ARTERY 20

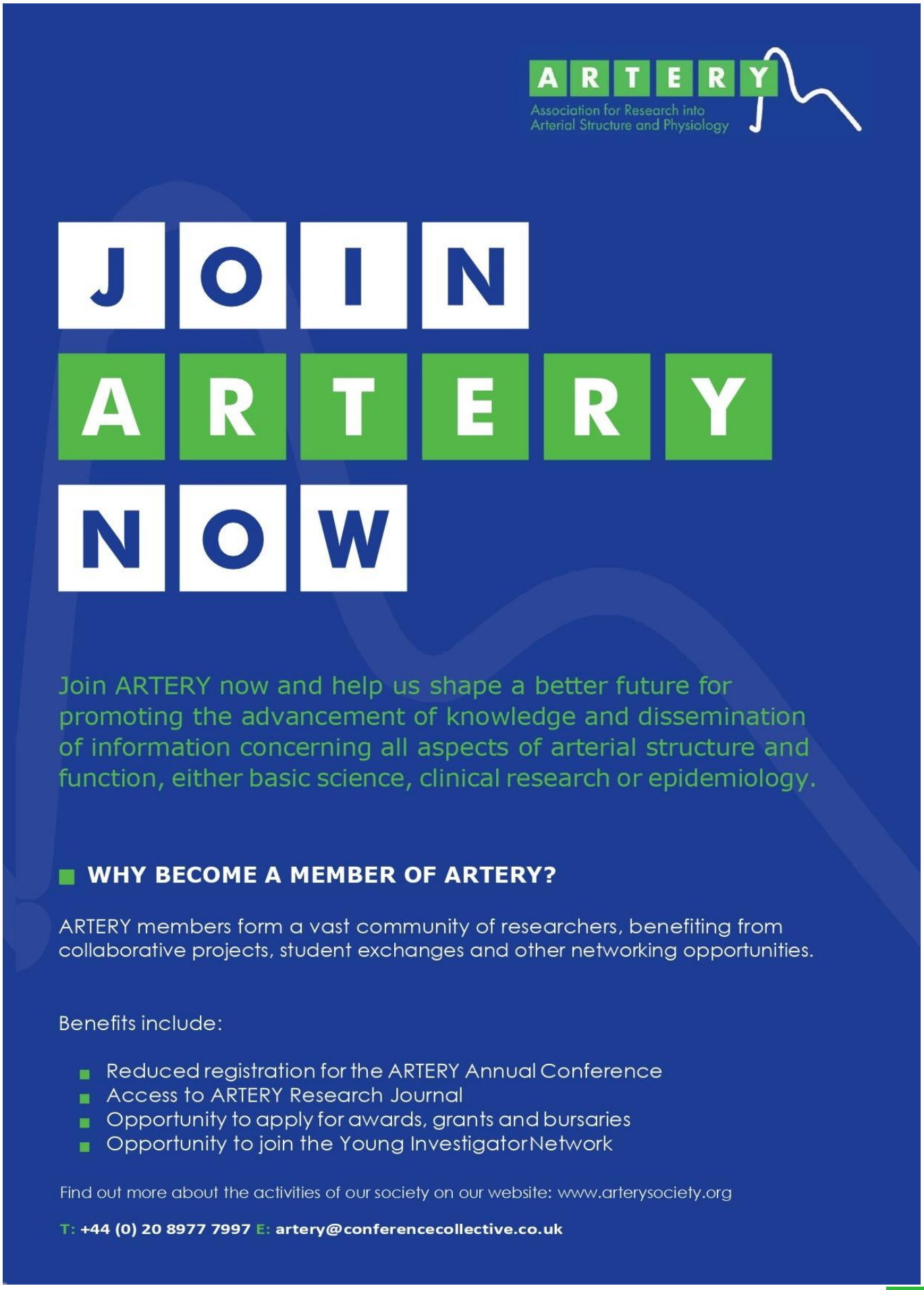
Virtual Conference

Final Programme and Book of Abstracts

Friday 23 October - 14.00-19.00 (CET)
Saturday 24 October - 08.00-13.15 (CET)

ARTERY
Association for Research into
Arterial Structure and Physiology

Join us!


ARTERY
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Join ARTERY now and help us shape a better future for promoting the advancement of knowledge and dissemination of information concerning all aspects of arterial structure and function, either basic science, clinical research or epidemiology.
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Opportunity to join the Young Investigator Network
Find out more about the activities of our society on our website: www.arterysociety.org
T: +44 (0) 20 8977 7997 E: artery@conferencecollective.co.uk

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Instructions on how to Join the Virtual Conference

To sign into the event on the day, please click here and enter the email address that you used to register for the Conference. Once you have done this, follow the instructions to activate your account. For further instructions on how to make the most of the system and for a short video, please click here.

Artery 20 Scientific Committee

ARTERY EXECUTIVE COMMITTEE

President:	**Professor Pierre Boutouyrie**	Paris	France
Vice-President:	**Professor Thomas Weber**	Wells	Austria
Secretary:	**Professor Chakravarthi Rajkumar**	Brighton	UK
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	Dr Bart Spronck	New Haven	United States
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Chair of Council:	**Dr Dimitrios Terentes-Printzios**	Athens	Greece

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Dr Patrick Lacolley, Nancy, France

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ARTERY COUNCIL MEMBERS

The Artery members listed below join the Executive Committee members to form the full Council.

Chair of Artery Council:	**Dr Dimitrios Terentes-Printzios**, Greece
Council Members:	**Professor Stéphane Laurent**, France
	Dr Gary Mitchell, USA
	Dr Koen Reesink, The Netherlands
	Professor Patrick Segers, Belgium
	Professor James Sharman, Australia
	Professor Siegfried Wassertheurer, Austria
	Professor Ian Wilkinson, UK
	Professor Reuven Zimlichman, Israel

Special thanks go to the Young Investigators who created the Virtual Conference Programme:

Dr Rosa Maria Bruno, Dr Andrea Guala, Dr Bernhard Hametner, Dr Bart Spronck, Dr Dimitrios Terentes-Printzios

SECRETARIAT

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Artery Society: www.arterysociety.org

Welcome

Dear Colleague,

2020 has been an exceptional and extremely challenging year for health professionals around the world. The Artery community is no exception; our international, professionally diverse network of members are working on the front line every day; developing new approaches and techniques to treat their patients.

In this challenging environment, we are very grateful for the continued support from members and colleagues across the world as we move our Annual Meeting to a new virtual space for 2020. We are very pleased to welcome colleagues from sister societies in North America (North American Artery), Latin America (LATAM Artery), Asia (Pulse of Asia) and Australia as well as our European network. By working together, we can continue to shape a better future for promoting the advancement of knowledge and dissemination of information concerning all aspects of arterial structure and function through the delivery of a dynamic, interactive and inclusive conference.

We received a large number of high-quality abstracts for this meeting and we are grateful to all our presenters and invited speakers who have agreed to commit their time to provide an engaging and meaningful experience for everyone in attendance. Please take time to attend the poster sessions, read the abstracts, give constructive feedback and discuss the work with the presenters during the conference.

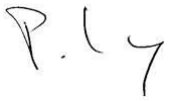
Although there is no better way to experience Artery than attending our annual conference in person, our goal is that this virtual conference will continue to provide the valuable opportunities to develop and maintain professional networks, present new science and research and develop new partnerships. You can help us to achieve this with your full participation in the virtual conference by attending all the sessions on Friday afternoon and Saturday morning. Submit your questions on-line, get involved in the discussions on Social Media and kick-start your Saturday morning with our Zumba session that will offer fun for all your family!

The presentations will be available online on the Artery website after the conference to support continued discussion, encourage research and exchange of ideas.

We are very grateful to Servier, who have continued their support of the Society and through their generosity, the Artery leadership has been able to provide bursaries to cover the cost of attendance.

We look forward to welcoming everyone to Nancy for Artery 2021 but in the meantime, take time out to get involved in this interactive Artery 2020 Virtual conference and enjoy the experience.

Professor Pierre Boutouyrie
President, Artery



Patrick Lacolley
Chair, Local Organising Committee, Artery 20

Programme

PLEASE NOTE THAT ALL TIMES ARE CET

FRIDAY 23 OCTOBER 2020	
14.00 (CET)	**Conference Opening** Chairs: P Boutouyrie, B Hametner and P Lacolley
14.10 (CET)	**Young Investigator Oral Session 1** Chairs: P Boutouyrie, B Hametner **YI 1.1 Aortic Impedance and Total Arterial Compliance from Regional Pulse Wave Velocities** Ms Vasiliki Bikia, École Polytechnique Fédérale de Lausanne, Switzerland **YI 1.2 Ideal cardiovascular health score declines from adolescence to emerging adulthood** Dr Chloe Park, University College London, London, United Kingdom **YI 1.3 Retinal microvascular calibers and incident depressive symptoms: The Multi-Ethnic Study of Atherosclerosis** Ms April Van Gennip, Maastricht University Medical Centre, Netherlands **YI 1.4 Increases in Circulating Trimethylamine-N-oxide Contribute to the Development of Age-Related Aortic Stiffness in Humans and Mice** Ms Abigail Casso, University of Colorado Boulder, United States **YI 1.5 Ten years of ageing in the middle-aged does not increase input impedance or wave reflection - insights from the Asklepios Study.** Mrs Daimé Campos Arias, Gent, Belgium **YI 1.6 Flow mediated slowing of pulse wave velocity as a measure of endothelial function** Ms Anju Sharma, Aiims, Rohtak, New Delhi, India **YI 1.7 Transmural quantification of murine vascular smooth muscle cell density distribution from 3D microscopy images** Mr Koen Van Der Laan, Maastricht University, Maastricht, Netherlands **YI 1.8 A computational model-based study on the effect of abdominal aortic aneurysm on pulse wave morphology** Mr Tianqi Wang, King's College London, United Kingdom
15.30 (CET)	**Break**
15.45 (CET)	**Debate:** Chairs: A Hughes, T Weber **EVA vs. SUPERNOVA** Dr Edward G. Lakatta, M.D. Georgetown University School of Medicine, Washington DC and Professor Stéphane Laurent, Emeritus Professor of Pharmacology, Paris Descartes Medical School
16.15 (CET)	**Break**
16.20 (CET)	**Invited Lecture** Chairs: R M Bruno, A Guala **Sympathetic control of arterial stiffness and central hemodynamics** Dr Gary Pierce, Associate Professor in the Department of Health and Human Physiology, and Department of Internal Medicine, Division of Nephrology and Hypertension, University of Iowa
16.40 (CET)	**Break**
16.45 (CET)	**Poster Session 1**
	Parallel 1 – Models and Methodologies Chairs: T Weber, B Spronck
	Parallel 2 – Epidemiology & Brain Chairs: C Rajkumar, T Willum Hansen
	Parallel 3 – Vascular Ageing 1 Chairs: S Laurent, B Hametner

	Parallel 4 – Vascular Biology and Pathophysiology Chairs: P Lacolley, A Guala
17.45 (CET)	**Break**
17.50 (CET)	**Artery Annual General Meeting** Chairs: P Boutouyrie, T Weber
18.20 (CET)	**Networking Session: Introduction** Chair: A Guala
18.25 (CET)	**Get your drinks break**
18.30 (CET)	**Networking Session: Shuffle 1**
18.40 (CET)	**Networking Session: Shuffle 2**
18.50(CET)	**Networking Session: Shuffle 3**
19.00 (CET)	**End of day one**

SATURDAY 24 OCTOBER 2020	
08.00 (CET)	**Virtual Zumba Session – everyone is invited to participate**
08.30 (CET)	**Break**
09.00 (CET)	**Saturday Opening** Chairs: T Willum Hansen, P Lacolley
09.05 (CET)	**CARTESIAN Update** Rosa Maria Bruno
09.10 (CET)	**Young Investigator Oral Session 2** Chairs: T Willum Hansen, P Lacolley **YI 2.1 Pulse wave velocity estimation from the radial pulse waveform using Gaussian process regression: A machine learning based study** Ms Weiwei Jin, King's College London, United Kingdom **YI 2.2 Spontaneous cardiovascular ageing of C57Bl6 mice results in the development of aortic stiffness prior to peripheral blood pressure alterations.** Miss Sofie De Moudt, University of Antwerp, Belgium **YI 2.3 Methylglyoxal, 3-deoxyglucosone, and glyoxal – precursors of advanced glycation endproducts – are not independently associated with indices of carotid stiffness: The Maastricht Study** MD Myrthe van der Bruggen, CARIM School for Cardiovascular Diseases, Maastricht University, Netherlands **YI 2.4 Neural baroreflex sensitivity and long-term effect of antihypertensive agents--a pharmacological substudy of the Paris Prospective Study III** Dr Catherine Fortier, Inserm-U970, Paris Cardiovascular Research Centre, France **YI 2.5 Direct measurement of stiffness index β of superficial arteries without blood pressure estimation** Mr. Rahul Manoj, Indian Institute of Technology Madras, Chennai, India **YI 2.6 Comparison of cardiovascular disease primary prevention guidelines between Australia, England and the United States.** Miss Niamh Chapman, University of Tasmania, Hobart, Australia
10.10 (CET)	**Break**
10.15 (CET)	**The History of the Artery Society** Chairs: C Rajkumar, B Spronck Professor John Cockcroft, Visiting Professor in the Department of Advanced Cardiology, Columbia University Medical Centre, New York, USA
10.30 (CET)	**Update on Artery Young Investigator Initiatives** Chairs: C Rajkumar, B Spronck Dr Bernhard Hametner, AIT Austrian Institute of Technology
10.40 (CET)	**Artery 2019 Exchange Grant Presentation** Chairs: C Rajkumar, B Spronck

	Tri-layered constitutive modelling of the arterial wall: Evaluation of a new approach based on uniaxial testing of isolated layers (See page 1 of Book of Abstracts for full abstract) Alessandro Giudici, Brunel University London
10.55 (CET)	**Break**
11.05 (CET) **11.20 (CET)**	**Young Investigator Network Session: The role of social media in research** Chairs: T Weber, D Terentes-Printzios **Communication strategy and how to best generate content to mainstream audiences and policymakers** Karima BenSalah, Senior Communications, European Cooperation in Science and Technology, Belgium **How young investigators can circulate and advertise their work through social media and other networks increasing the impact on their work?** (See page 2 of Book of Abstracts for full abstract) Sophia Sheridan, Digital Communications Officer, European Cooperation in Science and Technology, Belgium
11.35 (CET)	**Break**
11.40 (CET)	**Poster Session 2**
	Parallel 5 – Vascular Ageing 2 Chairs: C Mayer and D Terentes-Printzios
	Parallel 6 – Imaging Technologies Chairs: P Segers, R M Bruno
	Parallel 7 – Hypertension Chairs: A Hughes, B Spronck
	Parallel 8 – Other Chairs: V Regnault, Johannes Baulmann
12.40 (CET)	**Break**
12.45 (CET)	**Invited Lecture:** Chairs: V Regnault, R Climie **Reappraising the role of inflammation in arterial stiffness** Dr Esther Lutgens, Professor of Vascular Immunopathology, Amsterdam University Medical Centre
13.05 (CET)	**Awards Ceremony and Closing Remarks** Chairs: P Boutouyrie, P Lacolley
13.15 (CET)	**Virtual Conference Ends**

Virtual Exhibitors

On behalf of the Artery Committee we would like to thank ALF Distribution GmbH (UNEX/Imedos) for supporting the very first Artery Virtual Conference.
We encourage all delegates to visit the Exhibitor section within the Conference Platform, where you can find further information and have one-to-one meetings with the Exhibitor.

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ALF Distribution GmbH – Official representative for UNEX and Imedos medical device brands – Endothelial function measurement in the macro- and micro-circulation

Speakers

Karima BenSalah:

Karima BenSalah is currently the Senior Communication Officer of the COST Association. Her primary responsibility is to develop and implement a pan European media strategy to support the COST Actions in their communication and dissemination activities. The objective is to increase media presence and awareness about the COST Programme in Europe and support the Actions.

Before joining the communication team of the COST Association, Karima has worked for 14 years as a press officer and news desk coordinator at the European Parliament Press service. She managed a team of Press officers responsible for assisting radio and television professionals in obtaining relevant information for their news coverage of the EU. Karima BenSalah was alerting and informing most European Newsrooms and has contributed in developing the creation of new TV and radio program dedicated to European issues in the media landscape.

Previously, and as a former journalist, Karima BenSalah has worked for various media outlets and broadcasters for 10 years in the United Kingdom. While in London, Karima worked as an information analyst for Premium Press, a press agency providing news summaries on various topics. You can reach Karima at karima.BenSalah@cost.eu

Professor John Cockcroft:

Professor Cockcroft is visiting Professor in the Dept of Advanced Cardiology at Columbia University Medical Centre, New York.

He was a founder member and inaugural President of the Artery Society and is currently Editor in Chief of the Society's journal Artery Research.

His recent research focuses on cuffless measurement of blood pressure and the use of artificial intelligence (AI) to better define and manage hypertension

Dr. Alessandro Giudici:

Alessandro Giudici was born in Milan, Italy in 1992. He received the B.S. and M.S. degree in biomedical engineering from Politecnico di Milano University, Italy, in 2014 and 2017, dedicating the last years of his studies to biomechanics, tissue engineering and life support systems. He is currently pursuing the Ph.D. degree in biomedical engineering at Brunel University London, UK under the supervision of Professor A Khir.

His research focuses on the characterisation of arterial mechanics in both physiological and pathological conditions, combining ex-vivo experimental work and analysis of clinical data.

Alessandro Giudici was a recipient of the Artery Society Exchange Grant in 2019.

Dr. Bernhard Hametner:

Bernhard Hametner received his PhD in Applied Mathematics from the Vienna University of Technology in 2012. He is currently working as a Scientist at the AIT Austrian Institute of Technology in Vienna in the research group for Cardiovascular Diagnostics. He focuses on the development of mathematical models and algorithms for arterial pulse wave analysis, with the main goal to quantify wave transmission, wave reflection and arterial stiffness. Additionally, he is involved in the analysis of cardiovascular parameters obtained in clinicals studies. He is also active in teaching and the supervision of students, as well as in the management of research projects.

Dr. Edward Lakatta:

Edward is the founder and Director of the Laboratory of Cardiovascular Science, National Institute on Aging, National Institutes of Health. In a sustained 30-plus-year commitment to a broad-based research career, Edwards's studies range from molecules to humans, including translation of novel findings into the clinical realm. The overall goals of Edward's research program are 1) to identify age associated changes that occur within the cardiovascular system and to determine the mechanisms for these changes; 2) to determine how aging of the heart and vasculature interacts with chronic disease states to enhance the risk for CV diseases in older persons; 3) to study basic mechanisms in excitation-contraction coupling and how these are modulated by surface receptor signaling pathways in cardiac cells; 4) to elucidate mechanisms of pacemaker activity in sinoatrial nodal cells; 5) to elucidate mechanisms that govern cardiac and vascular cell survival; 6) to establish the potentials and limitations of new therapeutic approaches such as changes in lifestyle, novel pharmacologic agents or gene or stem cell transfer techniques in aging or disease states.

Professor Stéphane Laurent:

Stéphane Laurent is Emeritus Professor of Pharmacology in the Paris Descartes Medical School. Professor Laurent was head of the Department of Clinical Pharmacology in the Hôpital Européen Georges Pompidou in Paris between 2008 and 2018, and head of INSERM U 970, team 7 in the Paris Cardiovascular Research Center (PARCC) between 2009 and 2018. His research interests are hypertension pathophysiology and management, pharmacology of antihypertensive drugs, arterial stiffness and vascular ageing.

Professor Laurent has served as President of the European Society of Hypertension (ESH) (2007-2009), President of the ARTERY Society (2010-2012), and President of the French Society of Hypertension (2001-2002).

Professor Laurent is co-author of the 2007, 2013 and 2018 ESH-ESC Guidelines for the management of Hypertension, and the 2009 ESH document on the reappraisal of hypertension guidelines.

He is the author of over 400 referenced articles and 20 chapters in books. His "h" index is 81 (Web of Science) with 15 "highly cited" papers, and the total number of citations of his articles is higher than 47 156 (Web of Science). Google Scholar's H index is 97 with more than 120 000 citations. The expertise of Stephane Laurent ranks first among 21 040 published authors worldwide for "Arterial stiffness" and is in the Top 0.1% of more than 100 000 published authors worldwide on "Arterial pressure and hypertension" from 2010 through 2020 (Expertscape).

Professor Laurent has delivered more than 400 invited lectures at international venues.

MD PhD Esther Lutgens:

Esther Lutgens (1975) did an MD/PhD trajectory in Experimental Vascular Pathology (1994-2001) at the University of Maastricht (the Netherlands), with Prof. Mat Daemen, and subsequently did post-doctoral fellowships at Harvard University in Boston, MA, with Prof. Peter Libby, and at Dartmouth University in Hanover, NH, with Prof. Michael Simons.

After her return to the Netherlands in 2003, she set up her laboratory on Experimental Vascular Immuno-pathology within the Cardiovascular Research Institute Maastricht. She became an 'Established Investigator' of the Dutch Heart Foundation in 2009, and obtained a Sofja Kovalevskaja fellowship in Germany in 2008, which enabled her to open a second laboratory within the 'Institute for Cardiovascular Prevention' (LMU, Munich) headed by Prof. Christian Weber. In 2011, she was appointed as a full professor at the Amsterdam University Medical Center (the Netherlands).

