inelastic

inhomo-geneous

… because biological structures are …

me337 - goals

in contrast to traditional engineering structures living structures show the fascinating ability to **grow and adapt their form, shape and microstructure** to a given mechanical environment. this course addresses the phenomenon of growth on a theoretical and computational level and applies the resulting theories to classical biomechanical problems like bone remodeling, hip replacement, wound healing, atherosclerosis or in stent restenosis. this course will illustrate how classical engineering concepts like continuum mechanics, thermodynamics or finite element modeling have to be rephrased in the context of growth. having attended this course, you will be able to develop your own problemspecific finite element based numerical solution techniques and interpret the results of biomechanical simulations with the ultimate goal of improving your **understanding of the complex interplay between form and function**

me 337 - syllabus 2007

day	date		topic
tue	sep	21	motivation - everything grows!
thu	sep	23	basics and maths - notation and tensors
tue	sep	28	class project '07 - growth of tennis player arms
thu	sep	30	guided reading - no class
tue	oct	05	basics and mechanics - kinematics and balance equations of growth
thu	oct	07	guided reading - no class
tue	oct	12	density growth - growing bones
thu	oct	14	density growth - finite elements for growth / theory
tue	oct	19	density growth - finite elements for growth / matlab
thu	oct	21	density growth - growing bones
tue	oct	26	density growth - finite elements for growth
thu	oct	28	midterm
tue	nov	02	volume growth - growing tumors
thu	nov	04	volume growth - finite elements for growth / theory
tue	nov	09	volume growth - finite elements for growth / matlab
thu	nov	11	volume growth - growing arteries
tue	nov	16	volume growth - growing hearts
thu	nov	18	remodeling - remodeling arteries and tendons
tue	nov	30	class project - discussion, presentation, evaluation
thu	dec	02	class project - discussion, presentation, evaluation
thu	dec	02	written part of final projects due

me 337 - syllabus 2012

day	date		topic
tue	jan	10	motivation - everything grows!
thu	jan	12	basics maths - notation and tensors
tue	jan	17	basic kinematics - large deformation and growth
thu	jan	19	basic kinematics - large deformation and growth
tue	jan	24	guest lecture: class project
thu	jan	26	guest lecture: growing leaflets
tue	jan	31	basic balance equations - closed and open systems
thu	feb	02	basic constitutive equations - growing tumors
tue	feb	07	volume growth - finite elements for growth
thu	feb	09	volume growth - growing arteries
tue	feb	14	volume growth - growing skin
thu	feb	16	volume growth - growing hearts
tue	feb	21	basic constitutive equations - growing bones
thu	feb	23	density growth - finite elements for growth
tue	feb	28	density growth - growing bones
thu	mar	01	everything grows! - midterm summary
tue	mar	06	midterm
thu	mar	08	remodeling - remodeling arteries and tendons
tue	mar	13	class project - discussion, presentation, evaluation
thu	mar	15	class project - discussion, presentation, evaluation
thu	mar	15	written part of final projects due

me 337 - grading

- 30 % homework - 3 homework assignments, 10% each
- 30 % midterm - closed book, closed notes, one single page cheat sheet
- 20 % final project oral presentations - graded by the class
- 20 % final project essay - graded by instructor

introduction

The Stanford Daily

An Independent Publication

If there's one thing that's guaranteed, it's that Jeff Zeller strives to apply what he knows in various contexts. Whether it be while injured, in doubles or in singles matches, the sophomore will take his wisdom and work to develop his game.

Although the Zeller redshirted in 2006 because of injury, he was still able to observe his teammates in action and apply this knowledge to his own game when he returned to the court.

This year, Zeller has used this experience, and along with senior Eric McKean, has formed a successful doubles pair that draws inspiration from the Bryan brothers (Bob and Mike Bryan — both former Stanford players).

Throughout this season, Zeller has learned to apply doubles tactics in order to improve his singles game, too. These circumstances demonstrate why head coach John Whitlinger will attest to the fact that the Centennial, Colo. native is such a "good student of the game."

Zeller injured his hand in January of 2006 and took three months off from tennis. Even when Zeller started hitting again in the spring, he was not at 100 percent and, therefore, could not practice with the team. In his first year on The Farm, Zeller was forced into the role of an onlooker and was not able to contribute to the team on the court.

Stanford Daily File Photo

Jeff Zeller / SO

"When I got injured, I took on more of the observer role, but I got to watch my teammates succeed," Zeller said. "I got to watch [sophomore] Matt [Bruch] get ranked top 5 in the country; I got to watch KC [Corkery] get to the semifinals of NCAAs; I got to watch KC and [then-senior] James Pade play some amazing doubles. I think I really learned a lot from just sitting back and seeing what my teammates did well that allowed them to be successful.

student project - tennis player

The phenomenon of twisted growth: humeral torsion in dominant arms of high performance tennis players

R.E. Taylor[a], C. Zheng[a], R.P. Jackson[b], J.C. Doll[a], J.C. Chen[b], K.R.S. Holzbaur[c], T. Besier[d] and E. Kuhl[ab]*