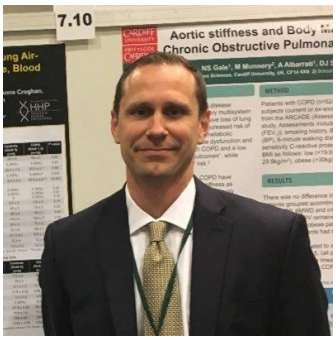
The focus of her research is the role of immune-modulation in atherosclerosis, especially on the role of co-stimulatory and co-inhibitory immune checkpoints and she has published numerous papers in this field in established journals such as *ATVB, Circulation, Eur Heart J, Cardiovasc Res, JACC* and *Nat Med.*

In 2013, she obtained the prestigious Vici grant (NWO), and in 2016, she was awarded the J. Hoeg award from the ATVB council (AHA), and in 2018 she received the outstanding achievement award for basic science from the European Society of Cardiology, and in 2020 the Arteriosclerosis recognition award from the AHA. In 2016, she received a European Research Council consolidator grant. Esther is part of many consortia.

Esther has guided over 30 master students, and 21 PhD students. She has mentored 13 postdocs and helped them pursue their career in science or in pharmaceutical industry. Moreover, Esther is a dedicated teacher who lectures on cardiovascular disease in (post)-graduate and PhD courses worldwide.

Esther is married and has 2 children, Willemijn (14) and Jasper (12).

Dr. Gary Pierce:



Dr. Gary Pierce is an Associate Professor in the Department of Health and Human Physiology, and Department of Internal Medicine, Division of Nephrology and Hypertension at the University of Iowa. He is also faculty member in the Abboud Cardiovascular Research Center, Environmental Health Sciences Research Center and the Fraternal Order of Eagles Diabetes Research Center in the University of Iowa Carver College of Medicine and is a fellow of the American Heart Association. He received a PhD in Exercise Physiology from the University of Florida and completed postdoctoral training in vascular aging/physiology in the Department of Integrative Physiology at the University of Colorado Boulder.

Sophia Sheridan:

Sophia Sheridan is the Digital Communications Officer for European Cooperation in Science and Technology; specialising in reaching large-scale and targeted science audiences through online platforms. She has over 10 years of experience in communications, marketing, and PR roles, including training NGOs and other not for profit organisations on effective digital strategies and social media engagement. Her experience means that she is able to give a broad range of practical tips to help strengthen the online presence and reach of both organisations and individuals alike.

She has previously worked as the Communications Lead for an NGO creating sustainable communities, the lead PR and Marketing Executive for a largescale healthcare company and as part of the international marketing and study abroad team for the University of Exeter. Sophia has also worked as a freelance journalist and writer, with soundscape, stage and radio plays aired in the UK and USA, including on the BBC.

Oral Presentations

YI 1.1 Aortic Impedance and Total Arterial Compliance from Regional Pulse Wave Velocities

Ms Vasiliki Bikia[1], Mr Georgios Rovas[1], Ms Stamatia Pagoulatou[1], Prof. Nikolaos Stergiopulos[1]
[1]École polytechnique fédérale de Lausanne, Lausanne, Switzerland

YI 1.2 Ideal cardiovascular health score declines from adolescence to emerging adulthood

Dr Chloe Park[1], Dr Siana Jones[1], Miss Suzanne Williams[1], Mrs Alicja Rapala[1], Ms Hannah Taylor[1], Dr Laura Howe[2], Dr Abigail Fraser[2], Professor Nish Chaturvedi[1], Professor Alun Hughes[1]
[1]University College London, London, United Kingdom, [2]University of Bristol, Bristol, United Kingdom

YI 1.3 Retinal microvascular calibers and incident depressive symptoms: The Multi-Ethnic Study of Atherosclerosis

Ms April C. E. van Gennip[1], Ms Sanaz Sedeghat[2], Ms Mercedes R. Carnethon[2], Ms Norrina B. Allen[2], Ms Barbara E. K. Klein[3], Ms Mary Frances Cotch[4], Ms Diana A. Chirinos[2], Mr Coen D. A. Stehouwer[1], Mr Thomas T. van Sloten[1]
[1]Department of Internal Medicine, Cardiovascular Research Institute Maastricht, Maastricht University Medical Centre, Maastricht , the Netherlands, [2]Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, Chicago, USA, [3]Ocular Epidemiology, University of Wisconsin-Madison, Madison, USA, [4]Division of Epidemiology and Clinical Applications, Intramural Research Program, National Eye Institute, National Institutes of Health, Bethesda, USA

YI 1.4 Increases in Circulating Trimethylamine-N-oxide Contribute to the Development of Age-Related Aortic Stiffness in Humans and Mice

Abigail G Casso[1], Rachel A Gioscia-Ryan[1], Zachary J Sapinsley[1], Nicholas S VanDongen[1], Amy E Bazzoni[1], Andrew P Neilson[2], Melanie C Zigler[1], Kevin P Davy[3], Douglas R Seals[1], Vienna E Brunt[1]
[1]University Of Colorado Boulder, Boulder, United States, [2]North Carolina State University, Raleigh, USA, [3]Virginia Tech, Blacksburg, USA

YI 1.5 Ten years of ageing in the middle-aged does not increase input impedance or wave reflection - insights from the Asklepios Study.

Daimé Campos Arias[1], Marc L. De Buyzere[2], Julio A. Chirinos[3,4], Ernst R. Rietzschel[2,5], Patrick Segers[1]
[1]IBiTech, Ghent University, Ghent, Belgium, [2]Cardiology Department, Ghent University Hospital, Ghent, Belgium, [3]Division of Cardiovascular Medicine, Hospital of the University of Pennsylvania, Philadelphia,, USA, [4]Perelman Center for Advanced Medicine, University of Pennsylvania, Philadelphia,, USA, [5]Biobanking and Cardiovascular Epidemiology, Ghent University Hospital, Ghent, Belgium

YI 1.6 Flow mediated slowing of pulse wave velocity as a measure of endothelial function

Anju Sharma[1], Dinu.S. Chandran[1], Ashok Jaryal[1], Kishore K Deepak[1]
[1]Aiims New Delhi, Delhi, India

YI 1.7 Transmural quantification of murine vascular smooth muscle cell density distribution from 3D microscopy images

Phd Koen W.F. van der Laan[1,2], PhD Koen D. Reesink[1,2], PhD, MD Myrthe M. van der Bruggen[1,2], PhD Armand M.G. Jaminon[1,3], PhD Remco T.A. Megens[1,2,4], PhD Leon J. Schurgers[1,3], Phd, MD Tammo Delhaas[1,2], PhD Bart Spronck[1,2,5]
[1]CARIM School for Cardiovascular Diseases, Maastricht University, Maastricht, Netherlands, [2]Department of Biomedical Engineering, Maastricht Univeristy, Maastricht, Netherlands, [3]Department of Biochemistry, Maastricht University, Maastricht, Netherlands, [4]Institute for Cardiovascular Prevention, Ludwig Maximilians University (LMU),, Munich, Germany, [5]Department of Biomedical Engineering, School of Engineering & Applied Science, Yale University, New Haven, USA

YI 1.8 A computational model-based study on the effect of abdominal aortic aneurysm on pulse wave morphology

Mr. Tianqi Wang[1,2], Dr. Jordi Alastruey[1], Dr. Fuyou Liang[2]
[1]Department of Biomedical Engineering, King's College London, United Kingdom, [2]School of Naval Architecture, Ocean and Civil Engineering, Shanghai Jiao Tong University, Shanghai, China

YI 2.1 Pulse wave velocity estimation from the radial pulse waveform using Gaussian process regression: A machine learning based study

Ms Weiwei Jin[1], Dr Phil Chowienczyk[2], Dr Jordi Alastruey[1,3]
[1]Department of Biomedical Engineering, School of Biomedical Engineering and Imaging Sciences, King's College London, United Kingdom, [2]British Heart Foundation Centre, Department of Clinical Pharmacology, St. Thomas' Hospital, King's College London, United Kingdom, [3]Institute of Personalized Medicine, Sechenov University, Moscow, Russia

YI 2.2 Spontaneous cardiovascular ageing of C57Bl6 mice results in the development of aortic stiffness prior to peripheral blood pressure alterations.

Miss Sofie De Moudt[1], Miss Jhana O. Hendrickx[1], Miss Dorien G. De Munck[1], Dr. Arthur J. Leloup[1], Prof. Wim Martinet[1], Prof. Guido R.Y. De Meyer[1], Dr Paul Fransen[1]
[1]University Of Antwerp, Antwerp, Belgium

YI 2.3 Methylglyoxal, 3-deoxyglucosone, and glyoxal – precursors of advanced glycation endproducts – are not independently associated with indices of carotid stiffness: The Maastricht Study

MD Myrthe van der Bruggen[1,2], PhD Marleen M.J. van Greevenbroek[1,3], PhD Koen D. Reesink[1,2], PhD, MD Coen D.A. Stehouwer[1,3], PhD, MD Tammo Delhaas[1,2], PhD Bart Spronck[1,2,4], PhD Casper G. Schalkwijk[1,3]
[1]CARIM School for Cardiovascular Diseases, Maastricht University, Maastricht, The Netherlands, [2]Department of Biomedical Engineering, Maastricht University, Maastricht, The Netherlands, [3]Department of Internal Medicine, Maastricht University Medical Centre+, Maastricht, The Netherlands, [4]Department of Biomedical Engineering, School of Engineering & Applied Science, Yale University, New Haven, USA

YI 2.4 Neural baroreflex sensitivity and long-term effect of antihypertensive agents--a pharmacological substudy of the Paris Prospective Study III

Nicolas Danchin[5], Catherine Guibout[3,4], Xavier Jouven[3,4], Marie-Cécile Perier[3,4], Frederique Thomas[5], Dr Catherine Fortier[1], Dr Jean-Philippe Empana[3,4], Dr Hakim Khettab[2], Dr Rosa-Maria Bruno[1,2], Dr Pierre Boutouyrie[1,2]
[1]INSERM, U970, Paris Cardiovascular Research Center, Cellular molecular and physiological mechanisms of heart failure (Team 7), Paris, France, [2]AP-HP, Pharmacology Unit, Hôpital Européen Georges Pompidou, Université de Paris, Paris, France, [3]INSERM U970, Paris Cardiovascular Research Centre (PARCC), University of Paris, Paris, France, [4]INSERM U970, Paris Cardiovascular Research Centre (PARCC), Integrative Epidemiology of Cardiovascular Disease (Team 4), Paris, France, [5]Preventive and Clinical Investigation Center (IPC), Paris, France

YI 2.5 Direct measurement of stiffness index β of superficial arteries without blood pressure estimation

Mr. Rahul Manoj[1], Dr. P M Nabeel[2], Mr. Kiran V Raj[1], Dr. Jayaraj Joseph[1,2], Dr. Mohanasankar Sivaprakasam[1,2]
[1]Department of Electrical Engineering, Indian Institute of Technology Madras, Chennai, India, [2]Healthcare Technology Innovation Centre, Indian Institute of Technology Madras, Chennai, India

YI 2.6 Comparison of cardiovascular disease primary prevention guidelines between Australia, England and the United States.

Dr Niamh Chapman[1], Dr Monique Breslin[1], Dr Sarah Lay-Flurrie[2], Dr Zhen Zhou[1], Prof. James Sharman[1], Prof. Mark Nelson[1], Prof Richard McManus[2]
[1]University Of Tasmania, 1Menzies Institute for Medical Research, Hobart, Australia, [2]University of Oxford, 2Nuffield Department of Primary Care Health Sciences, Oxford, United Kingdom

Poster Presentations

P.01 Where does the reflected wave observed in the ascending aorta come from?

Miss Shima Abdullateef[1], Professor Ashraf W Khir[1]
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P.02 Differential 'mediators' of low-flow 'mediated' constriction in healthy vs patients of ischemic heart disease

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P.03 Local Pulse Wave Velocity Estimation using a Double Gaussian Propagation Model

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P.04 A transfer-function-free technique for the non-invasive estimation of central arterial pressure

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P.05 Development and validation of a novel centroid method for estimating effective reflection time

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P.06 Comparison of Manual vs. Automated Haemodynamic Monitoring Systems in the Cardiac Catheterization Laboratory

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P.07 The Progression of Left Ventricular Ejection Time in Simulated Microgravity

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P.08 Biomechanical Characterization of Ascending Thoracic Aortic Aneurysms in Humans: A Continuum Approach to in vivo Deformations

MSc Shaiv Parikh[1,2], PhD Bart Spronck[1,2,3], BSc Gijs Debeij[1,4], MSc Berta Ganizada[1,4], MD Mitch Ramaekers[1,5,6], PhD Simon Schalla[1,5,6], PhD Ehsan Natour[1,4], PhD Jos Maessen[1,4], PhD Tammo Delhaas[1,2], PhD Wouter Huberts[1,2], PhD Elham Bidar[1,4], PhD Koen Reesink[1,2]
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P.09 Differential Low Flow Mediated Constriction (LFMC) responses in radial and brachial arteries of healthy humans are attributed to occlusion induced flow changes.

Ms Sakshi Sen[1], Dr Dinu Chandran[1], Dr Ashok Jaryal[1], Dr Kishore Kumar Deepak[1]
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P.10 Distal arterial occlusion at different grades of supra-systolic pressures differentially modulates flow velocity and shear rates in radial Artery

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P.12 Investigating the role of glycemic markers in pulse pressure amplification in young adults: The African-PREDICT study

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P.13 Pulse wave velocity trajectories during COVID-19 epidemic: effect of lockdown on cardiovascular health

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P.14 Transcranial colour duplex reveals haemodynamically significant venous flow alterations following resection of arteriovenous malformation of the brain

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P.15 Isolated systolic hypertension and central blood pressure: Implications from the National Nutrition and Health Survey in Taiwan

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P.16 Expanding on the observed correlation between the ambulatory arterial stiffness index and the lower limit of cerebral autoregulation during cardiac surgery

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P.17 Reduced isometric contractility and isobaric compliance of the ex vivo thoracic aorta of hypertensive APP23+/- overexpressing mice due to serum corticosterone levels

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P.18 Carotid Stiffness Parameters and Cerebral Blood Flow Pulsatility in Young Healthy Individuals across Races

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P.19 Intradialytic changes in cerebral blood flow and regional changes in arterial stiffness

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P.20 Evolving Structure-Function Correlates during Aortic Maturation and Aging

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P.21 Albuminuria intensifies the relationship between urinary sodium excretion and central pulse pressure: the Wakuya study

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P.22 Mortality in 98 Type 1 Diabetes Mellitus (T1DM) and Type 2 Diabetes Mellitus (T2DM) Individuals presenting to a Specialist Podiatry Clinic: Foot Ulcer Location is an Independent Risk Determinant

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P.23 Relationship between aortic stiffness, aortic, and carotid impedance with vascular aging in community-based healthy people.

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P.24 Factors associated with premature vascular aging in patients with arterial hypertension.

I.V. Inna Melekhina[1], E.G. Elizaveta Georgievna Medvedeva[1], S.V. Svetlana Ivanova[1], E.N. Elena Yushchuk[1], E.Yu. Ekaterina Trush[1]
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P.25 Radial-Digital pulse wave velocity: a non-invasive method for assessing stiffness of peripheral small arteries

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P.26 Liver Transglutaminase 2 Level Comparison Among Different Dietary Interventions

Miss Elif Oztemiz[1], Associate Prof Soner Dogan[1], Assistant Prof Bilge Guvenc Tuna[1]
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P.27 Mechanisms of NADPH oxidase participation in the regulation of diaphragm Artery contractile responses

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P.28 Comparison of regional vs local arterial parameters using new US technology

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P.29 Involvement of cannabinoid receptors in regulation of MMPs, cell proliferation and apoptosis in vascular smooth muscle cells

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P.30 Angiotensin II Infusion Leads to Aortic Dissection in LRP8 Deficient Mice

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P.31 Von Willebrand Factor Induces Vascular Smooth Muscle Cell Proliferation and Migration Through Low Density Lipoprotein-Related Receptor Protein 4 And αvβ3 Integrin

Cécile V. Denis[3], Patrick Lacolley[1], PhD Jeremy Lagrange[1], Peter J. Lenting[3], Jean-Baptiste Michel[2], Alexandre Raoul[1], Véronique Regnault[1]
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P.32 Non-invasive measures of arteriosclerosis across childhood and adolescence: Insights into the natural history of disease

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P.33 Changes in blood pressure, pulse wave velocity and augmentation index induced by postural changes and exercise

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P.34 Preeclampsia leads to the delayed development of sympathetic control of the cardiovascular system in the offspring

Ms Ekaterina Selivanova[1], Dr Anastasia Shvetsova[1], Dr Victoria Potekhina[1], Dr Dina Gaynullina[1], Dr Anna Borzykh[2], Dr Oxana Kiryukhina[3], Dr Vladislav Kuzmin[1], Dr Olga Tarasova[1]
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P.35 TASK-1 channels play an anticontractile role in rat septal coronary Artery under pharmacological blockade of endothelium

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P.36 Carotid Artery correlates with aorta reactivity to sympathetic stimulation in healthy individuals and patients with abdominal aortic aneurysm

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P.37 An assessment of potential sources of error that may arise in the measurement of carotid-femoral pulse wave velocity

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P.38 Comparison of arterial hemodynamics in early vascular aging (EVA), average vascular aging (AVA) and healthy vascular aging (HVA)

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P.39 The role of advanced glycation end products in vascular ageing. Which parameter is the most suitable as biomarker?

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P.40 Ambulatory Measurement of Carotid Stiffness with a Novel Accelerometric System

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P.41 Measurement of pressure-dependent intra-beat changes in carotid pulse wave velocity using image-free fast ultrasound

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P.42 The effects of chemotherapy on arterial stiffness in patients with Hodgkin lymphoma

Constantinos Anagnostopoulos[2], Stavroula Giannouli[3], Nikolaos Ioakimidis[1], Paulos Kafouris[4], Iosif Koutagiar[1], Anastasia Sioni[5], Doctor Eirini Solomou[1], Dimitrios Terentes-Printzios[1], Dimitrios Tousoulis[1], Charalampos Vlachopoulos[1]
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P.43 The association between early vascular aging and cyclothymic affective temperament

Dr. Milan Vecsey-Nagy[1], Dr. Bálint Szilveszter[1], Dr. Márton Kolossváry[1], Dr. Xénia Gonda[2,3,4], Dr. Zoltán Rihmer[3], Dr. Béla Merkely[1], Dr. Pál Maurovich-Horvat[1,5], Dr. János Nemcsik[6,7]
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P.44 Application of an algorithm developed for measuring gastrointestinal motility to the assessment of arterial mechanical properties.

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P.45 Characterization of the microcirculatory response to gravity-induced changes using thermal imaging

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P.46 Assessment of intraplaque hemorrhage by photoacoustics imaging (PAI): first in-vivo human validation study

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P.47 Feasibility evaluation of imaging-free ultrasound technology to measure diameters of brachial and radial arteries for assessment of endothelial function

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P.48 Ultrasound-based velocity and acceleration of the carotid atheromatous plaque in asymptomatic patients with moderate and severe stenosis

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P.49 Aortic root longitudinal strain by speckle-tracking echocardiography: comparison with cardiac magnetic resonance and predictive value in Marfan syndrome patients

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P.50 Radial Artery phenotyping in fibromuscular dysplasia through ultra-high frequency ultrasound: a radiomic approach

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P.51 POSTER WITHDRAWN BY AUTHOR

P.52 POSTER WITHDRAWN BY AUTHOR

P.53 Ascending aorta diameter and pulse wave velocity are increased and local hemodynamic is disrupted in patients with blunt traumatic thoracic aortic injury treated by TEVAR.

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P.54 Biomarkers and haemodynamic Predictors of Left Atrial Strain in Early Hypertension

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P.55 Dietary nitrate prevents progression of carotid subclinical atherosclerosis through BP-independent mechanisms in patients with or at risk of type 2 diabetes mellitus: results from the double-blind, randomized-controlled, factorial Vasera trial

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P.56 Differences in vascular effects between one session of moderate-intensity continuous physical exercise and high-intensity interval physical exercise in individuals with high blood pressure

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P.57 Acetylsalicylic acid reduces passive aortic wall stiffness and cardiac remodelling in a mouse model of advanced atherosclerosis

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P.58 Genetic Background Dictates Aortic Fibrosis in Hypertensive Mice

Dr. Bart Spronck[1,2], Dr. Marcos Latorre[1], Dr. Sameet Mehta[3], Dr. Alexander W. Caulk[1], Dr. Abhay B. Ramachandra[1], Dr. Sae-Il Murtada[1], Ms. Alexia Rojas[1], Dr. Chang-Sun He[4], Dr. Bo Jiang[4], Dr. Mo Wang[4], Dr. Matthew R. Bersi[5], Prof. George Tellides[4,6], Prof. Jay D. Humphrey[1,6]

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P.59 POSTER WITHDRAWN BY AUTHOR

P.60 Improvement in muscular strength within one year is associated with increased arterial stiffness in young male soccer players

MPH Lisa Baumgartner[1], Dr. phil. Heidi Weberruß[1], M. Sc. Katharina Appel[1], Dipl.-Sportwiss. Tobias Engl[1], Prof. Dr. Renate Oberhoffer-Fritz[1], Dr. Sportwiss. Thorsten Schulz[1]

[1]Institute of Preventive Pediatrics, TUM Department of Sport and Health Sciences, Technical University of Munich, Munich, Germany

P.61 Impact of kidney transplantation on arterial reservoir-wave analysis

Miss Nadège Côté[1,2], Miss Emy Philibert[1,2], Miss Mathilde Paré[1,2], Dr Rémi Goupil[3], PhD Catherine Fortier[1,2,4], PhD Martin G. Schultz[5], PhD James E. Sharman[5], Dr Mohsen Agharazii[1,2]

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P.62 Assessment of isoflavone and ethanolic extract of Inonotus obliquus on experimentally induced diabetes.

Mr Kingsley Duru[1], Dr Cara Hildreth[1], Prof Alberto P. Avolio[1], Prof Jacqueline K. Phillips[1], Dr Mark Butlin[1]
[1]Macquarie University, Eastwood, Australia

P.63 Sarcopenia and atherosclerotic occlusive disease: how much we know and what we need to know about this association?