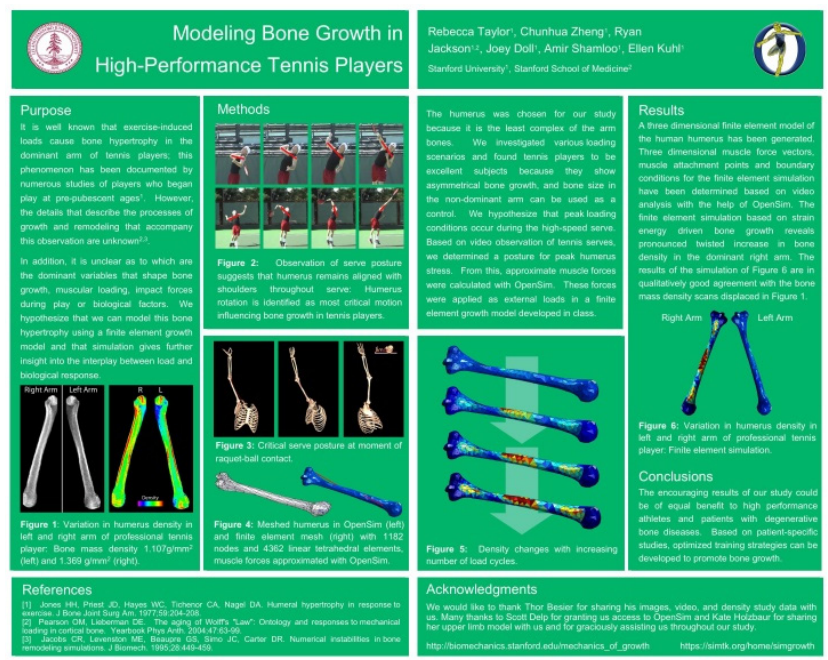
[a]Department of Mechanical Engineering, Stanford University, Stanford, CA, USA; [b]Department of Bioengineering, Stanford University, Stanford, CA, USA; [c]Department of Biomedical Engineering, Wake Forest University School of Medicine, Winston-Salem, NC, USA; [d]Department of Orthopaedic Surgery, Stanford University, Stanford, CA, USA

(Received 27 November 2007; final version received 2 May 2008)

This manuscript is driven by the need to understand the fundamental mechanisms that cause twisted bone growth and shoulder pain in high performance tennis players. Our ultimate goal is to predict bone mass density in the humerus through computational analysis. The underlying study spans a unique four level complete analysis consisting of a high-speed video analysis, a musculoskeletal analysis, a finite element based density growth analysis and an X-ray based bone mass density analysis. For high performance tennis players, critical loads are postulated to occur during the serve. From high-speed video analyses, the serve phases of maximum external shoulder rotation and ball impact are identified as most critical loading situations for the humerus. The corresponding posts from the video analysis are reproduced with a musculoskeletal analysis tool to determine muscle attachment points, muscle force vectors and overall forces of relevant muscle groups. Collective representative muscle forces of the deltoid, latissimus dorsi, pectoralis major and triceps are then applied as external loads in a fully 3D finite element analysis. A problem specific nonlinear finite element based density analysis tool is developed to predict functional adaptation over time. The density profiles in response to the identified critical muscle forces during serve are qualitatively compared to X-ray based bone mass density analyses.

Keywords: bone mass density changes; functional adaptation; musculoskeletal analysis; finite element analysis; sports medicine

student project - tennis player



Modeling Bone Growth in High-Performance Tennis Players

Rebecca Taylor[1], Chunhua Zheng[1], Ryan Jackson[1,2], Joey Doll[1], Amir Shamloo[1], Ellen Kuhl[1]

Stanford University[1], Stanford School of Medicine[2]

Purpose

It is well known that exercise-induced loads cause bone hypertrophy in the dominant arm of tennis players; this phenomenon has been documented by numerous studies of players who began play at pre-pubescent ages[1]. However, the details that describe the processes of growth and remodeling that accompany this observation are unknown[2,3].

In addition, it is unclear as to which are the dominant variables that shape bone growth, muscular loading, impact forces during play or biological factors. We hypothesize that we can model this bone hypertrophy using a finite element growth model and that simulation gives further insight into the interplay between load and biological response.

Right Arm Left Arm R L Density

Figure 1: Variation in humerus density in left and right arm of professional tennis player: Bone mass density 1.107g/mm² (left) and 1.369 g/mm² (right).

Methods

Figure 2: Observation of serve posture suggests that humerus remains aligned with shoulders throughout serve: Humerus rotation is identified as most critical motion influencing bone growth in tennis players.

Figure 3: Critical serve posture at moment of raquet-ball contact.

Figure 4: Meshed humerus in OpenSim (left) and finite element mesh (right) with 1182 nodes and 4362 linear tetrahedral elements, muscle forces approximated with OpenSim.

The humerus was chosen for our study because it is the least complex of the arm bones. We investigated various loading scenarios and found tennis players to be excellent subjects because they show asymmetrical bone growth, and bone size in the non-dominant arm can be used as a control. We hypothesize that peak loading conditions occur during the high-speed serve. Based on video observation of tennis serves, we determined a posture for peak humerus stress. From this, approximate muscle forces were calculated with OpenSim. These forces were applied as external loads in a finite element growth model developed in class.

Figure 5: Density changes with increasing number of load cycles.

Results

A three dimensional finite element model of the human humerus has been generated. Three dimensional muscle force vectors, muscle attachment points and boundary conditions for the finite element simulation have been determined based on video analysis with the help of OpenSim. The finite element simulation based on strain energy driven bone growth reveals pronounced twisted increase in bone density in the dominant right arm. The results of the simulation of Figure 6 are in qualitatively good agreement with the bone mass density scans displaced in Figure 1.

Right Arm Left Arm

Figure 6: Variation in humerus density in left and right arm of professional tennis player: Finite element simulation.

Conclusions

The encouraging results of our study could be of equal benefit to high performance athletes and patients with degenerative bone diseases. Based on patient-specific studies, optimized training strategies can be developed to promote bone growth.

References

[1] Jones HH, Priest JD, Hayes WC, Tichenor CA, Nagel DA. Humeral hypertrophy in response to exercise. J Bone Joint Surg Am. 1977;59:204-208.
[2] Pearson OM, Lieberman DE. The aging of Wolff's "Law": Ontology and responses to mechanical loading in cortical bone. Yearbook Phys Anth. 2004;47:63-99.
[3] Jacobs CR, Levenston ME, Beaupre GS, Simo JC, Carter DR. Numerical instabilities in bone remodeling simulations. J Biomech. 1995;28:449-459.

Acknowledgments

We would like to thank Thor Besier for sharing his images, video, and density study data with us. Many thanks to Scott Delp for granting us access to OpenSim and Kate Holzbaur for sharing her upper limb model with us and for graciously assisting us throughout our study.

http://biomechanics.stanford.edu/mechanics_of_growth https://simtk.org/home/simgrowth

Biomechan Model Mechanobiol (2007) 6:321–331
DOI 10.1007/s10237-006-0062-x

ORIGINAL PAPER

Computational modeling of arterial wall growth

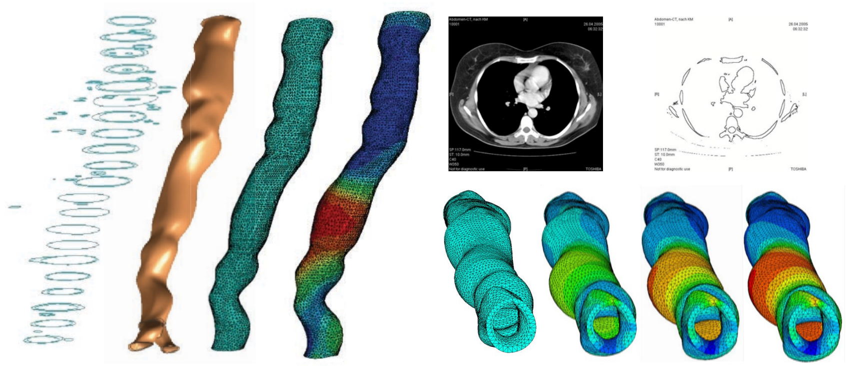
Attempts towards patient-specific simulations based on computer tomography

E. Kuhl · R. Maas · G. Himpel · A. Menzel

Received: 23 September 2005 / Accepted: 10 June 2006 / Published online: 22 November 2006

Abstract The present manuscript documents our first experiences with a computational model for stress-induced arterial wall growth and in-stent restenosis related to atherosclerosis. The underlying theoretical framework is provided by the kinematics of finite growth combined with open system thermodynamics. The computational simulation is embedded in a finite element approach in which growth is essentially captured by a single scalar-valued growth factor introduced as internal variable on the integration point level. The conceptual simplicity of the model enables its straightforward implementation into standard commercial finite element codes. Qualitative studies of stress-induced changes of the arterial wall thickness in response to balloon angioplasty or stenting are presented to illustrate the features of the suggested growth model. First attempts towards a patient-specific simulation based on realistic artery morphologies generated from computer tomography data are discussed.

patient specific virtual stent implantation

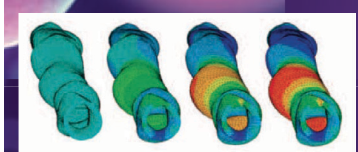


from ct to finite element model

On Simulating Growth And Form

For better or for worse, and on many levels, our tissues never stop growing and changing. While developing from childhood to old age, we grow not only bone, cartilage, fat, muscle and skin, but also toughened arteries, scars for our wounds, and, sometimes, deadly tumors.

"Computation is a great tool to study growth," says **Ellen Kuhl, PhD,** assistant professor of mechanical engineering and bioengineering at Stanford University, "because it lets us understand all those fascinating biological processes we can't otherwise see and predict."



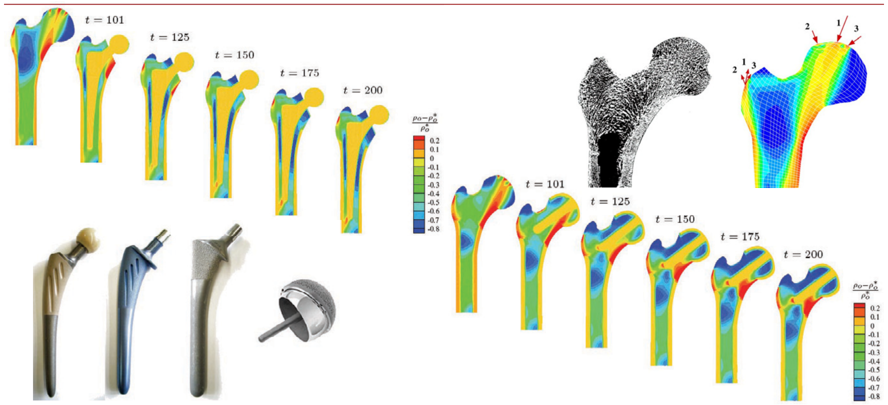
A simulated aorta, based on computer tomography data from a specific patient, highlights the effects of stent surgery (shown before surgery as well as 4 days, 16 days, and 33 days after surgery). Cyan shading shows artery walls with normal thickness; blue shading shows thinning walls; red shading show thickened walls. In a real patient, the tissue growth shown in red would have resulted in in-stent restenosis. Courtesy of Ellen Kuhl. Reprinted

Computational modeling of hip replacement surgery: Total hip replacement vs. hip resurfacing