Joana Ferreira[1,2,3], Alexandre Carneiro[4], Pedro Cunha[2,3,5,6], Armando Mansilha[7,8], Isabel Vila[2,3,5,6], Cristina Cunha[2,3,5,6], Cristina Silva[2,3,5,6], Adhemar Longatto-Filho[2,6,9,10,11], Maria Correia-Neves[2,9], Gustavo Soutinho[12], Luís Meira-Machado[13], Amilcar Mesquita[1], Jorge Cotter[2,3,5,6]
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P.64 Active vitamin D treatment does not improve arterial stiffness and markers of cardio-renal risk in patients with type 2 diabetes and stage 3 chronic kidney disease: a randomised controlled trial.

Dr Nikolaos Fountoulakis[1], Dr Salma Ayis, Dr Anastasios Mangelis, Dr Angeliki Panagiotou, Dr Maria Flaquer, Mr Stanimir Stoilov, Dr Giuseppe Maltese, Professor GianCarlo Viberti, Dr Stephen Thomas, Professor Luigi Gnudi, Dr Janaka Karalliedde
[1]King's College London, United Kingdom

P.65 Increased biomarkers of endothelial dysfunction and thrombotic microenvironment in patients with autoimmune rheumatic disorders free from cardiovascular comorbidities

Dr Eleni Gavriilaki[1], Dr Panagiota Anyfanti[1], Professor Stella Douma[1], Professor Eugenia Gkaliagkousi[1]
[1]Aristotle University of Thessaloniki, Thessaloniki, Greece

P.66 Radial Artery systolic-diastolic pulse transit time after kidney transplantation

Miss Emy Philibert[1,2], PhD Hasan Obeid[1,2,4,5], Miss Mathilde Paré[1,2], Miss Nadège Côté[1,2], PhD Catherine Fortier[1,2,4,5], Dr Rémi Goupil[3], Dr Mohsen Agharazii[1,2]
[1]CHU de Québec Research Center, L'Hôtel-Dieu de Québec Hospital, Québec, Canada, [2]Division of Nephrology, Faculty of Medicine, Université Laval, Québec, Canada, [3]Hôpital du Sacré-Cœur de Montréal, Montréal, Canada, [4]INSERM U-970, Paris Cardiovascular research Center (PARCC), Paris, France, [5]AP-HP, Pharmacology Unit, Hôpital Européen Georges Pompidou, Université de Paris, Paris, France

P.67 The effects of chemotherapy on arterial inflammation assessed by 18FDG PET-CT in patients with Lymphoma

Constantinos Anagnostopoulos[2], Stavroula Giannouli[3], Nikolaos Ioakimidis[1], Paulos Kafouris[4], Iosif Koutagiar[1], Anastasia Sioni[5], Doctor Eirini Solomou[1], Dimitrios Terentes-Printzios[1], Dimitrios Tousoulis[1], Charalampos Vlachopoulos[1]
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P.68 WaveGraft – a novel endovascular device concept for restoring the natural arterial cushioning effect

Dr Florian Stefanov[1], Mr Dave Veerasingam[2], Dr Sarah Sayed[1], Dr Patrick Delassus[1], Mr Jonathan Bouchier-Hayes[1], Dr Liam Morris[1]
[1]Galway-Mayo Institute of Technology (GMIT), Galway, Ireland, [2] University Hospital Galway (UHG), Galway, Ireland

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Artery 21


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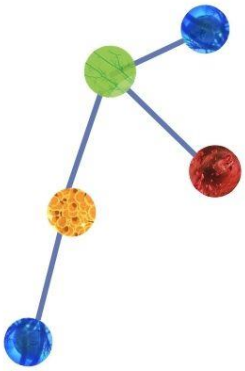
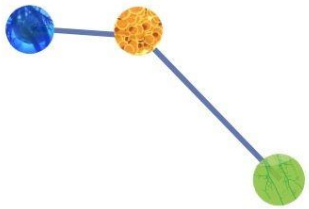
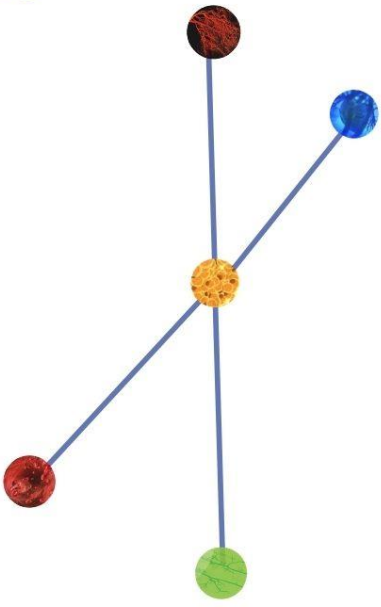
Thank you!





We would like to thank you for joining us in the first Artery Virtual Conference and we look forward to welcoming you in Nancy in 2021.



ARTERY 20

Virtual Conference

Book of Abstracts

ARTERY
Association for Research into Arterial Structure and Physiology

ARTERY 2019 Exchange Grant Presentation
Saturday 24 October 10.40 (CET)

Tri-layered constitutive modelling of the arterial wall: Evaluation of a new approach based on uniaxial testing of isolated layers

Mr Alessandro Giudici[1], Professor Ashraf W Khir[1], Dr Bart Spronck[2]
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[This abstract is the result of the work carried out thanks to the Artery 19 Research Travel Grant]

Background

Arterial wall mechanics is determined by the densities and structural arrangements of its constituents, which, however, are not uniform across the individual wall layers (intima, media, adventitia) (1). Although mechanical testing of individual layer samples provides mechanical insight, the contributions of the individual layers to whole arterial wall mechanics are not self-evident. Therefore, we aimed to develop a new modelling framework to combine experimental layer-specific mechanical information into a three-layered model of the arterial wall subjected to *in-vivo* realistic tension-inflation.

Methods

Circumferential and longitudinal arterial strips were isolated from the upper (UTA) and lower thoracic (LTA) lamb aorta (*n*=10) and subjected to uniaxial tensile testing. Samples were then separated into individual layers and testing was repeated on each layer. Layer-specific mechanical properties were modelled as described previously (2) and then combined into a tri-layered flat wall model matching the experimental whole wall stress-stretch relationship. Finally, the three-layered flat model was deformed into a cylindrical vessel, and physiological tension-inflation was simulated.

Results

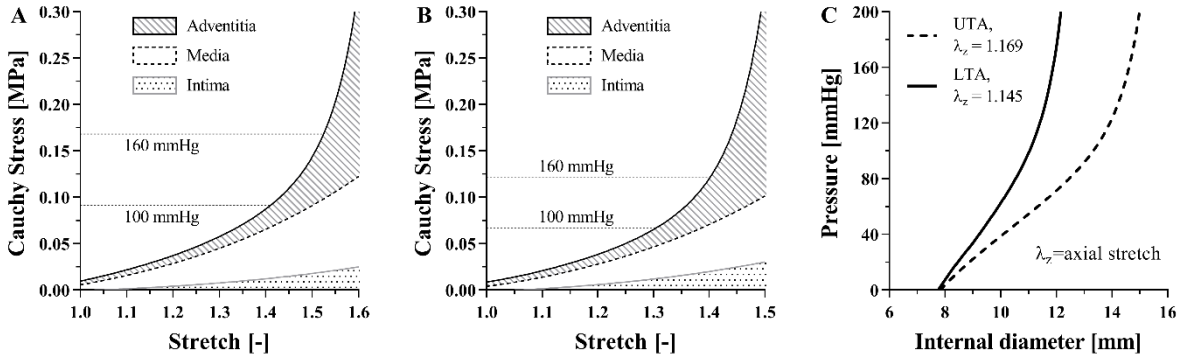


Figure panels A and B show typical stress-stretch contributions of the individual layers in the UTA and LTA; panel C shows the resulting pressure-diameter behaviour. At 100 mmHg, adventitial load bearing was approximately half of that of the media, while at 160 mmHg, the rapid stiffening of the adventitia equilibrated the stress partitioning among media and adventitia.

Conclusions

Our new approach provides valuable insight into the role of the individual arterial layers in arterial mechanics and may be useful for characterising arterial remodelling associated with ageing and pathologies.

Young Investigator Network Session: The role of social media in research
Saturday 24 October 11.20 (CET)

How young investigators can circulate and advertise their work through social media and other networks increasing the impact of their work?

Digital and social media is playing an ever-important role in how people consume news and information. As such, there has never been a better time for young investigators to build and develop their online profiles to maximise their career opportunities.

In this presentation, a brief overview is given on how to take the first steps in utilising digital media for professional purposes and which platforms are most likely to give the biggest impact for researchers when advertising and circulating their work.

Although aimed at early career investigators, the session is suitable for people at any stage in their career looking to build their digital presence.

YI 1.1

Aortic Impedance and Total Arterial Compliance from Regional Pulse Wave Velocities

Ms Vasiliki Bikia[1], Mr Georgios Rovas[1], Ms Stamatia Pagoulatou[1], Prof. Nikolaos Stergiopulos[1]
[1]*École polytechnique fédérale de Lausanne*

Background. In-vivo assessment of aortic characteristic impedance (Z_{ao}) and total arterial compliance (C_T) has been hampered by the need for invasive methods to access simultaneous recordings of aortic pressure and flow, wall thickness, and cross-sectional area. In contrast, regional pulse wave velocity (PWV) measurements are noninvasive and clinically available. Given that PWV is strongly related to aortic stiffness (1), we assume that carotid-to-femoral PWV (cfPWV) and carotid-to-radial PWV (crPWV) may contain sufficient information to evaluate the elasticity of the ascending aorta. Concretely, here, we present a noninvasive regression method for estimating Z_{ao} and C_T using cuff pressure, cfPWV, and crPWV.

Methods. Gradient Boosting is employed for predicting Z_{ao}, and C_T. The regressors are trained/tested using a pool of virtual subjects (n=4833) generated from a previously validated in-silico model (2). The cross validation is performed using a 10-fold cross-validation (3).The population used has been previously generated (4) and reflects a wide range of hemodynamical properties and states.

Results. Predictions had a high accuracy (Figure1) achieving a normalized-RMSE equal to 6.24±1.19 % (r=0.85,p<0.001) for Z_{ao}, and 4.38±0.36 % (r=0.97,p<0.001) for C_T, respectively. High errors were reported for high values of Z_{ao} due to the limited amount of similar data.

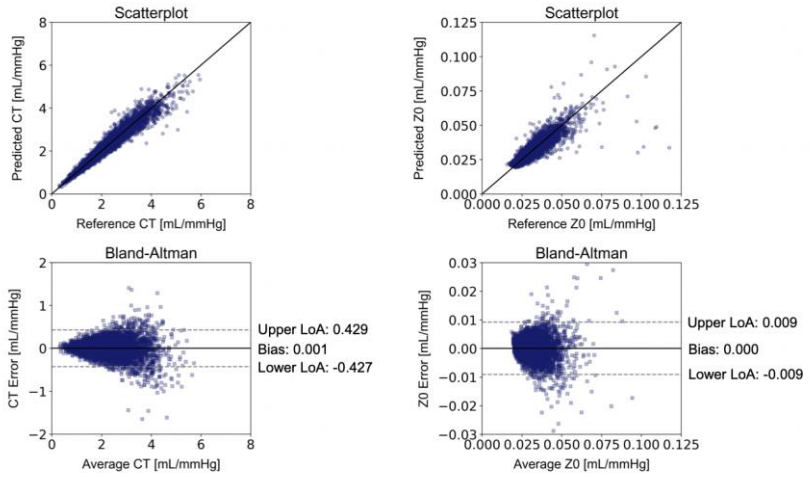


Figure 1. Comparison of the estimated values with the reference values. Scatterplot and Bland-Altman plot for the predicted Z_{ao} (left panel). Scatterplot and Bland-Altman plot for the predicted C_T (right panel). Solid line represents equality. Limits of agreement (LoA), within which 95% of errors are expected to lie, are defined by the two horizontal dashed lines.

Conclusions. The proposed approach constitutes a step forward to noninvasive screening of elastic vascular properties in human by exploiting easily obtained measurements. This study could introduce a valuable tool for assessing aortic stiffness reducing the cost and the complexity of the required measuring techniques. Clinical evaluation is required to validate the method in-vivo.

YI 1.2

Ideal cardiovascular health score declines from adolescence to emerging adulthood

Dr Chloe Park[1], Dr Siana Jones[1], Miss Suzanne Williams[1], Mrs Alicja Rapala[1], Ms Hannah Taylor[1], Dr Laura Howe[2], Dr Abigail Fraser[2], Professor Nish Chaturvedi[1], Professor Alun Hughes[1]
[1]University College London, [2]University of Bristol

Purpose: To define and compare cardiovascular (CV) health scores (CHS) from adolescence (17yrs) to emerging adulthood (24yrs) using longitudinal data from a large British birth cohort.

Methods: 3142 participants from the Avon Longitudinal Study of Parents and Children (ALSPAC) study attended clinical investigations at 17.8±0.4yrs and 24.0±0.8yrs (38% male). CV health was assessed using smoking status, body mass index (BMI), plasma glucose, cholesterol, sitting brachial blood pressure, left ventricle (LV) hypertrophy, arterial stiffness (carotid-to-femoral pulse wave velocity) and atherosclerosis (carotid intima-media thickness) metrics. Prevalence was stratified into poor (0_points), intermediate (1_point) and ideal (2_points) health categories and a composite, individual-level CHS for all 8 metrics was calculated (total range, 0–16 points). Prevalence of ideal health was assessed using ANOVA and linear mixed modelling assessed age ##sex modifications.

Results: Overall CHS was high at 17yrs but from 17-24yrs the proportion of ideal scores decreased for all metrics, in both sexes (Table). The average overall CHS decreased from 14.97±1.1 to 13.99±1.4 in males (p<0.0001) and 14.82±1.2 to 14.28±1.4 in females (p<0.0001, age##sex p=0.0001). Significant sex differences were observed in the proportion of individuals with ideal health at both ages, with males having a higher CHS than females at 17yrs but a lower CHS at 24yrs.

	Male							Female							Sex differences		
Age	**17 yrs**			**24 yrs**				**17 yrs**			**24 yrs**				**17**	**24**	**Age## sex**
Score	**0**	**1**	**2**	**0**	**1**	**2**	**p**	**0**	**1**	**2**	**0**	**1**	**2**	**p**	**p**	**p**	**p**
Smoking	22.6	19.3	58.1	27.8	35.6	36.6	<0.0001	27.8	24.3	47.9	25.7	36.5	37.8	<0.0001	<0.0001	0.29	<0.0001
BMI	4.9	13.3	82.1	10.4	30.0	59.6	<0.0001	6.7	15.4	77.9	14.6	21.5	63.8	<0.0001	0.006	0.97	0.02
Glucose	0.4	9.1	90.5	0.6	34.7	64.7	<0.0001	0.3	2.6	97.1	0.4	16.1	83.5	<0.0001	<0.0001	<0.0001	<0.0001
Cholesterol	0.0	1.5	98.5	2.1	11.7	86.2	<0.0001	0.1	4.5	95.4	3.0	13.6	83.3	<0.0001	0.002	0.07	0.14
Blood Pressure	0.2	0.5	99.3	0.6	5.4	94.0	0.0002	0.3	1.7	98.0	0.7	4.1	95.2	<0.0001	0.001	0.3	0.0001
LV hypertrophy	0.6	0.6	98.9	1.4	3.7	94.8	<0.0001	0.5	1.6	97.8	1.1	3.8	95.1	<0.0001	0.43	0.75	0.2
Arterial Stiffness	0.5	0.9	98.6	4.8	6.7	88.5	<0.0001	0.0	0.4	99.6	1.8	2.9	95.4	<0.0001	0.01	<0.0001	<0.0001
Atherosclerosis	0.0	0.9	99.1	0.0	1.2	98.8	0.0005	0.0	0.3	99.7	0.0	0.4	99.6	0.9	0.07	0.04	0.6
Average CHS	**14.97 ±1.1**			**13.99 ±1.4**			**<0.0001**	**14.82 ±1.2**			**14.28 ±1.4**			**<0.0001**	**0.01**	**<0.0001**	**<0.0001**
Data are % of participants in each category for each risk factor. O=poor, 1=intermediate and 2=ideal. Age##sex p value for modification.																	

Conclusions: Despite being relatively early in the life-course, CV health declines from 17yrs to 24yrs in both sexes, and more substantially in males. Emerging adulthood is a distinct period of lifestyle change and an important time to control CV risk factors to improve future CV health.

YI 1.3

Retinal microvascular calibers and incident depressive symptoms: The Multi-Ethnic Study of Atherosclerosis

ms April C. E. van Gennip[1], ms Sanaz Sedeghat[2], ms Mercedes R. Carnethon[2], ms Norrina B. Allen[2], ms Barbara E. K. Klein[3], ms Mary Frances Cotch[4], ms Diana A. Chirinos[2], mr Coen D. A. Stehouwer[1], mr Thomas T. van Sloten[1]
[1]Department of Internal Medicine, Cardiovascular Research Institute Maastricht, Maastricht University Medical Centre, [2]Department of Preventive Medicine, Feinberg School of Medicine, Northwestern University, [3]Ocular Epidemiology, University of Wisconsin-Madison, [4]Division of Epidemiology and Clinical Applications, Intramural Research Program, National Eye Institute, National Institutes of Health

Background
Cerebral microvascular dysfunction may contribute to depression via disruption of brain structures involved in mood regulation, but evidence is scarce. The retina allows for direct visualisation of a microvascular bed that shares anatomical and physiological similarities with the cerebral microvasculature. We investigated the association between baseline central retinal arteriolar and venular calibers (CRAE and CRVE) and 7.8-year change of CRAE and CRVE and incident depressive symptoms.

Methods
Longitudinal data are from the Multi-Ethnic Study of Atherosclerosis (MESA) of 3,999 participants (62.3±9.7 years; 48.2% women; 26.6% black) without depressive symptoms at baseline. Presence of depressive symptoms, defined as Centre for Epidemiological Studies Depression Scale score ≥16 and/or use of antidepressant medication, was determined in 2002-2004 (baseline, MESA exam 2) and at three follow-up examinations every 1.5-2 years thereafter. Fundus photography was performed at MESA exam 2 and exam 5 after a mean of 7.8 years.

Results
After a mean follow-up of 6.1 years, 21.7% (n=869) had incident depressive symptoms. After adjustment for socio-demographic, lifestyle and cardiovascular factors, one SD larger baseline CRVE (21.8 µm) was associated with a higher risk of depressive symptoms (hazard ratio: 1.10;95% confidence interval:1.02-1.18), but one SD larger baseline CRAE (14.1 µm) was not (hazard ratio: 1.05; 0.98-1.13). Neither 7.8-year change of CRAE nor CRVE were associated with depressive symptoms (odds ratios: 1.06; 0.90-1.24, and 1.06; 0.91-1.23, respectively).

Conclusions
Larger baseline CRVE is associated with a higher incidence of depressive symptoms. This might support the hypothesis that cerebral microvascular dysfunction contributes to the development of depression.

YI 1.4

Increases in Circulating Trimethylamine-N-oxide Contribute to the Development of Age-Related Aortic Stiffness in Humans and Mice

Abigail G Casso[1], Rachel A Gioscia-Ryan[1], Zachary J Sapinsley[1], Nicholas S VanDongen[1], Amy E Bazzoni[1], Andrew P Neilson[2], Melanie C Zigler[1], Kevin P Davy[3], Douglas R Seals[1], Vienna E Brunt[1]
[1]University Of Colorado Boulder, [2]North Carolina State University, [3]Virginia Tech

Age-related increases in aortic stiffness, assessed by pulse wave velocity (PWV), predict cardiovascular (CV)-related mortality, but the upstream drivers are incompletely understood.

Purpose: To determine if higher circulating levels of the gut microbiome-derived metabolite trimethylamine-N-oxide (TMAO) contribute to age-related aortic stiffening.

Methods and Results: Plasma TMAO concentrations were higher in healthy middle-aged-to-older (45-79y; N=83) vs. young (18-27y; N=14) humans (6.3±0.6 vs. 1.8±0.3μM; p<0.01) and positively related to carotid-femoral (c-f)PWV (r^2=0.15, p<0.0001). To determine the role of TMAO in established age-related aortic stiffness, we supplemented old mice (27mo; N=12-16/group) with 1% 3,3-dimethyl-1-butanol (DMB; suppresses microbiota-dependent TMAO production) in drinking water for 8-10 weeks vs. normal drinking water (control). Relative to young mice (3mo; N=23), old mice had higher aortic (a)PWV (412±17 vs. 349±11cm/s; p<0.01), but DMB had no effect on aPWV (p=0.58 vs. control) despite suppressing plasma TMAO (control: 8.7±6.3 vs. DMB: 4.3±1.2µM, p=0.07) to young levels (3.8±2.6µM). Next, to determine if TMAO contributes to the *development* of aortic stiffening, we initiated DMB at mid-life (18mo; i.e., before the onset of stiffening; N=8-21/age/treatment). aPWV was similar between young and 18 month-old mice (363±5cm/s; p=0.58), but increased progressively with age in control mice (24mo: 401±13cm/s, p=0.03 vs. young; 27mo: 442±10cm/s, p<0.001 vs. young), whereas age-related increases in PWV were considerably attenuated by DMB (24mo: 359±9cm/s; 27mo: 388±10cm/s, both p<0.01 vs. control).

Conclusions: Age-related increases in TMAO contribute to the development of aortic stiffness. TMAO-targeted interventions initiated in mid-life may prevent/delay age-related aortic stiffening and reduce CV risk.

Funding: HL134887-02S1, AG060884, HL140875, AG000279.

YI 1.5

Ten years of ageing in the middle-aged does not increase input impedance or wave reflection - insights from the Asklepios Study.

Daimé Campos Arias[1], Marc L. De Buyzere[2], Julio A. Chirinos[3,4], Ernst R. Rietzschel[2,5], Patrick Segers[1]
[1]IBiTech, Ghent University, [2]Cardiology Department, Ghent University Hospital, [3]Division of Cardiovascular Medicine, Hospital of the University of Pennsylvania, [4]Perelman Center for Advanced Medicine, University of Pennsylvania, [5]Biobanking and Cardiovascular Epidemiology, Ghent University Hospital

Background

The changes experienced by the arterial system due to ageing are still incompletely understood. The aim of this study is to analyze the 10-year longitudinal evolution of input impedance parameters and wave reflection indices in a middle-aged population, and how these effective changes compare to what was anticipated from a previous cross-sectional study [1].

Methods

The Asklepios study is a longitudinal population study including 2026 apparently healthy middle-aged subjects at inclusion (52% females), who underwent two rounds of measurements of carotid pressure (applanation tonometry) and central flow (ultrasound), with a follow-up time of 10.14±1.39 years. Subjects were classified into half-decades of age (35-40, 41-45, 46-50, 51-56) according to their age at baseline. Input impedance and wave reflection parameters were derived using frequency-domain methods. Arterial compliance was estimated from the pulse pressure method (C_{PPM}). Linear mixed-effects models were used to evaluate the longitudinal trajectories of the parameters.

Results

Figure 1 shows the predicted longitudinal trajectories and rates of change per decade of input impedance parameters and wave reflection indices. Longitudinal changes of some variables opposed to what was anticipated from the cross-sectional data. C_{PPM} increased with ageing mainly in younger males. Characteristic impedance decreased with age in younger subjects while increased for the older subjects in the study. Wave reflection decreased with ageing, whereas resistance increased in women and decreased in men.

Conclusions

We conclude that the effective impact of aging on arterial system properties is not well reflected by cross-sectional studies.

(figures on next page)

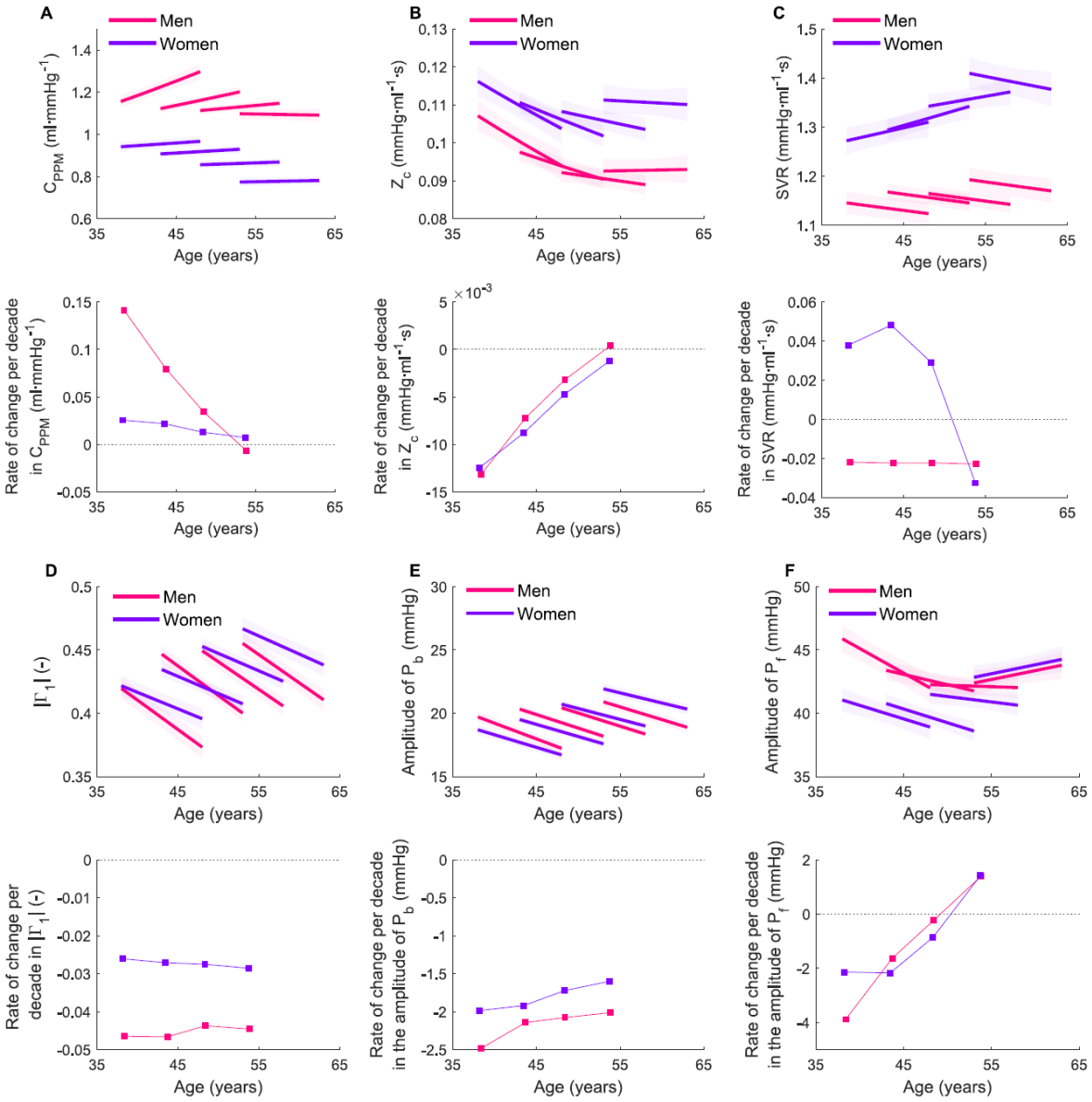


Figure 1. Predicted longitudinal trajectories and rate of change per decade in impedance parameters (A-C) and wave reflection indices (D-F), for men and women and per age category. Shaded area represents the 95% confidence intervals for the predictions. Regression models included follow-up time, entry age, height, weight, heart rate, or mean arterial pressure as potential covariates. C_{PPM}: total arterial compliance; Z_c: characteristic impedance; SVR: systemic vascular resistance; $|\Gamma_1|$: amplitude of the reflection coefficient at the heart frequency; P_b: backward pressure wave; P_f: forward pressure wave.

YI 1.6

Flow mediated slowing of pulse wave velocity as a measure of endothelial function

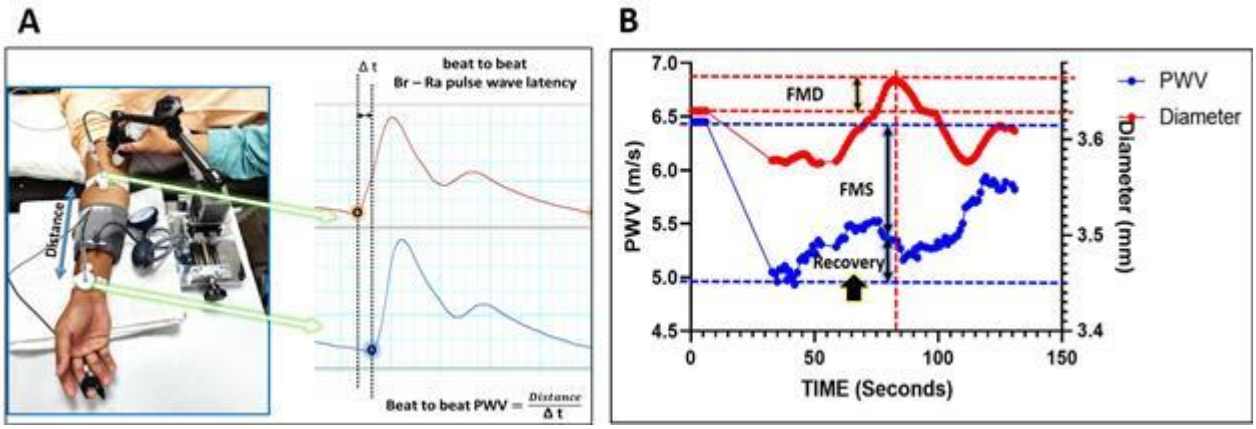
Anju Sharma[1], Dinu.S. Chandran[1], Ashok Jaryal[1], Kishore K Deepak[1]
[1]*Aiims New Delhi*

Purpose
Ultrasonographic measurement of flow mediated dilatation (FMD) of brachial artery is the gold standard non-invasive technique to measure endothelial function. However, measurement of FMD is technically cumbersome, operator dependent and requires trained manpower, all of which limits its clinical utility. Flow mediated slowing (FMS) of regional arterial pulse wave velocity (PWV) has been proposed as a feasible, operator-independent alternative to ultrasonographically measured FMD (1,2). We investigated the temporal correlation between brachial artery FMD and simultaneously recorded FMS of brachial-radial PWV in healthy volunteers.

Methods
Twenty-three healthy volunteers, involving 15 males and 8 females with age 26.39 ± 4.41 participated in the study. Endothelial function was assessed by measuring FMS of brachial-radial PWV using two-point applanation tonometry and simultaneously performed ultrasonographic imaging of brachial artery to record FMD. After 1-minute of baseline recording, distal supra-systolic arterial occlusion was produced by inflating a sphygmomanometer cuff to 250 mmHg for 5 minutes. During the phase of reactive hyperemia that follows the release of distal arterial occlusion, both the parameters were simultaneously recorded for a period of 6-minutes.

Results
Flow mediated slowing of pulse wave velocity didn't correlate with simultaneously recorded FMD. Recovery of PWV coinciding with peak FMD showed strong positive correlation with FMD (r = 0.60, p = 0.002). Time averaged recovery of PWV during the period 60-90s post release of occlusion correlated with FMD (r =0.46, p = 0.0253).

Conclusions
Recovery of brachial-radial PWV during post-occlusive reactive hyperemia could be used as a feasible, imaging independent alternative to ultrasonographically measured FMD.



YI 1.7

Transmural quantification of murine vascular smooth muscle cell density distribution from 3D microscopy images

Phd Koen W.F. van der Laan[1,2], PhD Koen D. Reesink[1,2], PhD, MD Myrthe M. van der Bruggen[1,2], PhD Armand M.G. Jaminon[1,3], PhD Remco T.A. Megens[1,2,4], PhD Leon J. Schurgers[1,3], Phd, MD Tammo Delhaas[1,2], PhD Bart Spronck[1,2,5]
[1]CARIM School for Cardiovascular Diseases, Maastricht University, [2]Department of Biomedical Engineering, Maastricht Univeristy, [3]Department of Biochemistry, Maastricht University, [4]Institute for Cardiovascular Prevention, Ludwig Maximilians University (LMU),, [5]Department of Biomedical Engineering, School of Engineering & Applied Science, Yale University

Purpose
Investigating the biomechanical role of smooth muscle cells (SMCs) in arteries requires knowledge of their structural distributions. Compared to histology, 3D microscopy offers non-destructive *ex vivo* imaging under realistic conditions [1]. Robust 3D segmentation of SMCs, however, is challenging. We propose a method for automatic SMC quantification, and assessed its potential using a murine SMC apoptosis model.

Methods
After euthanasia, carotid arteries (control and with induced SMC apoptosis: SM22α-hDTR [2]) were excised and mounted between micropipettes (**Figure A**). Nuclei were stained with SYTO41. Arteries were imaged using two-photon microscopy [1], while stretched to *in vivo* length and pressurised to 100 mmHg (**B**). Image stacks were processed as follows: 1) deconvolution; 2) nuclei segmentation using vesselness filtering [3,4] (**C**); 3) cylindrical coordinate system identification; 4) splitting of coincident nuclei, based on cores defined from groups of neighbouring voxels with similar orientations [3] (**D,E**); 5) cylindrical coordinate system re-identification; and 6) cell density-distribution quantification (**F**). Segmentation performance was assessed by comparing with manual cell counts.

Results
Figure E demonstrates the method's ability to split undersegmented coinciding nuclei. Cell counts were lower in SM22α-hDTR than in control; algorithm-derived counts were comparable to manual (**F**). The control sample showed multiple SMC layers, while the SM22α-hDTR sample showed a single SMC layer (**F**), which was confirmed visually.

Conclusion
We developed a precise tool to quantify SMC distributions in *ex vivo* murine arteries, to facilitate quantitative modelling of SMC biomechanics. We intend to expand the current approach to address cell orientation, shape, and size.

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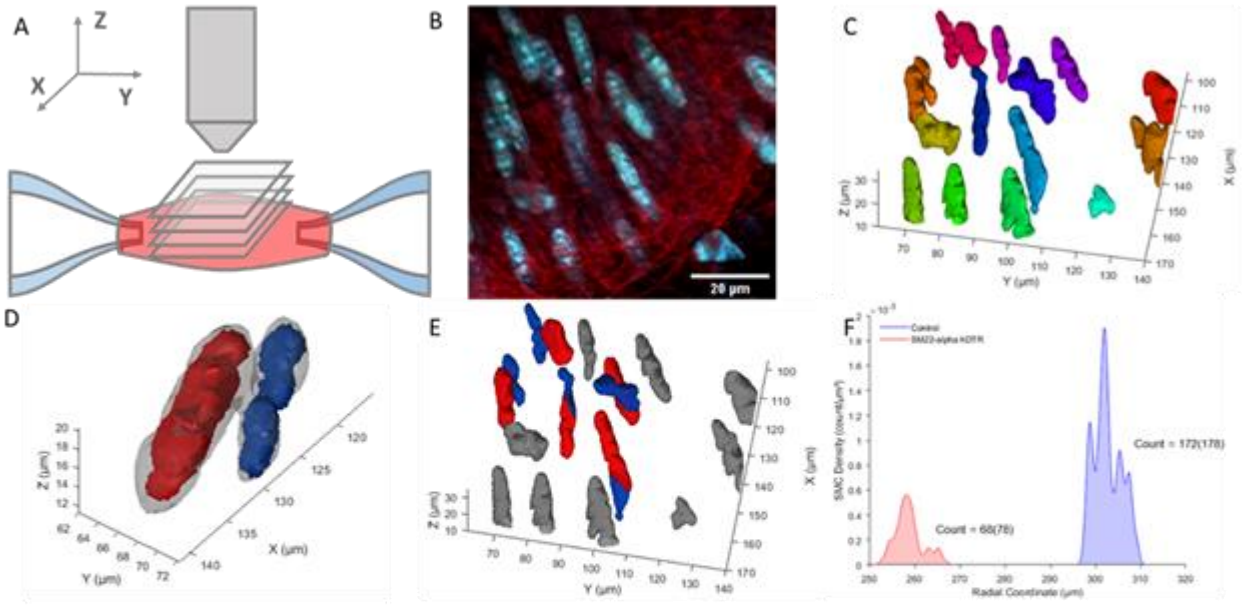

(**A**) Imaging set-up illustrating acquisition of z-stack of slices. (**B**) Example slice of 3D stack; cell nuclei are shown in blue while elastin fibres are shown in red. (**C**) Segmentation results from vesselness filtering of example image stack, colours indicate separated nuclei (step 2, Methods). (**D**) Coinciding nuclei, corresponding with the left orange nuclei in **C**, shown in grey, with cell cores shown in red and blue (step 4, Methods). (**E**) Coinciding nuclei splitting results of nuclei shown in **C**. Non-split nuclei are shown in grey, while split nuclei are shown in red and blue. (**F**) Transmural SMC densities and cell counts for one control and one SMC apoptosis sample; manual cell counts are given between parentheses.

YI 1.8

A computational model-based study on the effect of abdominal aortic aneurysm on pulse wave morphology

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Background
Abdominal aortic aneurysm (AAA) is usually asymptomatic and has an extremely high mortality if rupture occurs. Therefore, early detection and intervention are important. However, AAA is most often detected as an accidental finding during clinical imaging for other purposes (1). Considering that AAA has a systemic impact on the biophysical properties of the cardiovascular system, pulse-wave-based diagnosis of AAA may be a potential approach for effective detection. There have been only a few studies of pulse wave propagation with AAA by using computational modelling or hydraulic simulators (2). This study aims to provide some basic insights for pulse-wave-based diagnosis of AAA using computational modelling.

Methods
We simulated blood flow in the larger systemic arteries using the 65-year-old baseline model from the pulse wave database in Charlton *et al*. (3) (see figure, left). The influence on pulse waveforms of AAA morphology (including shape, maximum diameter and length) was simulated by adjusting the geometry of the abdominal aorta Ⅳ. In addition, the influence of the local AAA stiffness and global arterial stiffness (represented by carotid-femoral pulse wave velocity) was investigated by varying the relevant model parameters.

Results
Results show that maximum diameter is the dominant morphological factor in reshaping the pulse waveform, introducing considerable changes in pulse wave indices. Moreover, both local and global arterial stiffness have a considerable impact on wave morphology in the presence of AAA (figure, right).

Conclusions
AAA produces considerable changes in pulse wave morphology that could be used for AAA detection by using pulse wave analysis.

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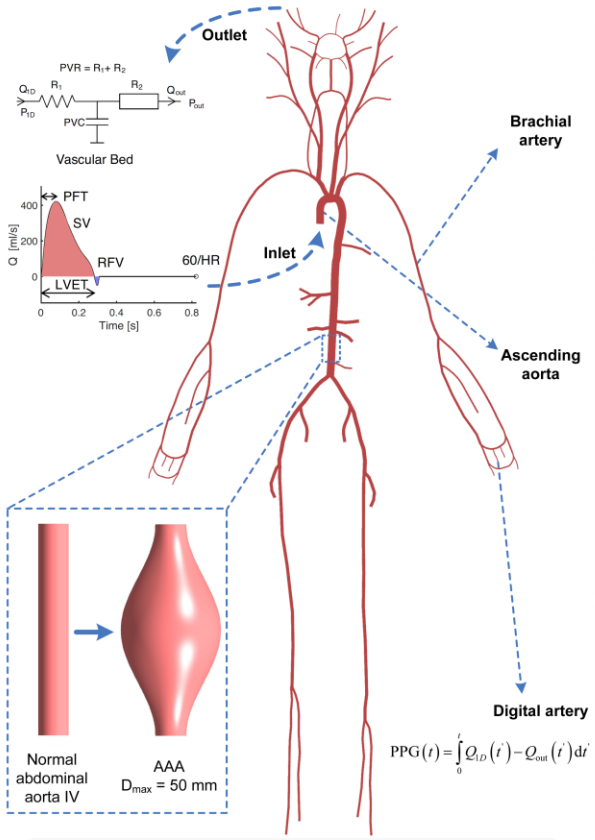
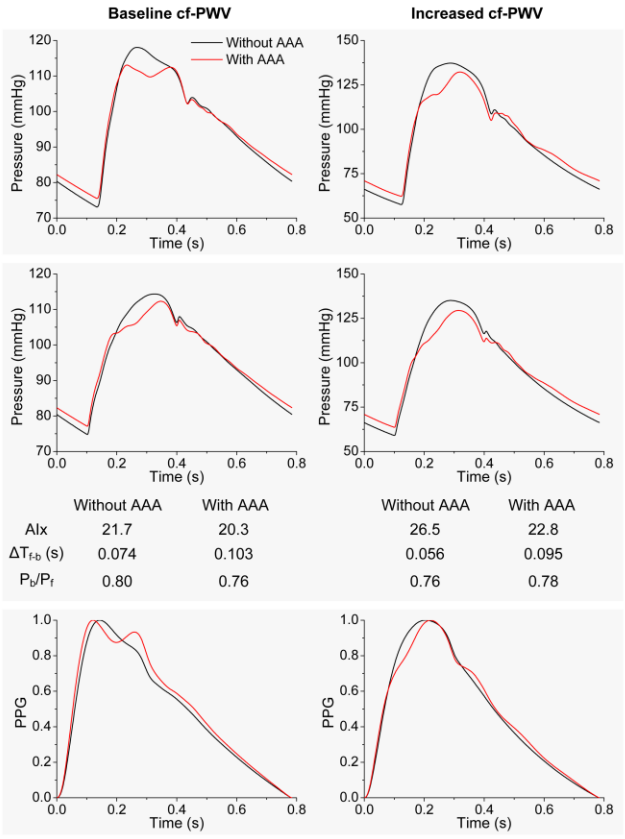


Blood flow in the larger systemic arteries was simulated by using the 65-year-old baseline model from the pulse wave database in Charlton *et al. Am J Physiol Heart Circ Physiol* 317: H1062–H1085, 2019.



	Without AAA	With AAA	Without AAA	With AAA
AIx	21.7	20.3	26.5	22.8
$\Delta T_{f\text{-}b}$ (s)	0.074	0.103	0.056	0.095
P_b/P_f	0.80	0.76	0.76	0.78

Pressure and PPG waves with (red lines) and without (black lines) the AAA. AIx, augmentation index; $\Delta T_{f\text{-}b}$, time delay between backward and forward pressure waves; P_b/P_f, ratio of the amplitudes of backward and forward pressure waves.

YI 2.1

Pulse wave velocity estimation from the radial pulse waveform using Gaussian process regression: A machine learning based study

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Objective and Motivation
Pulse wave velocity (PWV) is known to be associated with vascular ageing, a risk factor for cardiovascular disease (CVD) (1). The European gold standard measurement of PWV requires an experienced operator to measure pulse waveforms at multiple sites, sometimes together with an electrocardiogram (2,3). This study aims to estimate PWV from the radial pulse waveform using machine learning.

Methods
Radial pulse waveforms and carotid-femoral PWVs were acquired in 3,082 unselected twins (https://twinsuk.ac.uk). 14 fiducial points on each pulse waveform were extracted using an in-house algorithm (4). LASSO regression and principal component analysis (PCA) were used to identify the key features (timing and magnitude of the fiducial points) associated with PWV and exclude outliers. Finally, Gaussian process regression was used to estimate the PWV based on those key features only.