E. Kuhl & F. Balle

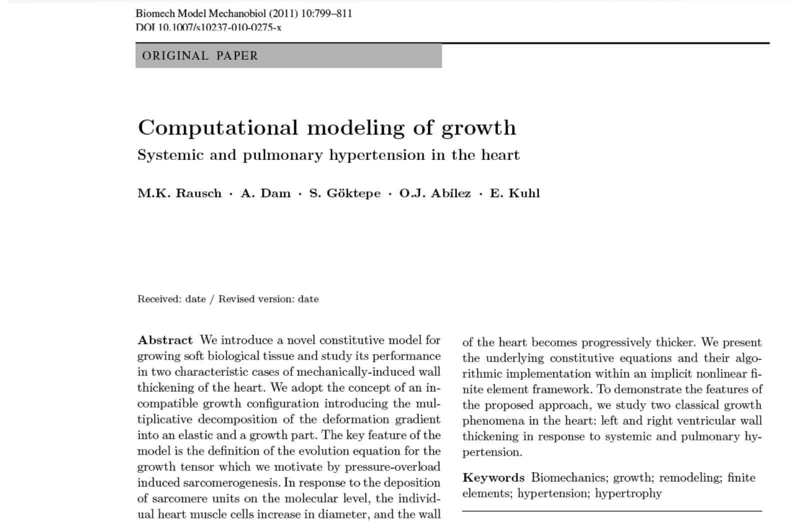
The motivation of the present work is the computational simulation of hip replacement surgery by means of a finite element approach based on open system thermodynamics. Its key feature is a non-constant material density, which is allowed to adapt with respect to changes in the mechanical loading environment. From a computational point of view, the density is treated as an internal variable. Its evolution is governed by a first order rate equation, the balance of mass, which is enhanced by an additional mass production term to account for growth. An implicit Euler backward scheme is suggested for its time discretization. The algorithmic determination of the material density based on a local Newton iteration is presented. To ensure quadratic convergence of the global Newton Raphson solution scheme, a consistent linearization of the discrete algorithmic equations is carried out. Finally, two alternative medical techniques in hip arthritis are compared, the conventional total hip replacement strategy and the more recent hip resurfacing technology. The result of the suggested remodeling algorithm is shown to agree remarkably well with clinically observed phenomena.

how do bones grow?



conventional hip replacement vs hip resurfacing

student project - hip replacement 22


Biomech Model Mechanobiol (2011) 10:799–811
DOI 10.1007/s10237-010-0275-x

ORIGINAL PAPER

Computational modeling of growth

Systemic and pulmonary hypertension in the heart

M.K. Rausch · A. Dam · S. Göktepe · O.J. Abilez · E. Kuhl

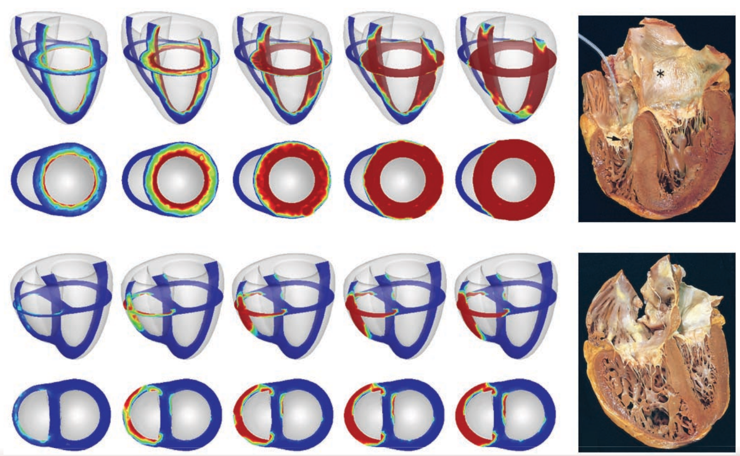
Received: date / Revised version: date

Abstract We introduce a novel constitutive model for growing soft biological tissue and study its performance in two characteristic cases of mechanically-induced wall thickening of the heart. We adopt the concept of an incompatible growth configuration introducing the multiplicative decomposition of the deformation gradient into an elastic and a growth part. The key feature of the model is the definition of the evolution equation for the growth tensor which we motivate by pressure-overload induced sarcomerogenesis. In response to the deposition of sarcomere units on the molecular level, the individual heart muscle cells increase in diameter, and the wall of the heart becomes progressively thicker. We present the underlying constitutive equations and their algorithmic implementation within an implicit nonlinear finite element framework. To demonstrate the features of the proposed approach, we study two classical growth phenomena in the heart: left and right ventricular wall thickening in response to systemic and pulmonary hypertension.

Keywords Biomechanics; growth; remodeling; finite elements; hypertension; hypertrophy

student project - hypertension 23

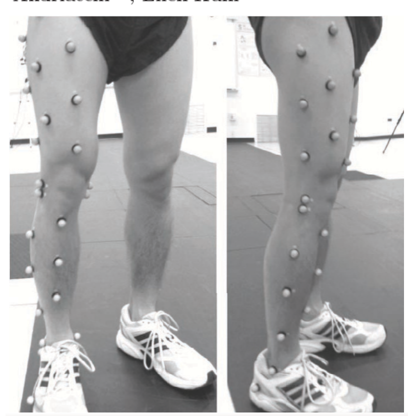
why does the heart wall get thicker?

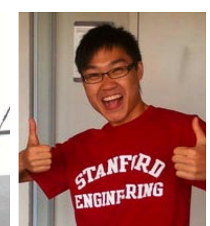


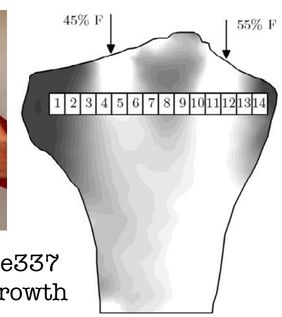
student project - hypertension 24

Computational modeling of bone density profiles in response to gait: A subject-specific approach

Henry Pang[1], Abhishek P. Shiwalkar[1], Chris M. Madormo[1], Rebecca E. Taylor[1], Thomas P. Andriacchi[1,2], Ellen Kuhl[1,3,4]





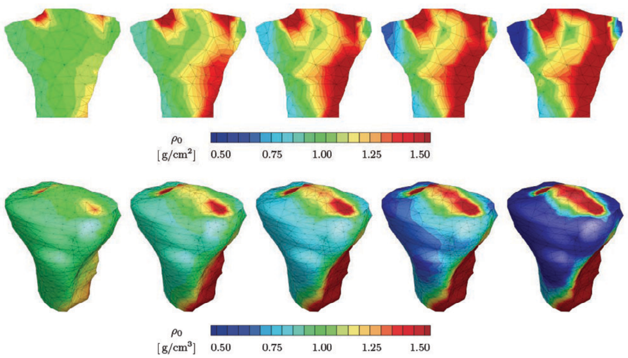


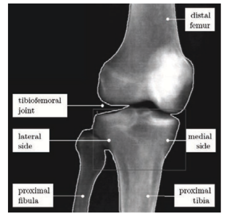
class project me337
mechanics of growth

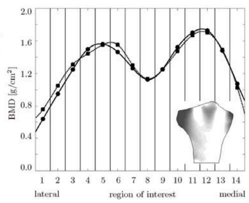
trial	I	II	III	mean
max knee force [N]	552	549	579	560
max knee force [% BW]	94.9	94.4	99.4	96.2

student project - henry's knee 25

how does henry's bone grow?







student project - henry's knee

Journal of the Mechanics and Physics of Solids 59 (2011) 2177–2190

Growing skin: A computational model for skin expansion in reconstructive surgery

Adrián Buganza Tepole [a], Christopher Joseph Ploch [a], Jonathan Wong [a], Arun K. Gosain [b], Ellen Kuhl [a,c,d,*]

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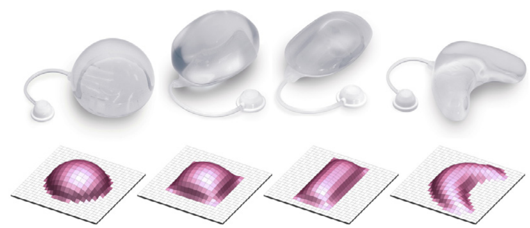
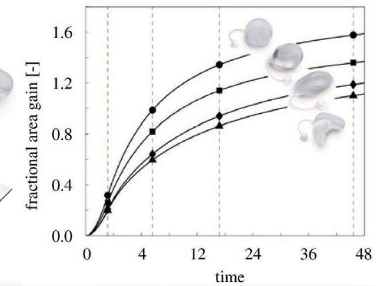
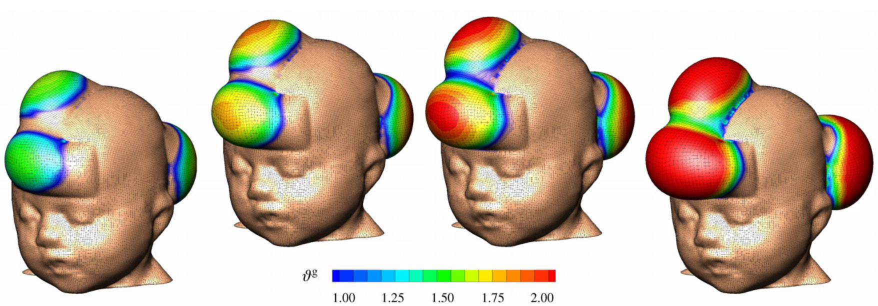
[b] Department of Plastic Surgery, Rainbow Babies and Children's Hospital, Case Western Reserve University, Cleveland, OH 44106, USA
[c] Department of Bioengineering, Stanford University, Stanford, CA 94305, USA
[d] Department of Cardiothoracic Surgery, Stanford University, Stanford, CA 94305, USA





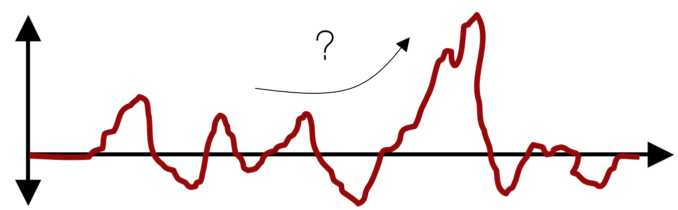
student project - skin growth

how does skin grow?



student project - skin growth

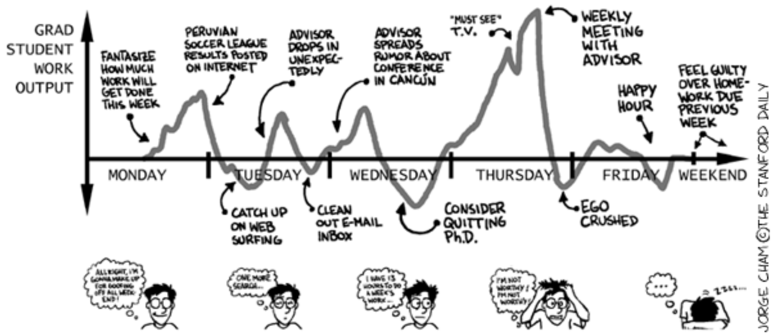
what's growing?