Results
Results show that PWV can be estimated from the radial pulse waveform only with an overall root mean squared error (RMSE) of 1.82 m/s (Fig. 1A). Most of the measured PWV values were within the 95% confidence interval range of the estimated PWV. The difference between measured and estimated PWV values increased with the increasing PWV. PWV estimation on a subgroup of twins with a healthy range of blood pressure and PWV values (5) was achieved with a RMSE of 1.38 m/s (Fig. 1B).

Conclusion
In this proof-of-concept study we have shown the possibility of estimating PWV from the radial pulse waveform using machine learning. This approach could make CVD detection more accessible to the wider population.

YI 2.2

Spontaneous cardiovascular ageing of C57Bl6 mice results in the development of aortic stiffness prior to peripheral blood pressure alterations.

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[1]*University Of Antwerp*

Background: Although generally assumed to be an adaptive response to increased blood pressure (BP), arterial stiffness is now recognized as an independent predictor of cardiovascular (CV) events (1). Moreover it precedes hypertension in at least two mouse models (2, 3). Therefore, the present study aims to investigate the temporal development of aortic stiffening and peripheral blood pressure (BP) alterations in spontaneously ageing mice.

Methods: A longitudinal cardiovascular characterization of spontaneously ageing C57Bl6 mice (2, 4, 6, 9, 12 and 24-month old) (male, n>8) was performed. This includes *in vivo* analysis of peripheral BP (Coda) and aortic pulse wave velocity (aPWV, Vevo2100), combined with *ex vivo* aortic studies of isometric reactivity (organ baths) and aortic stiffness measurements (Peterson modulus, Ep) in the Rodent Oscillatory Tension set-up for Arterial Compliance (ROTSAC).

Results: *In vivo* and *ex vivo* characterisation confirms that aortic stiffness precedes peripheral BP alterations in spontaneously ageing C57Bl6 mice, with significantly increased aPWV from 6 month of age (Fig A), whereas peripheral BP measurement only shows elevated pulse pressure in 24 month old mice (30% increase vs. all other ages, Fig.B). *Ex vivo* investigation of the thoracic aorta further reveals that the aortic stiffening is both contraction-independent (Fig.C) and dependent (Fig.D), since older mice display increased contractions to phenylephrine (PE) (Fig. E,F).

Conclusions. Spontaneously ageing C57Bl6 mice present with significant aortic stiffness by 6-months of age, which is both contraction-dependent and independent in origin. Aortic stiffness thereby precedes the development of peripheral BP alterations by 18 months.

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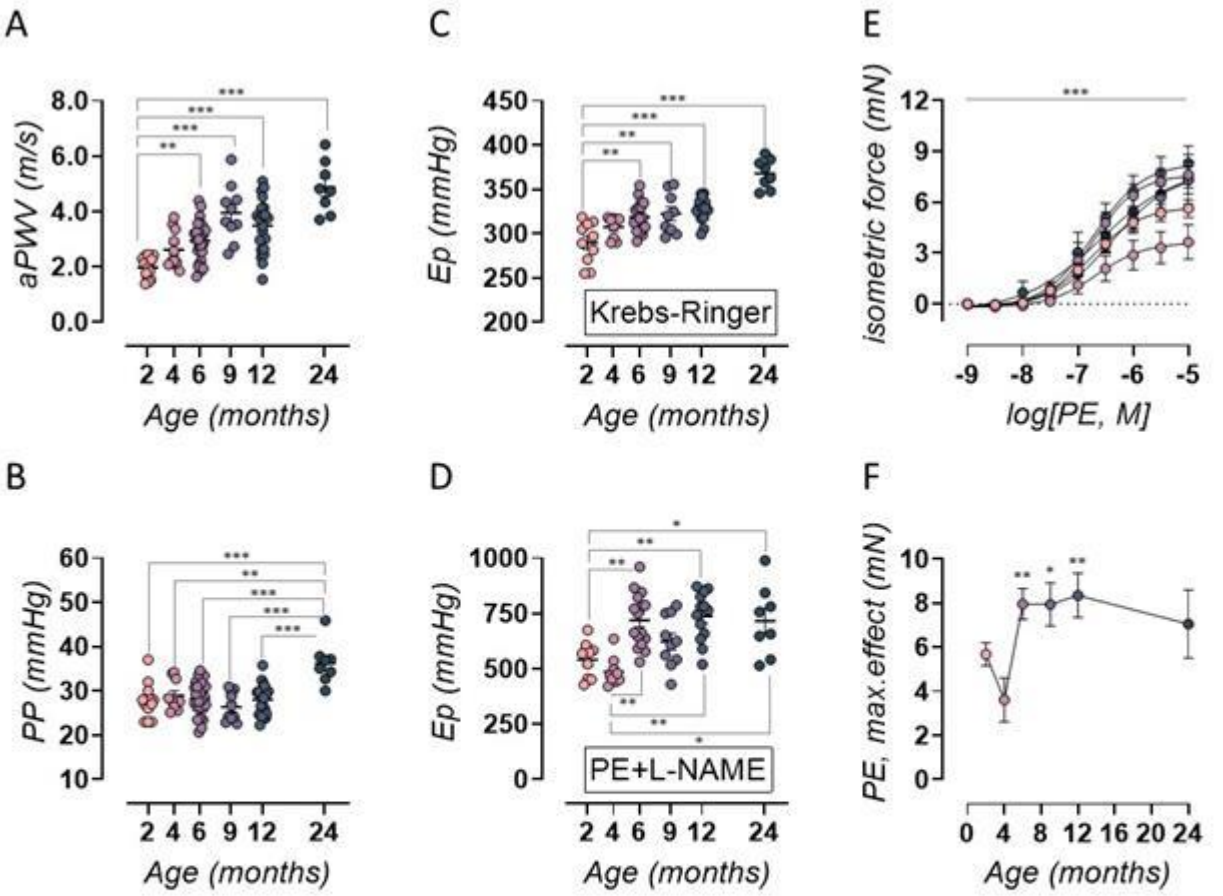

Significantly increased aPWV after 6-months (**A**) and increased peripheral pulse pressure after 18-months (**B**)

Isobaric *ex vivo* aortic stiffness in uncontracted (Krebs-Ringer, **C**) and maximally contracted (PE+L-NAME, **D**) aortic rings

Heightened contractions using a PE dose-response curve of *ex vivo* aortic rings (**E**) and its maximal effect (**F**).

YI 2.3

Methylglyoxal, 3-deoxyglucosone, and glyoxal – precursors of advanced glycation endproducts – are not independently associated with indices of carotid stiffness: The Maastricht Study

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Background: Arterial stiffness is a strong predictor of cardiovascular diseases and all-cause mortality (1). Increased fasting plasma concentrations of highly reactive dicarbonyl compounds – methylglyoxal (MGO), 3-deoxyglucosone (3-DG), and/or glyoxal (GO) – may cause arterial stiffening via for mation of advanced glycation endproducts, triggering maladaptive responses in vascular tissue, e.g. elastin degradation and collagen cross-linking (2). We assessed the cross-sectional associations between MGO, 3-DG, and GO concentrations with local carotid stiffness measures (distensibility coefficient (cDC), radius-wall thickness ratio (cRWT), pulse wave velocity (cPWV), and Young's elastic modulus (cE) using standardized main variables.

Methods: Fasting dicarbonyl concentrations were determined by ultra-performance liquid chromatography tandem mass spectrometry in EDTA plasma collected from 2275 participants (age: 60±8 years, mean±SD; 49% women, 605 with type 2 diabetes mellitus) of the Maastricht Study (3), an observational, population-based cohort study. Cross-sectional associations were assessed using multivariable linear regression analysis adjusting for age, sex, mean arterial pressure (MAP), heart rate, lifestyle factors, and medication. Since arterial stiffness measures are intrinsically pressure dependent, we additionally assessed the associations with pressure-corrected counterparts (4), instead of statistically correcting for MAP.

Results: Fasting dicarbonyl concentrations were associated with arterial stiffening (smaller cDC; larger cPWV and cE) in most crude models, but not in adjusted models (Table 1). cRWT was associated with 3-DG, but only in the crude model. The use of pressure-corrected metrics did not materially change the final models.

Conclusion: Fasting plasma concentrations of either MGO, 3-DG, or GO are not independently associated with carotid stiffness in this cross-sectional analysis.

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Table 1. Associations between fasting plasma dicarbonyls and carotid stiffness measures

	Model	cDC β (95% CI)	cRWT β (95% CI)	cPWV β (95% CI)	cE β (95% CI)
MGO	0	**-0.104(-0.145;-0.063)**	0.011(-0.030;0.052)	**0.103(0.062;0.144)**	**0.099(0.059;0.140)**
	1	-0.032(-0.070;0.005)	0.010(-0.032;0.052)	0.032(-0.006;0.071)	0.033(-0.007;0.072)
	2	0.005(-0.029;0.039)	0.000(-0.042;0.042)	-0.004(-0.039;0.030)	-0.006(-0.042;0.030)
	3	0.003(-0.031;0.037)	0.000(-0.043;0.042)	-0.003(-0.038;0.032)	-0.005(-0.040;0.031)
3-DG	0	**-0.164(-0.205;-0.123)**	**0.067(0.026;0.108)**	**0.149(0.108;0.190)**	**0.171(0.131;0.212)**
	1	**-0.102(-0.154;-0.051)**	0.024(-0.033;0.082)	**0.075(0.023;0.127)**	**0.093(0.039;0.147)**
	2	-0.037(-0.084;0.009)	0.009(-0.049;0.066)	0.012(-0.036;0.060)	0.027(-0.023;0.076)
	3	-0.018(-0.065;0.029)	-0.004(-0.062;0.054)	-0.007(-0.056;0.041)	0.004(-0.045;0.054)
GO	0	**-0.054(-0.095;-0.013)**	-0.032(-0.074;0.009)	**0.054(0.012;0.095)**	0.038(-0.003;0.079)
	1	-0.015(-0.051;0.022)	-0.014(-0.055;0.026)	0.015(-0.022;0.052)	0.008(-0.030;0.046)
	2	0.002(-0.031;0.035)	-0.015(-0.056;0.025)	-0.002(-0.035;0.032)	-0.008(-0.042;0.027)
	3	-0.009(-0.042;0.024)	-0.013(-0.053;0.028)	0.011(-0.022;0.045)	0.005(-0.030;0.040)

Model 0: crude associations. Model 1: model 0 + age, sex, and glucose metabolism status. Model 2: model 1 + mean arterial pressure and mean heart rate, and anti-hypertensive drugs. Model 3: model 2 + body mass index, smoking status, physical activity, use of lipid-modifying drugs, fasting triglycerides and total-to-high-density lipoprotein cholesterol levels, alcohol use, history of cardiovascular disease, kidney function, and Dutch healthy diet score. MGO, methylglyoxal; 3DG, 3-deoxyglucosone; GO, glyoxal; cDC, carotid distensibility coefficient; cRWT, carotid radius-wall thickness ratio; cPWV, carotid pulse wave velocity; cE, carotid Young's elastic modulus. Significant associations ($p<0.05$) printed in bold.

YI 2.4

Neural baroreflex sensitivity and long-term effect of antihypertensive agents--a pharmacological substudy of the Paris Prospective Study III

Nicolas Danchin[5], Catherine Guibout[3,4], Xavier Jouven[3,4], Marie-Cécile Perier[3,4], Frederique Thomas[5], **Dr Catherine Fortier[1]**, Dr Jean-Philippe Empana[3,4], Dr Hakim Khettab[2], Dr Rosa-Maria Bruno[1,2], Dr Pierre Boutouyrie[1,2]
[1]INSERM, U970, Paris Cardiovascular Research Center, Cellular molecular and physiological mechanisms of heart failure (Team 7), [2]AP-HP, Pharmacology Unit, Hôpital Européen Georges Pompidou, Université de Paris, [3]INSERM U970, Paris Cardiovascular Research Centre (PARCC), University of Paris, [4]INSERM U970, Paris Cardiovascular Research Centre (PARCC), Integrative Epidemiology of Cardiovascular Disease (Team 4), [5]Preventive and Clinical Investigation Center (IPC)

Background/Objectives: The baroreflex is a crucial mechanism acutely modulating vascular tone and heart rate response to maintain blood pressure (BP) in an optimal range. A decrease in baroreflex sensitivity (BRS) is associated with ageing, and pathological conditions such as hypertension and diabetes. Antihypertensive agents are generally known to have beneficial effect on the BRS, however it is still uncertain if the effect is mediated through a more compliant arterial wall or a sympathoinhibitory action.

Methods: In the Paris Prospective Study III(1), spontaneous baroreflex, carotid stiffness and pharmacological drugs intake were available in 7967 adults (aged 55-75 years). The neural component of the baroreflex sensitivity (nBRS) was obtained with a cross-spectral analysis of variations in carotid distention rate and R-R intervals. Pharmacological classes were analysed according to the Anatomical Therapeutic Chemical (ATC) classification. Individuals with a BP lowering medication (BP-treated) were paired to non-BP treated individuals with a similar cardiovascular risk (controls) using a propensity score matching procedure (n=1182 pairs).

Results: Amongst pharmacological classes of BP lowering agents, only agents acting on the renin-angiotensin system (ACEi-ARB) were associated with nBRS (β=-0.08, p=0.045). Compared to their matched controls, ACEi-ARB users had lower nBRS (2.79±0.66 vs. 2.90±0.62, p=0.03). In multivariate analysis, ACEi-ARB remained significant (stdβ=-0.09, p=0.025) after adjustment for carotid stiffness (stdβ=0.25, p<0.001) and systolic pressure (stdβ=-0.20, p<0.001).

Conclusions: In this epidemiological study, ACEi-ARB were negatively associated with nBRS. This effect is independent of BP and stiffness, which may suggest an inhibition of sympathetic activity by ACEi-ARB.

YI 2.5

Direct measurement of stiffness index β of superficial arteries without blood pressure estimation

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[1]Department of Electrical Engineering, Indian Institute Of Technology Madras, [2]Healthcare Technology Innovation Centre, Indian Institute of Technology Madras

Background: Arterial stiffness index (β) is a clinically accepted vascular metric, calculated from arterial pressure and diameter obtained simultaneously from a single arterial site (1). Hence, accurate measurement of β can only be performed on arteries where pressure can be recorded along with the diameter. We present a method to evaluate β from superficial arteries using arterial force (F) and diameter (D) waveforms, employing mathematical models (shown below) exploiting the non-linear pressure-diameter relationship (2), without requiring absolute pressure.

Methods: Pilot functionality assessment was performed on eight participants (24±5 years). A custom-developed frequency-matched system, combining single-element ultrasound and force-sensing transducers, was used to measure D and F waveforms from the common carotid artery. A hemodynamic-loop was formed from these measures and optimised to eliminate viscous components, and evaluate the elastic stiffness index β. Traditional β-formula (2) yielded reference values for comparison.

$$f_e(t) = f_{mx} \times \left(\frac{e^{\beta\left(\frac{D(t)}{D_d}-1\right)} - 1}{e^{\beta\left(\frac{D_s}{D_d}-1\right)} - 1} \right)$$

Mathematical Model

$f_e(t)$: Viscosity eliminated arterial force
f_{mx} : Maxima of viscosity eliminated arterial force
β : Stiffness index
$D(t)$: Arterial diameter
D_s : Systolic diameter
D_d : Diastolic diameter

Results: The system captured high fidelity D and F waveforms, adequate for reliable β evaluation. Measured group-average β (4.7±0.8) was concurrent with literature. The measured β values statistically agreed (LoA = ±0.83 and bias = –0.32; non-significant $p > 0.05$) and strongly correlated ($r = 0.93$, $p < 0.001$) with the reference values. Further, they exhibited acceptable beat-to-beat repeatability (variation < 7%) and accuracy (RMSE = 0.53).

Conclusions: The proposed method demonstrated the functionality by estimating reliable carotid β. Its key advantage is the applicability to superficial arteries, especially from sites where direct pressure measurement is challenging. Further studies demonstrating its potential for clinical and research applications are underway.

YI 2.6

Comparison of cardiovascular disease primary prevention guidelines between Australia, England and the United States.

Dr Niamh Chapman[1], Dr Monique Breslin[1], Dr Sarah Lay-Flurrie[2], Dr Zhen Zhou[1], Prof. James Sharman[1], Prof. Mark Nelson[1], Prof Richard McManus[2]
[1]University Of Tasmania, 1Menzies Institute for Medical Research, [2]University of Oxford, 2Nuffield Department of Primary Care Health Sciences

Objective. Cardiovascular disease (CVD) primary prevention guidelines recommend absolute CVD risk estimation to guide blood pressure and lipid therapy recommendations but are inconsistent despite relying on similar evidence. This study aimed to compare the populations recommended for treatment according to guidelines in Australia, England and the United States.

Methods. Cross-sectional analysis of national health survey data from Australian, English and United States (n=4,056; n=2,994; n=2,943; respectively) adults aged ≥40 years. Participants were classified as recommended for therapy based on clinical characteristics denoting high risk and absolute CVD risk stratification according to each country's guidelines.[1-6] Agreement in therapy recommendation assessed by Kappa statistic.

Results: Agreement in therapy recommendation was minimal to weak (κ=0.35-0.54). Proportions recommended for either blood pressure or lipid lowering treatment ranged between 26-32%, 47-52% and 43-47% in Australia, England and United States. There was minimal to strong agreement in therapy recommendation according to clinical criteria (κ=0.38-0.83) and minimal to moderate agreement according to absolute CVD risk (κ=0.28-0.64) across guidelines.

Conclusion: Despite similar evidence apparently underpinning guidance, there is little agreement in the populations targeted for CVD primary prevention with Australia recommending far few people for treatment in comparison to England or the United States. This is due to differences in both clinical characteristics considered high risk and absolute CVD risk stratification. Whilst different countries may adopt different policies on the appropriate level of risk to target, these findings suggest a need to develop international consensus definition for high CVD risk in primary prevention guideline

P.01

Where does the reflected wave observed in the ascending aorta come from?

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[1]*Department of Mechanical and Aerospace Engineering, Brunel University London*

Background
Wave reflections play a major role in changing the shape of the pressure waveform. Reflections measured at the aortic root (AR) are thought to be due to the tapering of the aorta (1) and multiple reflection sites, however, there is no consensus on the source of those reflected waves. This research aims to better understand the origin of the reflected waves observed in AR.

Methods
A 1D computational model of arterial wave propagation was used to study the reflections in an arterial network that consists of 37 segments of large arteries (2). A pulse was inserted in 3 peripheral vessels (Figure1) and followed as it travelled back towards AR. A pressure ratio (PR) was described as the ratio between the pressure at AR to the inlet pressure to allow for comparisons between the effect of various reflected sites.

Results
The pulse wave lost its magnitude travelling back towards the heart. The pulse inserted from the iliac artery could hardly be observed in AR (Figure2), and only 1% of the waves' magnitude could be detected. PR of the wave inserted at the carotid artery is approximately 18 times larger than those generated at the iliac artery; both measured in the ascending aorta.

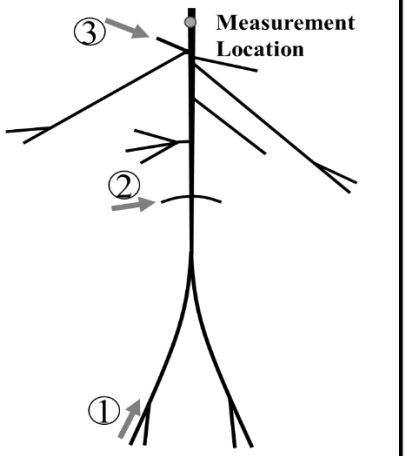

Figure 1. A schematic representation of the arterial model and locations of pulse insertions

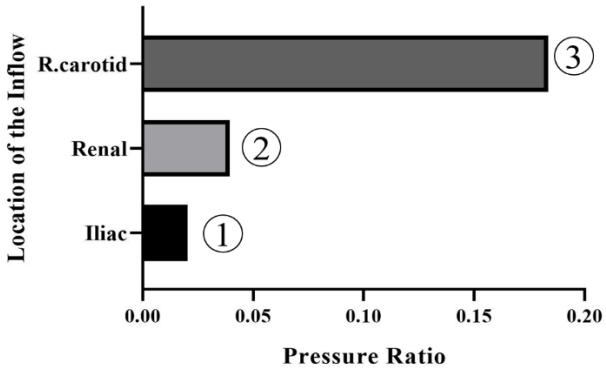

Figure 2. The ratio of input pressure prescribed from peripheral vessels to the pressure measured at the ascending aorta (PR)

Conclusion
Waves reflected from the carotid bifurcation and the cerebral circulation are more likely to be seen in AR in comparison to reflected sites such as renal and iliac arteries. Further work is warranted to establish the contribution of reflections generated from various sites along the arterial bed.

P.02

Differential ‘mediators’ of low-flow ‘mediated' constriction in healthy vs patients of ischemic heart disease

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[1]*All India Institute Of Medical Sciences, New Delhi*

Background – Low-flow mediated constriction (LFMC) has emerged as a non-invasive tool for assessment of endothelial dysfunction(1). There is insufficient data on association between change in artery diameter during occlusion with its possible stimulus; ‘low flow’ state. This study aims to evaluate the association between change in brachial-artery diameter during constriction with alterations in retrograde, anterograde and oscillatory flow profile in healthy subjects and patients with ischemic heart disease (IHD).

Methods – Brachial-artery responses to occlusion were assessed from artery diameter and blood flow using B-mode and pulsed-wave doppler ultrasound respectively in 89 patients with IHD and 29 healthy subjects. Change in anterograde, retrograde and net flow velocity, shear rate (AFV, RFV, NFV, ASR, RSR and NSR respectively) and oscillatory shear index (OSI) during forearm occlusion at 50mmHg above systolic pressure, from baseline was calculated.

Results – Diameter deceased significantly in healthy subjects and patients during occlusion compared to baseline. Interestingly, in stepwise forward-selection analysis, change in maximum AFV, ASR, RSV and RSR emerged in best fit model, explaining 76.2% of total variability in delta LFMC in IHD patients, with maximum contribution by ASR (70.4%). On the other hand, in healthy, change in maximum RFV, RSR, NFV and NSR emerged in best-fit model explaining 89.9% of total variability, of which 47% was by NSR and 33% by RSR.

Conclusion –Brachial-artery constriction during occlusion is ‘mediated’ by decrease in ASR in patients of IHD and decrease in NSR and increase in RSR in healthy, highlighting the possibility of differential ‘mediators’ of constriction in healthy vs diseased.

P.03

Local Pulse Wave Velocity Estimation using a Double Gaussian Propagation Model

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Background

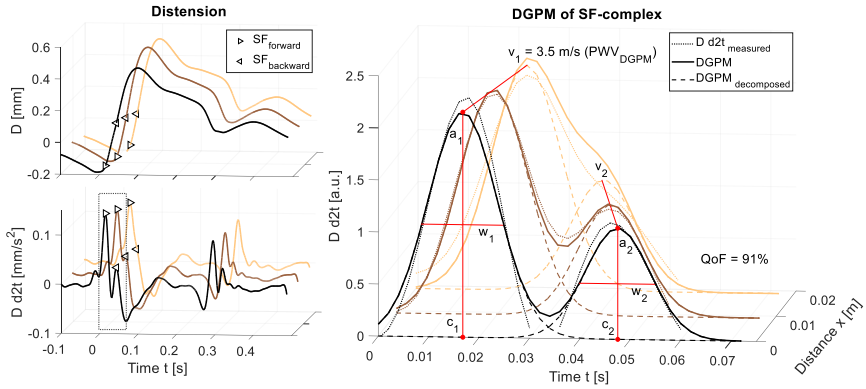
Pulse wave velocity (PWV) is an established marker of arterial stiffness(1). Local PWV estimates, however, are affected by confluence of incident and reflected waves, biasing the spatiotemporal propagation of the systolic foot (SF) in the distension waveform(2,3). We, therefore, propose a Double Gaussian Propagation Model (DGPM) to estimate PWV in consideration of local wave dynamics.

Methods

Ten subjects (38±10 years) were measured in rest for 2 minutes, repeatedly in 3 sessions over 3 weeks. From carotid ultrasonography (*Vantage64,VerasonicsInc.*,USA), we acquired 32 distension waveforms over a 19mm wide arterial segment, simultaneously with noninvasive continuous blood pressure (*NOVA,FinapresMedicalSystemsB.V.*,NL). The DGPM, fitted to the detrended second derivative (of the SF-complex, was defined as:
with time t[s], segment distance x[m] and 8 parameters modelling all 32 waveforms, i.e. a(mplitude)[a.u.], c(entroid)[s], w(idth)[s] and v(elocity)[m/s] of the forward($_1$) and backward($_2$) propagating wave, respectively (see Figure). Quality of fitting (QoF) was assessed as percentage of the waveform accounted by DGPM relative to the mean amplitude.
Per cardiac cycle, PWV_{DGPM} (=v_1), spatiotemporal PWV(PWV_{ST}) from linear regression of SF distances and timings, and Bramwell-Hill PWV (PWV_{BH}) were computed(4). Pearson correlation coefficients were computed between session means of local PWV measures and PWV_{BH}.

Results

The DGPM adequately models the SF-complex (mean QoF=85% for >20.000 cardiac cycles). For PWV_{BH}, PWV_{DGPM} shows a significantly higher predictive utility compared to PWV_{ST} (r:0.64 vs. 0.10).



ConclusionThe proposed DGPM demonstrates significant predictive utility for PWV by accounting for wave confluence. This may facilitate the clinical practicality of local arterial stiffness estimation.

P.04

A transfer-function-free technique for the non-invasive estimation of central arterial pressure

Mr Alessandro Giudici[1], Ioana Cretu[1], Madalina Negoita[1], Professor Ian B Wilkinson[2], Professor Ashraf W Khir[1]

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Background: Central aortic pressure (*CAP*) is important for the determination of the cardiovascular risk. Transfer function (TF)-based techniques allow for estimating CAP non-invasively from pressure waveforms acquired distally in the circulation. However, TF-based CAP might preserve the high frequencies of the distal waveform (1). Therefore, we propose a new method where *CAP* is estimated from local direct non-invasive measurements of diameter (*D*) and blood velocity (*U*) waveforms.

Methods: Aortic root *D* and *U* were measured using an ultrasound scanner (GE,Vivid E95) in 10 healthy volunteers (46±15 years,5 men), and used to determine local wave speed (*PWV*) using the lnDU-loop method (2). Brachial systolic (P_s) and diastolic blood pressure (P_d), as well as central P_s, were also estimated using a sphygmomanometer (Uscom,BPPLUS-R7). *CAP* was determined as:

, where , and blood density =1060 kg/m^3.

Results: Mean brachial P_s and P_d were 124.1±9.5 and 77.9±5.4 mmHg, respectively. Mean *PWV* was 3.07±0.71 m/s, leading to =1.00±0.46. The average calculated P_s was only -0.4% lower than TF-P_s (116.4±11.2 vs. 116.9±8.9, mmHg). Estimated calculated aortic and measured brachial mean pressure were almost identical with a difference of 0.01% (97.8±6.9 vs. 97.8±6.5, mmHg).

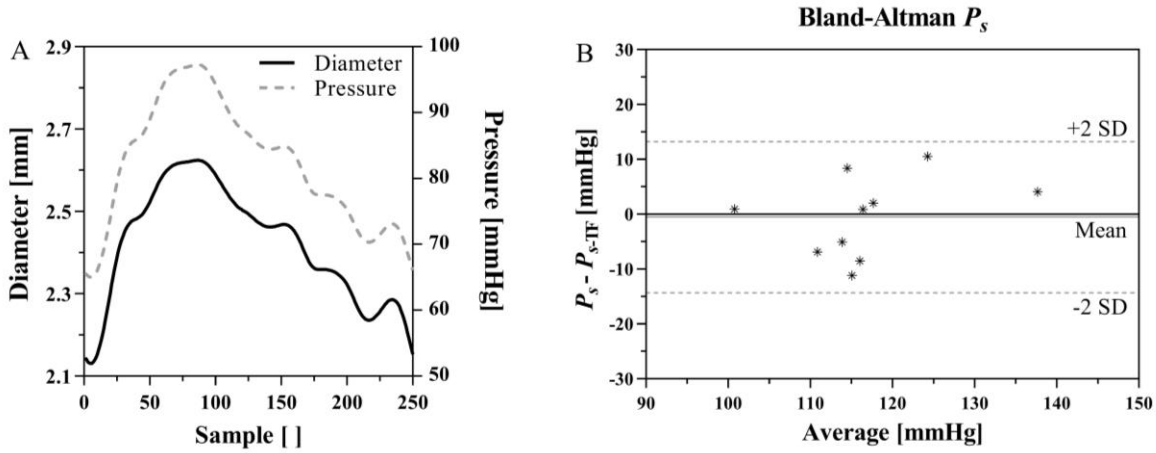

Figure – A: typical example of aortic *D* converted into *CAP*.

B: Bland-Altman comparison between calculated and TF P_s.

Discussion: This proof-of-concept study shows that the *CAP* waveform can be estimated non-invasively from measurements of brachial P_d, and aortic D and U obtained using equipment available in almost every cardiovascular clinic. Further studies are warranted to establish the full utility of the new technique.

P.05

Development and validation of a novel centroid method for estimating effective reflection time

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[1]*Murdoch Children's Research Institute,* [2]*University of Melbourne,* [3]*Royal Children's Hospital*

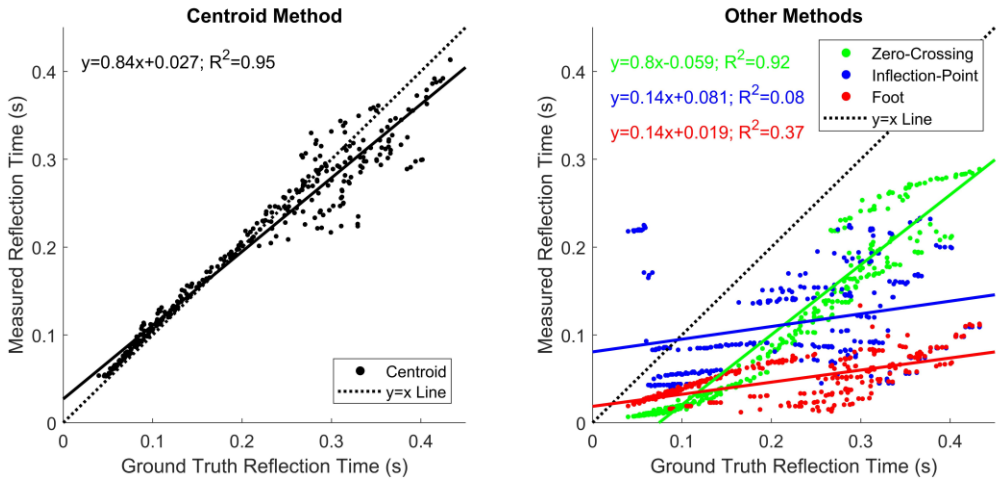
Title: Development and validation of a novel centroid method for estimating effective reflection time

Background: The time at which reflected waves arrive at central arteries has an important influence on ventricular afterload. Current methods of estimating reflection time (RT), including zero-crossover (1), inflection-point (2), and foot methods (3), use only a single point on the pressure waveforms, and their accuracy is uncertain because no ground truth reflection time (GTRT) has been available. We here introduce a novel centroid method that accounts for the entire waveform and compare the accuracy of RT methods by comparison with a GTRT for the first time.

Methods: Using computational linear wave-tracking, we followed an impulse as it traversed through an anatomical model of the systemic arterial circulation; GTRT was calculated as the weighted mean arrival time of reflected waves at the inlet. Linear convolution of the resulting impulse response with a realistic input waveform (flow waveform multiplied by characteristic impedance) produced a pressure waveform that was separated into forward and backward components. The time difference between the centroids of the backward pressure and input waveforms was taken as RT in the centroid method. We also conducted a parameter sweep (n=300) on the model to test the accuracy and robustness of the various methods.

Results: Compared to the zero-crossover, inflection-point, and foot methods, the centroid method estimated RT with the least mean difference to GTRT (104, 107, 171 vs. 8 ms; $p<0.001$) and least standard deviation (34, 109, 97 vs. 28 ms).

Conclusions: The centroid method substantially improved accuracy and robustness for estimating RT compared with current methods.



P.06

Comparison of Manual vs. Automated Haemodynamic Monitoring Systems in the Cardiac Catheterization Laboratory

Mr. AbdulRehman Alanezi[1], Dr. Fayaz Mohammad Khan[1], Mr. Taher Alotaibi[1], Mr. Bandar Alhaddadi[1], Mr. Fahad Alanazi[1], Mr. Mohammad Alqahtani[1], Mr. Jaber Alsheri[1], Mr. Ali Masrahi[1], Mr. Faisal Aljumah[1], Ms. Hanan AlShamamry[1], Mr. Ziyad Alwasel[1], Dr. Mohammad Balghith[1], Dr. Kamal Ayoub[1], Dr. Ali Al Ghamdi[1], Dr. Azra Mahmud[1]
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Background: Hemodynamic monitoring is an integral part of a cardiac catheterization procedure; however it is prone to many distortions, including damping and resonance[1].

Objectives: We sought to compare damping ratio, ascending aortic pressure waveform and invasive blood pressure between Manifold and ACIST CVi® devices in subjects undergoing cardiac catheterization.

Methods: This prospective randomised, single-blind, cross-over study was conducted in 81 adults subjects (mean age 59.2±12, 24% females) undergoing cardiac catheterization. The fast-flush test[2] was performed at the beginning of the procedure with both Manifold and ACIST. The square wave was analysed to calculate the damping coefficient. Data analyzed by JMP Pro (SAS for Windows, Version 13) $p<0.05$ considered significant.

Results: The mean damping ratio was 0.63±0.11(range 0.34-0.95) with Manifold vs. 0.94±0.25(range 0.53-2.1) with ACIST, mean difference 0.30, $p<0.0001$. The pressures were significantly different between the two devices; systolic -2.85($p<0.05$) ; diastolic -5.2 ($p<0.0001$) and mean pressure 3.5 ($p<0.01$), mm Hg. The inter-device BP difference showed a wide scatter; systolic, -24 to +67; diastolic, -44 to +25 and mean pressure,-24 to + 54 mm Hg.

Conclusions: To the best of our knowledge, this is the first study comparing a manual haemdynamic monitoring system to an automated one commonly used in the cardiac cath lab. The Manifold meets the international recommendations for accurate haemodynamic monitoring, compared with an overdamped ACIST which also underestimated pressures in our study. Manifold may be the preferred device for haemodynamic monitoring, particularly patients haemodynamically unstable, with cardiomyopathies and valvular heart disease.

P.07

The Progression of Left Ventricular Ejection Time in Simulated Microgravity

Dipl. Ing, BSc Stefan Orter[1,2], MSc Stefan Möstl[3], Dr. Martin Bachler[1], Dr. Med. Fabian Hoffmann[3], Dr. Christopher C. Mayer[1], Ao.Univ.Prof. Dipl.Ing. Dr.techn. Eugenijus Kaniusas[2], MSc Michaela Reisinger[1], Dr. Siegfried Wassertheurer[1], Prof. Dr. Med. Jens Tank[3], Dr. Bernhard Hametner[1]

[1]Austrian Institute Of Technology, [2]Technical University of Vienna, [3]German Aerospace Center

Introduction

Microgravity in space is known to cause major alterations in the cardiovascular system. Left ventricular ejection time (LVET) can be measured by the time from the onset point of the pressure wave to the incisura of the dicrotic notch. The aim of this study was to simulate microgravity by head-down tilt bedrest (HDT) to examine changes in LVET in female and male subjects.

Methods

24 healthy subjects (16 males and 8 females, height 176±7 cm, weight 77±6 kg, age 37±10 years) were enrolled in a HDT study. The bed rest study applied strict -6° HDT for 60 days. Pulse wave measurements were taken using an oscillometric pressure cuff on the brachial artery. LVET index (LVETi) was calculated according to Weissler et al (1). LVETis of different measurement times were compared using repeated measures ANOVA with post-hoc analysis using paired t-tests and Bonferroni correction.

Results

Fig. 1 shows a decrease of LVETi during bed rest, followed by a sharp rise of LVETi after bed rest. Repeated measures ANOVA confirmed significant differences between measurement times. The increase of LVETi from each HDT measurement day to 4 days after HDT (R+4) was significant (P<.001). There were no significant differences comparing

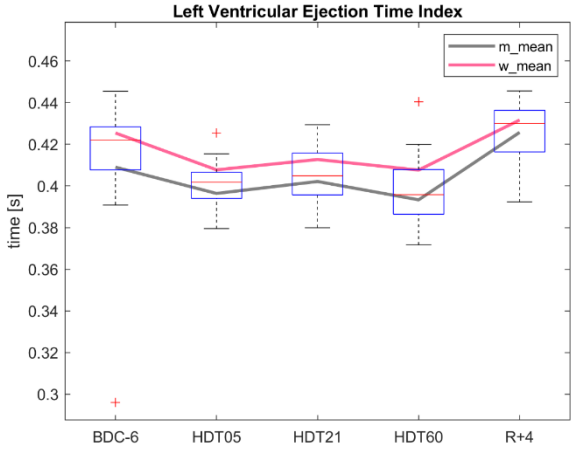

Fig. 1 LVETi measured 6 days before (BDC-6), on the 5th day (HDT05), on the 21st day (HDT21), on the 60th day (HDT60), and 4 days after (R+4) bedrest. Bold red and gray lines indicate female and male mean values.

Discussion/Conclusion

Overall, we conclude that LVETi decreased during HDT and reached four days after bed rest a similar level as before for both female and male subjects. As LVETi removes heart rate induced effects on LVET, the change in LVETi might be a result of change in ventricular ejection and afterload.

P.08

Biomechanical Characterization of Ascending Thoracic Aortic Aneurysms in Humans: A Continuum Approach to in vivo Deformations

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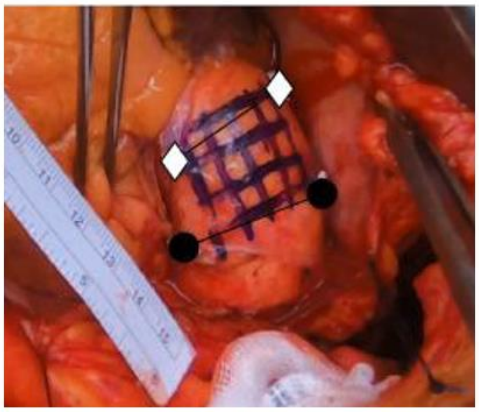
Background: Dysfunctional cellular mechanosensing appears central to aneurysm formation[1]. We aimed to derive material parameters of aneurysm tissue from *in vivo* deformations, which may increase insight into the underlying structural integrity of the pathological tissue.

Methods: Videos of tracking markers (example **Video** in supplement, screenshot in **Figure**) placed on ascending aortic segments were captured alongside radial arterial blood pressure in patients undergoing open-thorax ascending thoracic aorta aneurysm (ATAA) repair (n=5) and coronary bypass (controls; n=2). Normalised cross-correlation was used to determine marker displacements, resulting in estimates of systolic/diastolic diameters, distensibility, and cyclic axial engineering strain. A thin-walled, cylindrical geometry was assumed, with amorphous (Neo-Hookean) and fibrous (two-family) constitutive contributions [2]. This framework was fitted to individual patient measurements, by varying parameters c (amorphous material constant), k_1 and k_2 (fiber stiffness and strain stiffening parameter), β (fiber angle w.r.t. circumferential direction), unloaded intact length (L), and internal radius (R_i).

Results: Axial strain tended to be lower (expected) and distensibility larger (unexpected) in aneurysm than controls (**Figure**). However, the intrinsic pressure-dependence of distensibility must be considered when drawing conclusions related to differences in structural stiffness between both groups [3]. Material stiffness parameters (c and k_1) appeared higher in aneurysm patients than in controls which is in line with previous studies in mice [4].

Conclusion: We are developing a method to determine ATAA material properties from *in vivo* deformations and observed increased material stiffness in ATAA.



		Aneurysm	Control
Measured outcomes			
Diastolic diameter	[mm]	40 ± 5	23 ± 3
DBP	[mmHg]	58 ± 11	34 ± 2
SBP	[mmHg]	90 ± 18	93 ± 7
Distensibility	[MPa^{-1}]	4.3 ± 3.0	3.7 ± 1.1
Axial strain	[%]	4.3 ± 2.1	7.6 ± 3.5
Estimated properties			
c	[kPa]	37 ± 29	15 ± 13
k_1	[kPa]	43 ± 26	24 ± 24
R_i	[mm]	17 ± 1	10 ± 1
β	[degrees]	35 ± 3	36 ± 2
k_2	-	34 ± 9	37 ± 3
L	[mm]	24 ± 5	15 ± 2

Figure. Left: Example of ascending aortic region of interest with tracking markers. Right: Data presented as mean ± standard deviation. SBP/DBP, systolic/diastolic blood pressure. Estimated properties are defined in text.

P.09

Differential Low Flow Mediated Constriction (LFMC) responses in radial and brachial arteries of healthy humans are attributed to occlusion induced flow changes.

Ms Sakshi Sen[1], Dr Dinu Chandran[1], Dr Ashok Jaryal[1], Dr Kishore Kumar Deepak[1]
[1]*Department of Physiology, All India Institute of Medical Sciences*

Background: Literature describes differences in Low flow mediated constriction (LFMC) of radial versus brachial artery (1,2). We investigated whether differences in occlusion induced changes in luminal flow and shear rates could explain the observed radial vs brachial differences in LFMC responses.

Methods: Twenty Healthy volunteers (Age 23.50 ± 2.06 years) underwent examination of Brachial and radial arteries of both the arms. Arteries were visualized in Pulsed wave doppler in duplex mode (Vivid-e; GE Healthcare) using a 12 MHz probe at baseline and during low flow state produced using an occluding cuff placed around the forearm (for brachial artery) and over the wrist joint (for radial artery) inflated to supra-systolic pressures.

Results: LFMC in Radial artery was significantly higher than that of Brachial artery [delta LFMC of -0.58 mm (-0.24 – -0.76) in radial artery *vs* 0.03 mm (0.21 – -0.13) in brachial artery; $p < 0.0001$). Occlusion induced reduction in anterograde shear rate was comparatively higher in radial artery than brachial artery [-67.9% (-43 – -77) in radial *vs* -56.7% (-40.9 – -65.4) in brachial; p=0.0098] whereas, the rise in retrograde shear rate and changes in oscillatory shear index were comparable between both. Percentage changes in anterograde shear rate and anterograde flow velocities emerged as independent predictors in the regression model that explained 86% of the variance in LFMC responses of brachial artery.

Conclusion: Discrepancies in the LFMC responses of radial *vs* brachial artery could be attributed to the occlusion induced differences in the anterograde shear rate and anterograde flow velocities.