classical engineering materials are not!

introduction

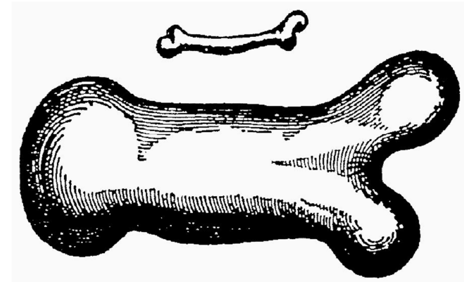
what's growing?



grad student work output

J. Cham "Piled higher and deeper", [1999]

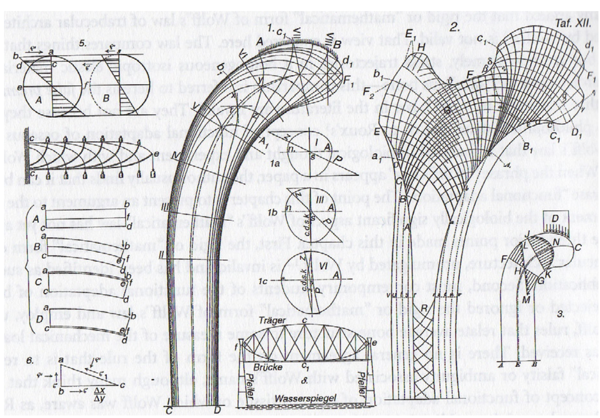
history - 17th century



„...dal che e manifesto, che chi volesse mantener in un vastissimo gigante le proporzioni, che hanno le membra in un huomo ordinario, bisognerebbe o trouar materia molto piu dura, e resistente per formarne l'ossa o vero ammettere, che la robustezza sua fusse a proporzione assai piu fiacca, che negli huomini de statura mediocre; altrimente crescendogli a smisurata altezza si vedrebbono dal proprio peso opprimere, e cadere..."

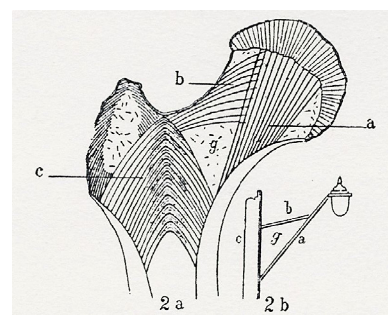
Galileo,"Discorsi e dimostrazioni matematiche", [1638]

history - 19th century



Culmann & von Meyer „Graphic statics" [1867]

history - 19th century



„...es ist demnach unter dem gesetze der transformation der knochen dasjenige gesetz zu verstehen, nach welchem im gefolge primaerer abaenderungen der form und inanspruchnahme bestimmte umwandlungen der inneren architectur und umwandlungen der aeusseren form sich vollziehen..."

Wolff „Gesetz der Transformation der Knochen" [1892]

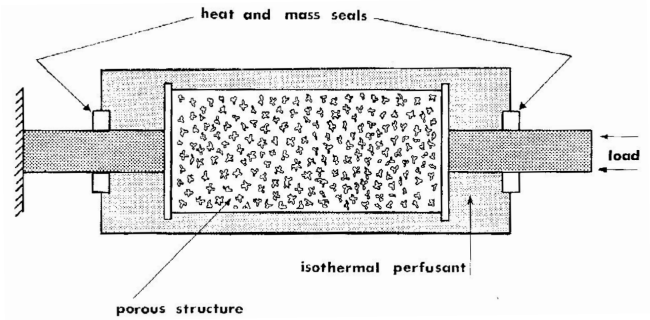
history - 19th century

carson pirie scott store
Sullivan[1904]

„…whether it be the sweeping eagle in his flight or the open apple-blossom, the toiling work-horse, the blithe swan, the branching oak, the winding stream at its base, the drifting clouds, over all the coursing sun, form ever follows function, and this is the law…"

Sullivan „Form follows function" [1896]

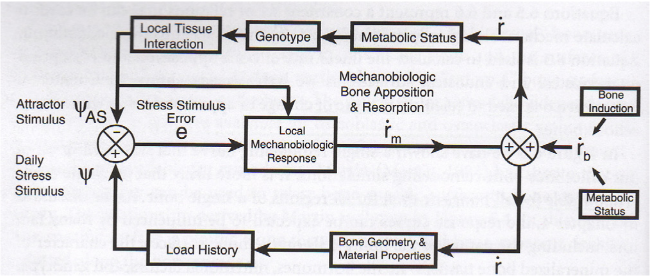
history - 20th century



„…the system consisting of only the porous structure without its entrained perfusant is open with respect to momentum transfer as well as mass, energy, and entropy transfer. we shall write balance and constitutive equations for only the bone…"

Cowin & Hegedus „Theory of adaptive elasticity" [1976]

history - 20th century



„…the relationship between physical forces and the morphology of living things has piqued the curiosity of every artist, scientist, or philosopher who has contemplated a tree or drawn the human figure. its importance was a concern of galileo and later thompson whose writings remind us that physical causation plays an inescapable role in the development of biological form…"

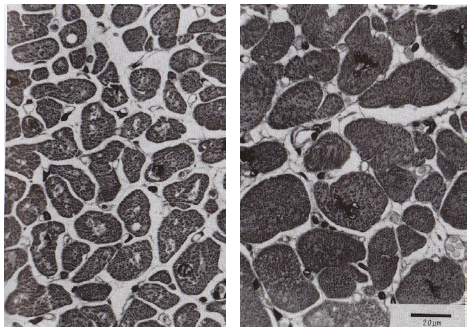
Beaupré, Carter & Orr „Theory of bone modeling & remodeling" [1990]

history - 20th century

„hypertrophy of the heart: comparison of cross sections of a normal heart (bottom), a heart chronically overloaded by an unusually large blood volume (left) and a heart chronically overloaded by an unusually large diastoic and systolic left ventricular pressure (right)"

Fung „Biomechanics - Motion, flow, stress, and growth" [1990]

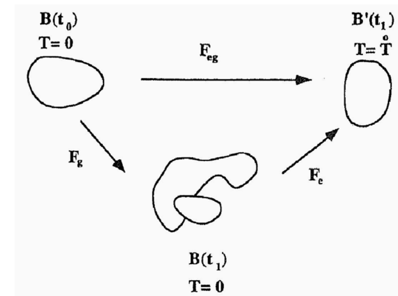
history - 20th century



„hypertrophy of the heart: histology of a normal heart (left) and pressure overloaded heart (right) photographed at the same magnification - muscles in the hypertropic heart (right) are much bigger in diameter than those of the normal heart (left)."

Fung „Biomechanics - Motion, flow, stress, and growth" [1990]

history - 21th century



Rodriguez, Holger & McCulloch [1994]

„…the process of growth can be seen as an evolution of material point neighbourhoods in a fixed reference configuration. the growth process will cause the development of material inhomogeneities responsible for residual stresses in the body…"

Epstein & Maugin „Theory of volumetric growth" [2000]

growth, remodeling and morphogenesis

growth [groʊθ] which is defined as added mass, can occur through cell division (hyperplasia), cell enlargement (hypertrophy), secretion of extracellular matrix, or accretion @ external or internal surfaces. negative growth (atrophy) can occur through cell death, cell shrinkage, or resorption. in most cases, hyperplasia and hypertrophy are mutually exclusive processes. depending on the age of the organism and the type of tissue, one of these two growth processes dominates.

Taber „Biomechanics of growth, remodeling and morphogenesis" [1995]

growth, remodeling and morphogenesis

remodeling [ri'mɑd. l.ɪŋ] involves changes in material properties. These changes, which often are adaptive, may be brought about by alterations in modulus, internal structure, strength, or density. for example, bones, and heart muscle may change their internal structures through reorientation of trabeculae and muscle fibers, respectively.

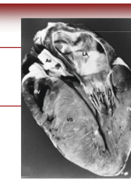
Taber „Biomechanics of growth, remodeling and morphogenesis" [1995]

growth, remodeling and morphogenesis

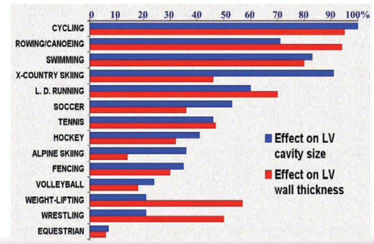
morphogenesis [mɔːr.foˈdʒen.ə.sɪs] is the generation of animal form. usually, the term refers to embryonic development, but wound healing and organ regeneration are also morphogenetic events. morphogenesis contains a complex series of stages, each of which depends on the previous stage. during these stages, genetic and environmental factors guide the spatial-temporal motions and differentiation (specification) of cells. a flaw in any one stage may lead to structural defects.

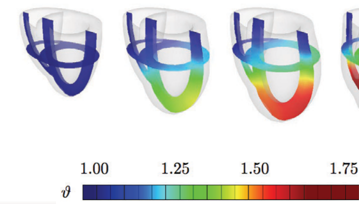
Taber „Biomechanics of growth, remodeling and morphogenesis" [1995]

athelete's heart - a patient-specific simulation



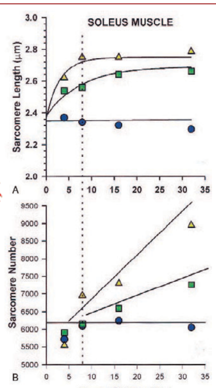
- growth beyond normal size due to **significant exercise**
- training-induced changes are typically **reversible**
- adaptation driven by **elevated pressure** and **increased filling**
- cardiac **output increases from 6 l/min** at rest **up to 40 l/min**
- cardiac **mass increases up to 50%**

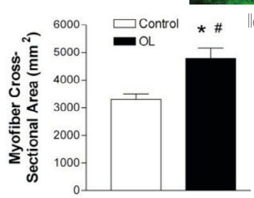


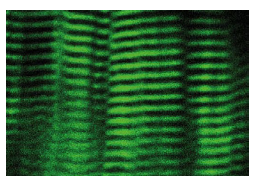


how do skeletal muscles grow when we train?

nbl stanford



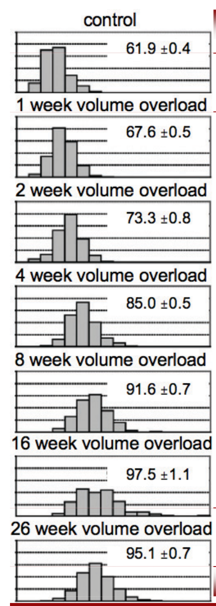




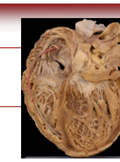
llewellyn, barretto, delp schnitzer [2008]

Figure. Response of rat soleus muscle to overstretch (left). Sarcomere length (top) and sarcomere number (bottom). During the initial phase of loading, days 4 and 8, sarcomere length progressively increases. Beyond this point, however, sarcomere length remains constant and there is a progressive increase of the number of sarcomeres in series. Response of rodent skeletal muscle to overload (right). Myofiber cross section area increases with increased loading.