References:

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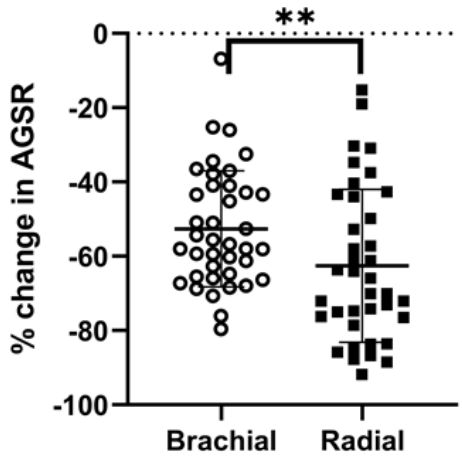
Comparison of occlusion induced % reduction in Anterograde shear rate (AGSR) in Brachial *vs* Radial artery



P.10

Distal arterial occlusion at different grades of supra-systolic pressures differentially modulates flow velocity and shear rates in radial artery

Miss Anchal Singh[1], Dr. Smriti Badhwar[2], Dr. Dinu Chandran[2], Prof Ashok Jaryal[2], Prof Kishore Kumar Deepak[2]

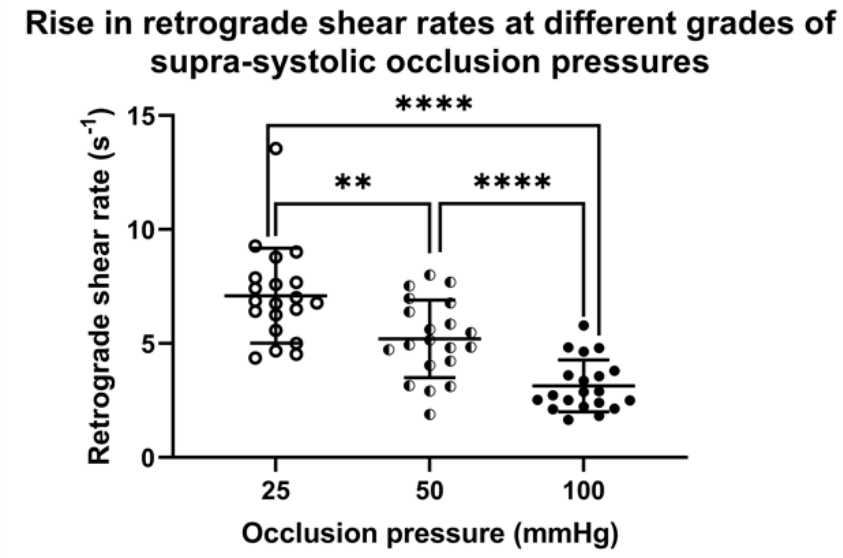
[1]*All India Institute Of Medical Sciences,* [2]*All India Institute Of Medical Sciences*

Background: Low-flow-Mediated Constriction (LFMC) is considered as a measure of resting endothelial function and is believed to occur in response to reduction in flow during supra-systolic occlusion(1). However, supra-systolic occlusion may alter flow profile with differential effects on anterograde and retrograde flow, which could potentially affect LFMC response. The current study investigated effect of graded supra-systolic occlusion on radial artery flow profile.

Materials and methods: Pulsed-wave Doppler in Duplex mode was used to record luminal flow velocity and arterial diameter in 20 healthy volunteers. Anterograde shear rates (AGSR), Retrograde shear rates (RGSR) and Oscillatory shear index (OSI) were calculated using standard formulae. Changes in radial artery flow velocities and shear rates were measured at baseline and in response to graded supra-systolic occlusion at 25, 50 and 100 mmHg applied for 5 minutes.

Results: Significant decrease in anterograde flow velocities and AGSR and increase in retrograde flow velocities and RGSR at all grades of occlusion was observed compared to respective baselines. Percentage reduction in AGSR was significantly higher at 100 mmHg compared to lower pressures(50.34±19.48 *vs* 21.37± 24.27 at 25 mmHg &35.48± 21.27 at 50 mmHg; $p < 0.05$). Occlusion induced rise in RGSR and OSI peaked at 25 mmHg occlusion and showed stepwise decrement at higher grades of occlusion; $p<0.05$ for all comparisons.

Conclusion: Graded supra-systolic occlusion differentially modulates AGSR, RGSR and OSI in radial artery. Therefore, quantification of resting endothelial function by LFMC may be influenced by grade of distal supra-systolic occlusion pressure applied to induce low flow state.



P.11

Higher ventricular and arterial stiffness in women partly explain sex-difference in left ventricular remodelling pattern by 3D echocardiography and all-cause mortality: Findings from the Southall and Brent revisited (SABRE) study

This abstract has been withdrawn by the Author.

P.12

Investigating the role of glycemic markers in pulse pressure amplification in young adults: The African-PREDICT study

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[1]Hypertension in Africa Research Team (HART), North-West University, [2]MRC Research Unit for Hypertension and Cardiovascular Disease, North-West University

Objective: Pulse pressure amplification (PPA) is described as the amplification of pulse pressure from central arteries to the periphery[1] and individuals with a decreased PPA have an increased risk of cardiovascular disease[2]. Adverse changes in PPA are evident in diabetic populations[3]; however, it is unclear whether PPA differs along varying degrees of glycaemia in young healthy populations. We therefore investigated whether PPA is attenuated with higher levels of glycemic markers and whether PPA is associated with glycemic markers independent of other known risk factors.

Methods: We included 1195 men and women from the African-PREDICT study, aged 20–30 years, with no prior diagnosis of chronic disease. We determined supine central PP (cPP) using the SphygmoCor XCEL device and PPA was defined as the ratio of the amplitude of the PP between the distal and proximal locations (bPP/cPP). Fasting glucose and glycated haemoglobin were determined and the study population was stratified by tertiles of each glycemic marker.

Results: The mean PPA was lower in the highest tertile of fasting glucose when compared to the lowest tertile (1.11 vs. 1.23; P≤ 0.001). PPA declined with increasing levels of fasting glucose (P–trend ≤ 0.001) after adjustment for age, sex, ethnicity, height, heart rate and mean arterial pressure. In multivariable adjusted regression, we found an independent inverse association between PPA and fasting glucose (β = −0.15, P≤ 0.001).
Conclusion: PPA decreases with an increase in fasting glucose in adults younger than 30 years, exemplifying early vascular changes which may increase future cardiovascular risk.

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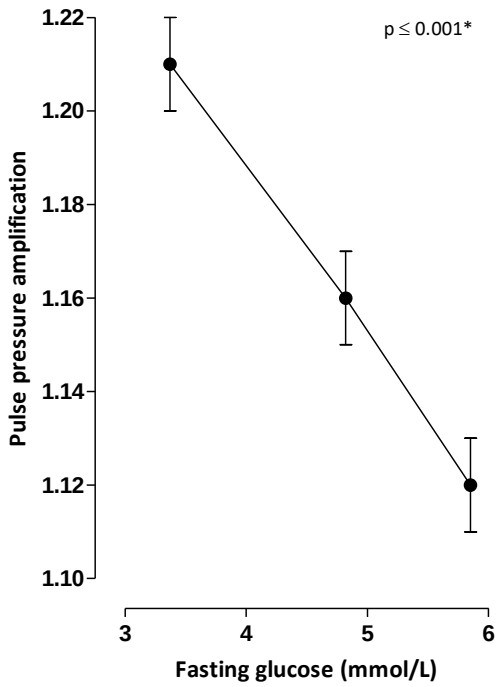

Figure 1: Pulse pressure amplification plotted against tertiles of fasting glucose. Values are adjusted for age, sex, heart rate, height and mean arterial pressure. * indicates P-value < 0.05 between the lowest and highest glucose group.

Adjusted R^2	**Pulse pressure amplification 0.15**	
	β (SE)	**P**
Age (years)	-0.17 (0.03)	< 0.001
Sex	0.10 (0.04)	0.023
Ethnicity	0.02 (0.03)	0.573
Height (cm)	0.13 (0.04)	0.005
Waist circumference (cm)	-0.06 (0.03)	0.150
Mean arterial pressure (mmHg)	0.17 (0.03)	< 0.001
Heart rate (bpm)	0.10 (0.03)	< 0.001
LDL-c(mmol/L)	-0.04 (0.03)	0.179
Fasting glucose (mmol/L)	-0.15 (0.03)	< 0.001
Total energy expenditure (kCal)	0.06 (0.04)	0.091
Self-reported smoking (%)	-0.01 (0.02)	0.809
Self-reported alcohol use (%)	-0.03 (0.02)	0.350

Table 2: Forward stepwise multiple regression analyses between PPA and fasting glucose

Data expressed as beta-values and standard errors. LDL-c, low-density lipoprotein cholesterol. P-values ≤ 0.05 regarded as significant.

P.13

Pulse wave velocity trajectories during COVID-19 epidemic: effect of lockdown on cardiovascular health

Dr Rosa Maria Bruno[1], Prof. Jean-Louis Pepin[2], Rui-Yi Yang[3], Vincent Vercamer[3], Paul Jouhaud[3], Pierre Escorrou[3], Pierre Boutouyrie[3]
[1]Inserm U970, Université de Paris, [2]INSERM U1042, University Grenoble Alpes, [3]Withings

Aim: to investigate PWV trajectories before and during the lockdown period among Withings Body Cardio bathroom scale users in France, in which a strict total lockdown was imposed, and Germany, in which partial social distancing measures were adopted.

Methods: The study population is constituted by Withings BodyCardio Bathroom scale users with at least one recorded weight in the period from week 8 (start 17 February 2020) to week 17 of 2020 (end 26 April 2020) in France (n=14,131) and Germany (n=20,104). Subgroup analysis in owner of activity trackers and the blood pressure (BP) oscillometric devices were conducted. Linear growth curve modeling and clustering trajectories analysis were used.

Results: French participants experienced during total lockdown a marked reduction in PWV, weight and physical activity, with no change in BP. German participants showed a higher PWV at baseline (difference 0.29m/s, $p<0.0001$), but French participants showed a steeper reduction over time (difference in slope -0.8cm/s/week, $p<0.0001$). Conversely, German participants had a greater weight at baseline than French participants, but also a greater weight reduction during lockdown, with a marginal reduction in PWV.
In the French population three clusters were identified: decreasing (24.4%), stable (56.6%) and increasing PWV (19.0%). Decreasing and increasing PWV clusters had similarly higher PWV at baseline than stable PWV cluster, whereas only decreasing PWV cluster showed a significant weight reduction (-500 g).

Conclusions: Total lockdown induced a reduction in PWV in a significant proportion of French bathroom scale users, thus representing an opportunity to improve their cardiovascular health.

P.14

Transcranial colour duplex reveals haemodynamically significant venous flow alterations following resection of arteriovenous malformation of the brain

Ms Kathryn Busch[1], A/Prof Andrew Davidson[1], Dr Mark Butlin[1], Prof Alberto Avolio[1], Prof Hosen Kiat[1]
[1]*Faculty of Medicine, Health, and Human Sciences*

Background
Resection of a brain arteriovenous malformation (bAVM) can impose several post-operative challenges including post-operative haemorrhage (POH). (1) The mechanism of such complications remains controversial although deliberately inducing hypotension has proven successful for POH prevention. (2) Daily non-invasive monitoring for patients may be useful in predicting POH.

Transcranial color duplex (TCCD) and central aortic pressures (CAP) can be derived non-invasively providing pressure and haemodynamic measurements. These techniques may provide valuable insight into the process of vessel remodeling following bAVM resection.

Methods
This prospective study evaluated fifteen patients with bAVMs. CAP and TCCD were studied pre-operatively and then daily for up to 14 days post-operatively. CAP and TCCD parameters were compared with a group of normal volunteers and a control group of participants having craniotomies.

Results
The post-operative venous changes were dramatic and have not been previously studied. In the early post-operative period there was a marked prominence of venous flow adjacent to the middle cerebral artery. The venous signals demonstrated significantly increased velocities and pulsatility. Furthermore, two of the patients with sustained increased venous prominence, velocity, and pulsatility suffered a POH. After the initial period of post-operative venous flow alteration, the venous parameters returned to normal values.

Conclusion
These preliminary findings provide insight into vessel remodeling and elucidate the time frame for venous flow alterations after surgery. If confirmed in future validation studies, the period of increased venous velocity, pulsatility and prominence may correlate to the time at which patients are at risk of POH.

P.15

Isolated systolic hypertension and central blood pressure:
Implications from the National Nutrition and Health Survey in Taiwan

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Objectives We aimed to investigate the association between isolated systolic hypertension (ISH) and central blood pressure (BP) in a nationally representative population.

Methods A total of 2029 adults without anti-hypertensive medicine, aged ≥ 19 years old participated in the 2013–2016 National Nutrition and Health Survey in Taiwan. Central and brachial BP were simultaneously measured using a cuff-based stand-alone central BP monitor purporting to measure invasive central BP (type II device). Central hypertension[1] was defined by central systolic (SBP)/diastolic BP (DBP) ≥130 or 90 mm Hg, and ISH was defined by brachial SBP ≥140 and DBP < 90 mm Hg.

Results The prevalence of ISH was 6.51% among adults aged ≥19 years old (2.15% [n=21] for young adults [aged <50 years] and 10.54% [n=111] for older adults [aged ≥ 50 years]). ISH subjects had significantly higher central pulse pressure (PP) (62.8 mm Hg for the young and 72.4 mmHg for elders) than those subjects with either isolated diastolic hypertension (brachial SBP<140 and DBP≥90 mmHg, central PP 44.8 mmHg) or systolic diastolic hypertension (brachial SBP≥140 and DBP≥90 mmHg, central PP 60.2 mmHg). There was a U-shaped trend in the association between age and ISH prevalence, and between age and central PP. The ISH prevalence was 2.95%, 1.73% and 10.54%, and the average central PP was 49.5, 47.0, and 54.0 mmHg for subjects aged <30, between 30-50, and ≥50 years, respectively. Moreover, all ISH adults had central hypertension and a higher prevalence of obesity than the normotensives (body mass index ≥27 Kg/m^2, 71% vs. 17%, for age<50 years, and 27% vs. 17% for age≥50 years).

Conclusions All subjects with ISH, young or older, had central hypertension. Central PP was higher in the young and older age groups in comparison to the middle age group. The U-shaped trend corresponded to the association between age and ISH prevalence.

P.16

Expanding on the observed correlation between the ambulatory arterial stiffness index and the lower limit of cerebral autoregulation during cardiac surgery

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Background
The lower limit of cerebral autoregulation (LLA) refers to the mean blood pressure (BP) below which cerebral blood flow becomes pressure-dependent, resulting, among others, in an increased stroke risk. The LLA measured during cardiac surgery, correlates with the vascular measure Ambulatory Arterial Stiffness Index (AASI) determined from intraoperative continuous radial BP before cardiopulmonary bypass [1]. Using these data we investigated added factors that may enhance this correlation.

Design and method
The study population included 167 patients undergoing cardiac surgery (age 71±8 years, 68% males) with good-quality BP records. The AASI. Additionally tested predictors were body-mass index (BMI), the coefficient of variation (SD/mean) of the systolic BP (SBP_CV), the composite variables BMI*(1-AASI), and its linear combination with SBP_CV. The odds ratio (OR) was determined by applying logistic regression to dichotomized predictors (by medians) and LLA- (by selected thresholds) adjusted to age, sex, diabetes mellitus, heart rate and preoperative diastolic BP.

Results
The Table shows that the LLA of individuals correlated significantly with each of the (continuous) predictors, and the adjusted OR increased for the composite predictors (dichotomized), while showing insensitivity to adjustors. The ORs reached a maximum for a LLA threshold of 55mmHg.

Conclusions
The newly-defined composite predictors that increased the likelihood of predicting a LLA higher than 55 mmHg enhances our knowledge regarding the cerebral vasculature and autoregulation, and BP variability determinants of LLA under anesthesia

Predictor	Univariate regression	Adjusted OR
	r(P-value)	Mean[95%CI](P-value
AASI	0.27(0.0004)	2.41[1.16-5.00](0.02)
BMI	-0.26(0.0007)	3.77[1.78-8.00](0.0005)
SBP_CV	-0.29(0.0002)	3.50[1.71-7.16](0.0006)
BMI*(1-AASI)	-0.35(0.000005)	4.51[2.16-9.40](0.00006)
80*SBP_CV+BMI*(1-AASI)	-0.44(<0.000001)	8.20[3.67-18.3](<0.000001)

P.17

Reduced isometric contractility and isobaric compliance of the ex vivo thoracic aorta of hypertensive APP23+/- overexpressing mice due to serum corticosterone levels

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Objective: Alzheimer's disease (AD) is characterized by noticeable neuropsychiatric symptoms and cognitive decline (1). In addition, cardiovascular disease (CVD) is a known etiological hallmark of AD pathogenesis (2). Recent epidemiological evidence suggests an interplay between arterial stiffness (AS) and AD (3). Therefore, we aimed for an in-depth vascular characterization of the APP23$^{+/-}$ overexpressing AD mouse model (APP23$^{+/-}$).

Methods: Blood pressure (BP, CODA) and aortic pulse wave velocity (aPWV, VEVO2100) were measured *in vivo,* whereas isometric vascular reactivity (organ chambers), isobaric AS (Peterson modulus (Ep)) and compliance (Rodent Oscillatory Tension Set-up for Arterial Compliance) were determined *ex vivo* in thoracic aorta segments of APP23$^{+/-}$ mice (male, n=10) vs. C57BL/6 mice (male, n=18) at the age of 6 months. Corticosterone levels were analysed on blood serum by means of ELISA. The data are given as mean ± SEM.

Results: APP23$^{+/-}$ mice showed elevated corticosterone levels (Fig.1A) associated with increased peripheral systolic BP (Fig.1B) and aPWV *in vivo* (Fig.1C), and decreased isometric adrenoreceptor-dependent contractions e*x vivo* upon phenylephrine stimulation (Fig. 1D). *Ex vivo* isobaric AS measurements at baseline disclosed a smaller aortic diameter of APP23$^{+/-}$ mice (Fig.2A) resulting in reduced compliance (Fig.2B), with no Ep differences (Fig.2C). Upon phenylephrine treatment, a smaller effect on aortic constriction (Fig.2D), compliance (Fig.2E) and Ep (Fig.2F) was observed for APP23$^{+/-}$ animals, corresponding to reduced isometric contractions (Fig. 1D).

Conclusion: APP23$^{+/-}$ mice have increased corticosterone levels leading to increased BP, aPWV and reduced isometric contractility, resulting in decreased isobaric compliance, but with unchanged arterial wall biomechanics.

(figures on next page)

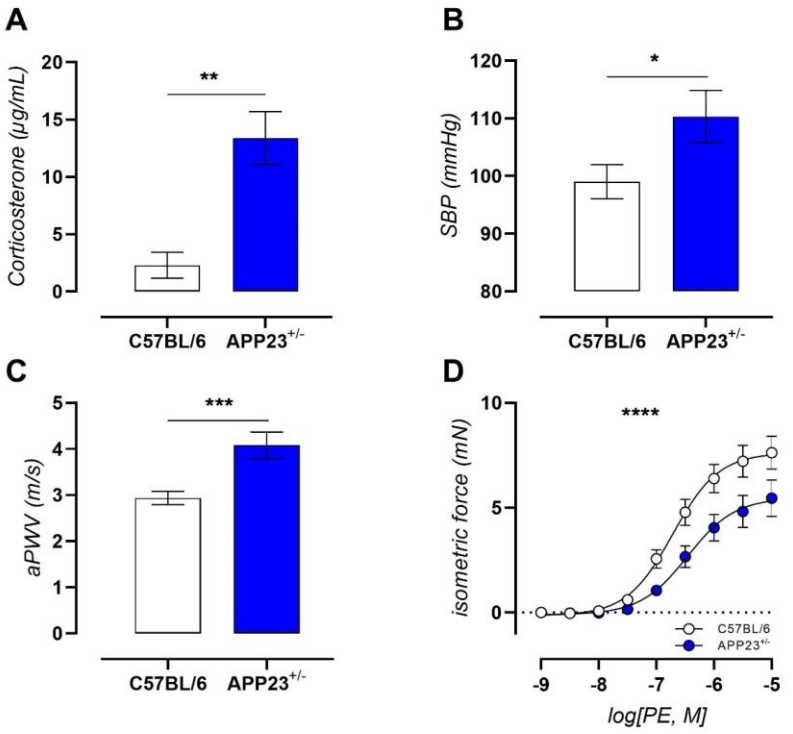

Figure 1: Significantly increased serum corticosterone levels **(A)**, systolic blood pressure (SBP) **(B)**, abdominal pulse wave velocity (aPWV) **(C)** and decreased isometric force **(D)** upon phenylephrine contraction in 6 months old APP23+/- vs. C57BL/6 mice.

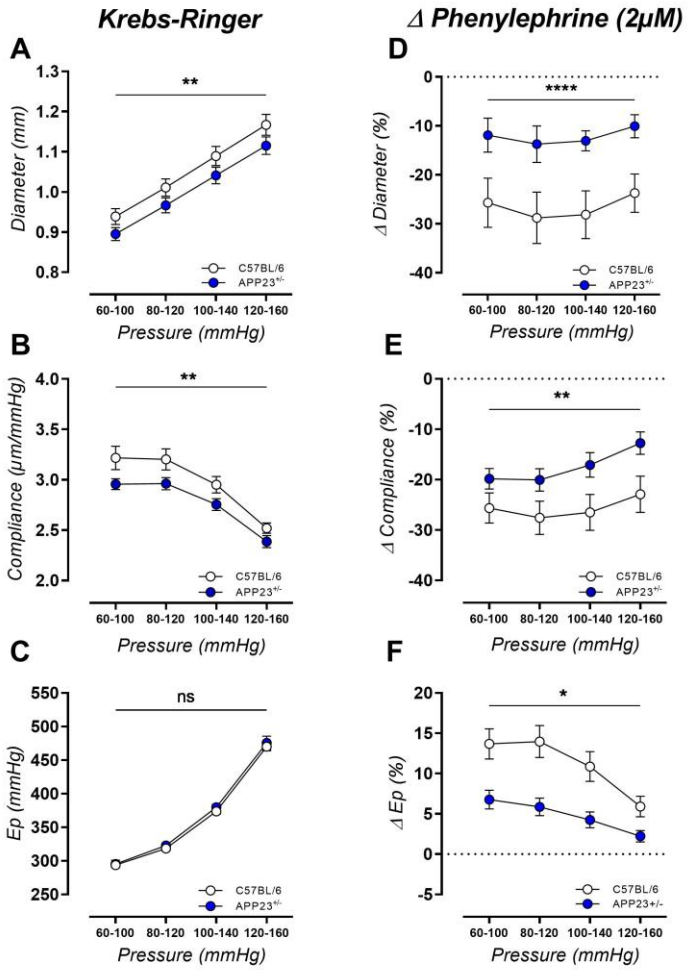

Figure 2 Significant smaller aortic diameter **(A)** and compliance **(B)** in baseline Krebs-Ringer solution for APP23+/- mice vs. C57BL/6 mice, without differences in Ep **(C)**. Limited aortic constriction **(D),** compliance **(E)** and Ep **(F)** upon phenylephrine (2μM) stimulation for APP23+/- mice vs. C57BL/6 mice.