caizzo et al. [2002], adams et al. [2002]



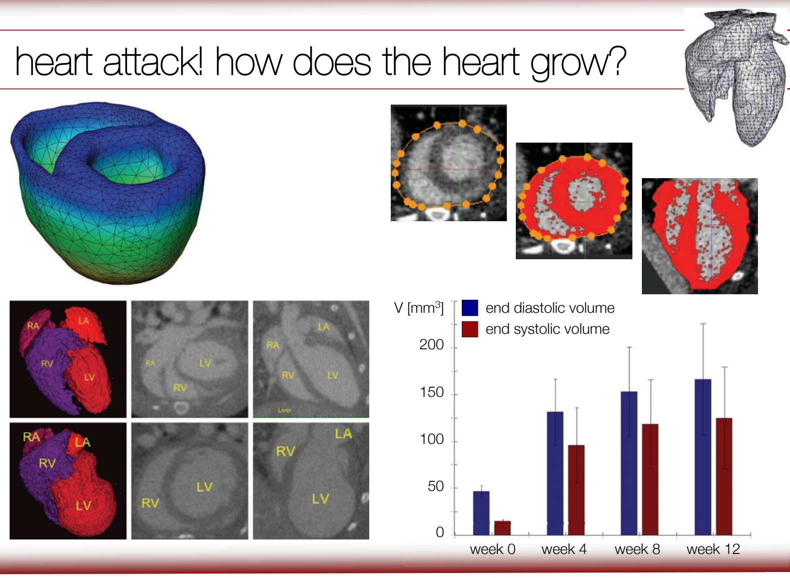
how do cells grow?



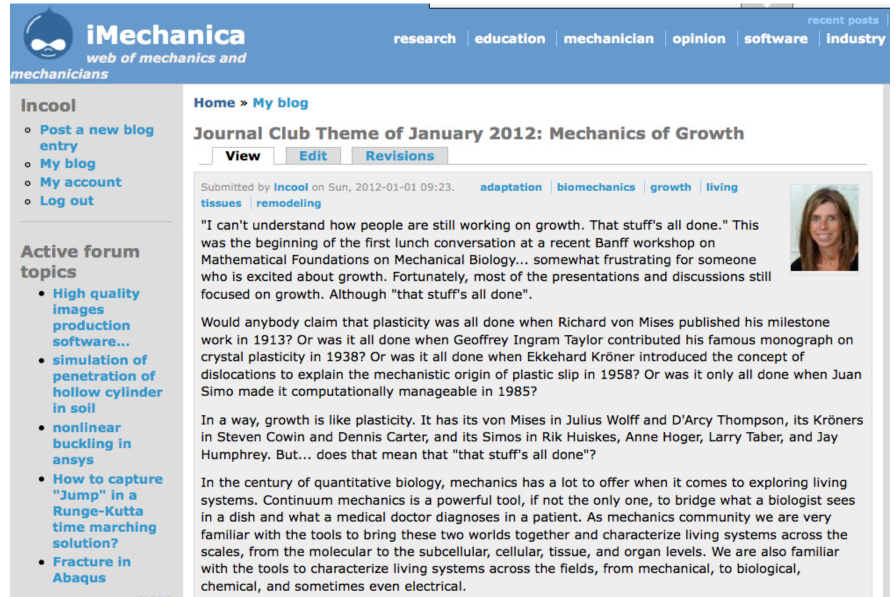
- how do cardiomyocytes change their length?
- left ventricle **enlarged 4-28%** and **dilated 14% at week one** after volume overload
- LV dilation by **cardiomyocyte elongation**
- myocyte elongation by **serial sarcomere deposition**
- sarcomere **number increases linearly** from 62 to 85
- sarcomere deposition **rate is linear in weeks 1 to 4** & decays smoothly from week 4 to saturation at 26

a literature study / review paper

heart attack! how does the heart grow?



group project - mouse heart



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Journal Club Theme of January 2012: Mechanics of Growth

View | Edit | Revisions

Submitted by lncool on Sun, 2012-01-01 09:23. adaptation | biomechanics | growth | living tissues | remodeling

"I can't understand how people are still working on growth. That stuff's all done." This was the beginning of the first lunch conversation at a recent Banff workshop on Mathematical Foundations on Mechanical Biology... somewhat frustrating for someone who is excited about growth. Fortunately, most of the presentations and discussions still focused on growth. Although "that stuff's all done".

Would anybody claim that plasticity was all done when Richard von Mises published his milestone work in 1913? Or was it all done when Geoffrey Ingram Taylor contributed his famous monograph on crystal plasticity in 1938? Or was it all done when Ekkehard Kröner introduced the concept of dislocations to explain the mechanistic origin of plastic slip in 1958? Or was it only all done when Juan Simo made it computationally manageable in 1985?

In a way, growth is like plasticity. It has its von Mises in Julius Wolff and D'Arcy Thompson, its Kröners in Steven Cowin and Dennis Carter, and its Simos in Rik Huiskes, Anne Hoger, Larry Taber, and Jay Humphrey. But... does that mean that "that stuff's all done"?

In the century of quantitative biology, mechanics has a lot to offer when it comes to exploring living systems. Continuum mechanics is a powerful tool, if not the only one, to bridge what a biologist sees in a dish and what a medical doctor diagnoses in a patient. As mechanics community we are very familiar with the tools to bring these two worlds together and characterize living systems across the scales, from the molecular to the subcellular, cellular, tissue, and organ levels. We are also familiar with the tools to characterize living systems across the fields, from mechanical, to biological, chemical, and sometimes even electrical.

get engaged! imechanica journal club