P.18

Carotid Stiffness Parameters and Cerebral Blood Flow Pulsatility in Young Healthy Individuals across Races

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Background: Higher cerebral blood flow (CBF) pulsatility was found to be associated with severer brain white matter lesions in the elderly[1]. It was hypothesized that the central/elastic arterial stiffness/compliance may directly affect CBF pulsatility. However, it is still unclear which carotid stiffness parameters may better reflect this impact, and whether race and sex differences are present.

Methods: To study the correlations among those parameters with comparisons between different races and sexes, we enrolled 35 young healthy subjects (19 females), aged 29±5 (18~40) years, with three races of comparable age and sex ratio, i.e. White (*n*=16), Black (*n*=7), and Asian (*n*=12). All subjects were in resting seated position, with continuous transcranial Doppler recording of CBF velocity at middle cerebral artery (MCA), simultaneous 1-min ultrasound echo-tracking on bilateral common carotid arteries, and multiple measurements of brachial blood pressure (BP).

Results: All derived parameters[2], including MCA pulsatility index (PI), showed no significant racial differences but with significantly ($P < 0.05$) higher carotid stiffness index (β), Peterson's pressure modulus (Ep), BP pulsatility index (mostly driven by higher systolic BP but similar diastolic BP), and lower arterial compliance (AC, $P = 0.07$) in males than in females. Only AC (but not β and Ep) showed a significant correlation with PI ($r = 0.49$, $P = 0.004$) even after controlling for BP pulsatility index, which negatively correlated with AC ($\rho = -0.35$, $P = 0.038$).

Conclusions: Higher carotid AC (i.e. decreased stiffness) seems to enhance CBF pulsatility in young healthy populations, which might differ from the elderly.

P.19

Intradialytic changes in cerebral blood flow and regional changes in arterial stiffness

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Purpose/background/objective: Cognitive decline is highly prevalent amongst end-stage renal disease (ESRD) patients and is accelerated upon initiation of hemodialysis (HD)[1]. ESRD increases aortic stiffness and blood flow pulsatility, which may damage small vessels of target organs like the brain[2]. In this pilot study, we aimed to evaluate the acute effect of HD on cerebral blood flow and its relation to arterial stiffness.

Methods: Before, every hour during and after HD (T0-T4), we measured cerebral flow velocity (FV) using transcranial Doppler, blood pressure (BP) via digital finger cuff (Nexfin), cardiac activity using ECG and aortic pulse wave velocity (PWV) with Mobile-O-Graph. FV pulsatility index (PI) and transit times between ECG peak and the foot of both FV and BP waveforms (cerebral dT; digital dT) were computed using in house MATLAB-based analysis. Changes during HD were evaluated with Generalized Estimating Equation models adjusting for multiple comparisons in SPSS 26.0.

Results: In eight participants aged 63 ± 17 y. old (4 diabetics, 3 women), peak FV decreased from baseline at T1 and T2 (-11.2 cm/s, p=0.007; -12.2 cm/s, p<0.001), PI decreased at T1 (0.81 to 0.77, p=0.005), whilst minimum FV, mean BP and partial pressure of CO_2 remained unchanged. Digital dT increased at T3 (0.19 to 0.22, p<0.001) and cerebral dT increased throughout HD (T1-T4, p<0.005), whereas aortic PWV did not change.

Conclusions: During hemodialysis, cerebral and digital transit times increased, suggesting decreased stiffness of small peripheral vessels, without significant changes in aortic stiffness. Reduced stiffness of cerebral arteries may partially explain decreased cerebral flow pulsatility.

P.20

Evolving Structure-Function Correlates during Aortic Maturation and Aging

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Introduction

Unraveling aortic cellular and extracellular microstructural and mechanical mechanisms triggered to maintain homeostasis in murine aortae during maturation and aging is fundamental to better understand remodeling in human arteriopathies [1].

Methods

This study, combining ex-vivo extension-inflation testing [2], multiphoton microscopy and optical histology, aimed to quantify multiple microstructural parameters of primary extracellular components – collagen, elastic lamellae – and cells – endothelial, smooth muscle and adventitia cells – of the aorta with a dynamic and multiregional 3D approach. The analysis focused on the quantification and correlation of the histo-mechanical properties of the thoracic aorta as a function of age from 21 days to 1 year after birth, that is, from the time of weaning to maturation and therefore the natural aging.

The parameters quantifying the three-dimensional microstructural phenomena of deposition, remodeling and removal of aortic components under pressure and stretch conditions equivalent to those in vivo were layer thicknesses, straightening, alignment and thickness of collagen bundles, number and size of elastic lamellae, density and alignment of the different vascular cells.

Results

Changing dynamics at different ages were characterized, such as the smooth muscle cell population reduction and hypertrophy with the interlamellar widening from an intermediate age. Significant correlations indicated the fundamental role of both cells and deposited extracellular proteins such as the reduction in endothelial and smooth muscle cell densities but also the increase in straightness and thickness of collagen bundles in relation to the increase in circumferential and axial stiffness of the aortic wall.

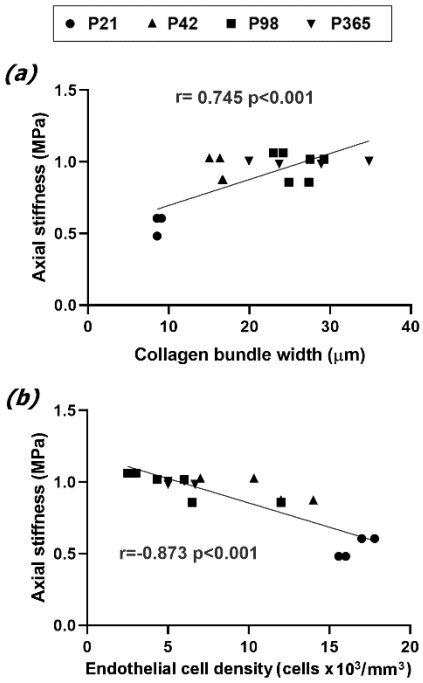

Figure 1 Microstructure-mechanics correlates

P.21

Albuminuria intensifies the relationship between urinary sodium excretion and central pulse pressure: the Wakuya study

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Objectives: Central pulse pressure (cPP) is responsible for vital organ hemodynamics,[1,2] and its monitoring is important for cardiovascular disease prevention.[3] Excess sodium intake and (micro)albuminuria, a manifestation of renal microvascular damage, are also known as strong predictors of cardiovascular disease.[4,5] We sought to investigate the cross-sectional relationships among dietary sodium consumption, albuminuria and cPP in the general population.

Methods: Subjects were 933 apparently healthy adults in Wakuya town, Miyagi, Japan (mean age, 56±10 years). Radial pressure waveforms were recorded with applanation tonometry to estimate mean arterial pressure (MAP), cPP, forward and backward pressure amplitudes, and augmentation index. Urinary sodium/creatinine ratio (UNaCR) and albumin/creatinine ratio (UACR) were measured in spot urine samples.

Results: Median values of UNaCR, UACR and cPP were 139 (interquartile range, 89-205) mEq/g, 5 (4-11) mg/g and 38 (33-45) mmHg, respectively. Both UACR and UNaCR were positively correlated with cPP, even after adjusted for MAP (P<0.001). Moreover, UACR and UNaCR had a synergistic influence on increasing cPP, which was independent of age, sex, estimated glomerular filtration rate, hyperlipidemia and diabetes (interaction P<0.05). A similar synergistic influence between UACR and UNaCR was found on the forward but not backward pressure amplitude or augmentation index. The overall results were not altered on replacement of UACR with the existence of chronic kidney disease.

Conclusions: (Micro)albuminuria strengthens the positive association between urinary sodium excretion and central pulse (and systolic forward) pressure. Excess sodium intake may magnify cardiovascular risk through widening aortic pulsatile pressure, particularly in the presence of concomitant chronic kidney disease.

P.22

Mortality in 98 Type 1 Diabetes Mellitus (T1DM) and Type 2 Diabetes Mellitus (T2DM) Individuals presenting to a Specialist Podiatry Clinic: Foot Ulcer Location is an Independent Risk Determinant

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Purpose/Background
We previously demonstrated in both longitudinal study and meta-analysis (pooled relative-risk RR, 2.45)[1,2] that all-cause and cardiovascular mortality is significantly higher in people with diabetes foot ulceration(DFU) than with those without a foot ulcer. In this prospective study, we looked at the factors linked to mortality after presentation to podiatry with DFU.

Methods
98 individuals recruited consecutively from the Salford Royal Hospital Multidisciplinary Foot Clinic in Spring 2016 were followed for up to 48 months. Data concerning health outcomes were extracted from the electronic patient record (EPR).

Results
Seventeen people(17) had type 1 diabetes mellitus (T1DM), and 81 had type 2 diabetes mellitus (T2DM). 31 were women. The mean age (range) was 63.6 (28-90) years with maximum diabetes duration 45 years. Mean HbA1c was 72 (95%CI: 67-77) mmol/mol. 97% had neuropathy (International Working Group on the Diabetic Foot (IWGDF) monofilament)[2]. 62% had vascular insufficiency (Doppler studies). 69% of ulcers were forefoot and 23% of ulcers were hind foot in location.
40/98 (39.2%) died in follow-up with 27% of death certificates including sepsis (not foot-related) and 35% renal failure as cause of death. Multivariate regression analysis indicated a 6.3 (95%CI 3.9-8.1) fold increased risk of death with hind foot ulcer, independent of age/BMI/gender/HbA1c/eGFR/total cholesterol level.

Conclusion
This prospective study has shown a close relation between risk of sepsis/renal failure and presentation to a specialist podiatry clinic with hind foot ulceration an independent risk factor, highlighting again the importance of addressing cardiovascular risk factors[3] as soon as people present with DFU.

P.23

Relationship between aortic stiffness, aortic, and carotid impedance with vascular aging in community-based healthy people.

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Background: Stiffening of the aorta has been associated with microvascular structural brain damage and cognitive dysfunction in cross-sectional and longitudinal studies, probably due to the reduced wave reflection at the interface between carotid and aorta and transmission of excessive flow pulsatility into the brain. The present study investigated whether age-related stiffening of the proximal aorta is disproportionately greater than the carotid arteries and whether the coupling of stiffness between proximal aorta and carotid arteries affects wave reflection at the interface.

Methods: Comprehensive pulsatile hemodynamics evaluation using applanation tonometry, carotid ultrasonography and echocardiography was performed in 1236 apparently healthy community residents (age range 40 to 96 years, average 62.4±10.5 years) who had no history of cardiovascular events and stroke. Aortic (Zc) and carotid (CCI) characteristic impedance were estimated in the time domain. Carotid pulsatility index (PI) was computed from the carotid flow and a carotid reflection coefficient was computed from bilateral carotid impedance and distal aortic impedance.

Results: CFPWV (Standardized Beta=0.56, P<0.001), CCI (Beta=0.46, P<0.001) and carotid reflection coefficient (Beta=0.26, P<0.001) significantly increased with age. In contrast, PI (Beta =-0.06, P=0.026) significantly decreased with age, and a U-shape association between Zc (Beta =0.09, P=0.002) and age was found. Carotid flow PI was significantly related to the aorta-carotid reflection coefficient negatively (r =-0.28, P<0.001).

Conclusions: In this healthy population, stiffening of the proximal aorta was not disproportionately greater than that of the carotid arteries. This might favorably maintain the wave reflection at the carotid-aorta interface so that carotid flow PI did not increase with age.

(figures on next page)

Table 1. Subjects characteristics (subdivided into groups according to the age)

Age group n=1236	40~49 n=137	50~59 n=356	60~69 n=445	≧70 n=298	P value for trend
Age (years)	45(3)	55(3)	64(3)	76(5)	<0.001
Male (male,%)	53(41.7)	153(43.7)	207(47.2)	159(55.0)	0.002
Height (cm)	162.5(8.1)	161.5(8.1)	159.5(7.5)	158.3(8.2)	<0.001
Weight (kgw)	64.0(12.7)	64.7(10.6)	63.0(10.4)	62.0(10.9)	0.004
bmi (kgw/m^2)	24.1(3.6)	24.7(3.3)	24.7(3.3)	24.7(3.4)	0.31
HTN (n, %)	9(7.1)	76(21.7)	172(39.2)	158(54.7)	<0.001
DM (n, %)	5(3.9)	32(9.1)	78(17.8)	71(24.6)	<0.001
Hyperlipidiemia (n, %)	6(4.7)	50(14.3)	229(52.2)	144(49.8)	<0.001
Creatinine (mg/dL)	0.77(0.45)	0.73(0.21)	0.79(0.27)	0.93(0.57)	<0.001
LDL (mg/dL)	116.5(34.8)	120.0(35.9)	117.7(34.1)	113.3(35.1)	0.083
Glucose (mg/dL)	94.5(18.1)	100.4(21.2)	104.3(28.2)	105.3(26.3)	<0.001
Smoking (n, %)					0.08
Non-smoker	95(74.8)	255(72.9)	341(77.7)	204(70.6)	
Current smoker	14(11.0)	44(12.6)	38(8.7)	14(4.8)	
former smoker	18(14.2)	51(14.6)	60(13.7)	71(24.6)	
MMSE score<24	0	3(0.9)	13(3.0)	31(10.8)	<0.001
Echocardiography variables					
Ea (mmHg/ml)	1.86(0.44)	1.78(0.43)	1.70(0.43)	1.70(0.53)	<0.001
Ees (mmHg/ml)	4.00(1.06)	4.09(1.06)	3.67(1.12)	3.99(1.73)	0.12
Ea/Ees	0.48(0.09)	0.45(0.09)	0.48(0.12)	0.45(0.13)	0.87
Stroke volume (ml)	58.5(12.3)	64.0(14.2)	69.5(14.9)	72.5(17.6)	<0.001
Cardiac index (L/min/m^2)	2.4(0.6)	2.6(0.6)	2.7(0.6)	2.9(0.8)	<0.001
EF (%)	69.5(6.2)	70.7(6.4)	68.8(7.1)	68.5(7.9)	0.001

Data were presented as mean(SD), BMI: body mass index, HTN: hypertension, DM: diabetes mellitus, LDL: low density lipoprotetin, MMSE: mini-mental state examination, Ea: arterial elastance, Ees: left ventricular end-systolic elastance, LVEDV: left ventricular end-diastolic volume, LVESV: left ventricular end-systolic volume

Table 2. Hemodynamics parameters(subdivided into groups according to the age)

Age group n	40~49 n=137	50~59 n=356	60~69 n=445	≧70 n=298	P value for trend
Central					
SBP (mmHg)	107.0(16.2)	112.5(13.9)	118.0(15.5)	121.6(14.9)	<0.001
DBP (mmHg)	74.3(12.5)	77.8(10.2)	78.8(10.6)	77.5(9.7)	0.024
PP (mmHg)	33.6(7.1)	35.5(7.4)	40.1(8.7)	45.3(9.9)	<0.001
Brachial					
SBP (mmHg)	120.2(17.3)	125.3(15.1)	131.2(16.8)	134.6(16.5)	<0.001
DBP (mmHg)	73.3(12.5)	76.8(10.2)	77.5(10.5)	76.1(9.7)	0.070
PP (mmHg)	41.2(9.1)	43.7(11.7)	48.9(11.7)	53.4(12.6)	<0.001
MAP (mmHg)	90.9(14.3)	95.0(12.4)	98.4(13.6)	99.9(12.5)	<0.001
Heart rate (bpm)	69(9.7)	69(10.3)	66(10.2)	66(10.3)	<0.001
Pf (mmHg)	28.8(7.1)	28.1(6.6)	30.2(6.9)	34.5(8.7)	<0.001
Pb (mmHg)	14.5(3.1)	15.4(3.3)	17.7(3.8)	19.5(4.4)	<0.001
CFPWV (m/s)	9.4(1.9)	10.8(2.4)	12.4(3.1)	15.2(4.4)	<0.001
Aortic flow-pressure					
Zc (dyne·s/cm^5)	102.3(32.2)	89.6(31.6)	90.8(38.4)	104.7(50.7)	0.027
SVR (dyne·s/cm^5)	1782.0(551.4)	1685.2(435.7)	1683.7(441.9)	1664.0(543.7)	0.06
Mean flow (ml/s)	63.4(16.6)	69.6(19.7)	70.7(17.4)	73.7(20.6)	<0.001
normalized Zc	0.06(0.02)	0.05(0.02)	0.05(0.02)	0.06(0.02)	<0.001
Carotid flow-pressure					
CCI (dyne·s/cm^5)	2060.2(520.5)	2198.2(708.0)	3183.0(1030.9)	3328.3(1154.5)	<0.001
CVR (dyne·s/cm^5)	9421.3(2472.4)	9860.9(2049.4)	11879.2(2612.3)	12347.4(3022.2)	<0.001
Mean flow (ml/s)	13.2(2.5)	13.1(2.3)	11.2(2.2)	10.9(2.2)	<0.001
normalized CCI	0.22(0.05)	0.23(0.06)	0.27(0.08)	0.27(0.08)	<0.001

Data were presented as mean(SD), SBP: systolic blood pressure, DBP: diastolic blood pressure, PP: pulse pressure, MAP: mean arterial pressure Pf: forward pressure, Pb: backward pressure, CFPWV: carotid-femoral pulse velocity, Zc: aortic characteristics impedance, SVR: systemic vascular resistance, CCI: carotid characteristics impedance, CVR: carotid vascular resistance,

P.24

Factors associated with premature vascular aging in patients with arterial hypertension.

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Objectives. Identification of factors associated with premature vascular aging in patients with arterial hypertension (AH).

Methods. The study included 61 patients (29 men, 32 women) with 1-2 grade AH without cardiovascular complications, aged 40 to 60 years, of which 28 (45.9%) had type 2 diabetes. The duration of AH was 5.0 [2.0;10.0] years, type 2 diabetes - 2.0 [1.0;4.0] years. The assessment of vascular stiffness using cardio-ankle vascular index (CAVI) and vascular age (VA) was performed by volumetric sphygmography (VaSera-1500). All patients were divided into 2 groups: with normal VA (the passport age corresponds to the VA, n=35) and premature vascular aging (the VA is higher than the passport age, n=26).

Results. There were no significant differences in gender, smoking, dyslipidemia, and the presence and duration of type 2 diabetes in the comparison groups. Significantly higher values of blood pressure (BP) were observed in the group of patients with premature vascular aging - 153.7±26.2/95.3±13.2 versus 137.9±18.6/88.4±13.3 mm Hg., higher duration of AH 10.0 [4.0;13.0] vs. 3.0 [1.0;8.0] years, higher blood urea level 5.7 [5.3;6.8] vs. 4.5 [3.8;5.8] mmol/l, increased CAVI average values on the right and on the left 8.8 [8.4;9.4] vs. 7.4 [6.7;7.9], intima-media thickness (IMT) of CCA (0.92±0.13 vs. 0.80±0.09 mm) and LV mass index (LVMI), 96.1±21.8 vs. 86.1±20.7 g/m^2.

Conclusion. Premature vascular aging in patients with AH was associated with the degree and duration of hypertension, increased IMT, vascular stiffness [1], blood urea levels and LVMI.

P.25

Radial-Digital pulse wave velocity: a non-invasive method for assessing stiffness of peripheral small arteries

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Pulse wave velocity (PWV) has been used to evaluate arterial stiffness of large arteries. Here, we examine the feasibility of radial-digital PWV (RD-PWV) as a measure of stiffness of smaller arteries, and its response to changes in local mean arterial pressure.
In 29 healthy subjects, we used Complior probes to record arterial pulse wave at radial artery and tip of the index. To determine transit time, we used both second derivative and intersecting tangents of the entire recordings using the device-embedded algorithms, in house Matlab analyses of only reliable waves, and by numerical simulation using arterial tree model. In 15 subjects, we examine the response of RD-PWV to changes in local MAP by vertical displacement of the hand above and below the mid-axillary line.

Using second derivative, RD-PWV were 4.68 ± 1.18, 4.69 ± 1.21, 4.32 ± 1.19 m/s respectively for device-embedded, Matlab-based and numerical simulation analyses, respectively. Using intersecting tangents RD-PWV were 4.73 ± 1.20, 4.45 ± 1.08, 4.50 ± 0.84 m/s, respectively for device-embedded, Matlab-based and numerical simulation analyses, respectively. The strongest correlation (r=0.92) was seen between device-embedded and Matlab-based second derivatives. The intersession coefficients of variation were 7.0 ± 4.9% and 3.2 ±1.9% (P=0.04) for device-embedded and Matlab-based second derivative algorithms. We estimated that each increase of 10 mm Hg in local MAP by vertical displacement of the hand resulted in an increase in RD-PWV of 0.28 m/s.

This study shows that RD-PWV can be used for the non-invasive assessment of stiffness of small-sized arteries.

P.26

Liver Transglutaminase 2 Level Comparison Among Different Dietary Interventions

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Purpose/Background/Objectives: Tissue transglutaminase (TG2) is a highly expressed protein especially in endothelial cells. TG2 has several functions including transamidation activity which is important in several processes such as extracellular matrix remodeling (1). TG2 activity takes place in aortic stiffness regulation and atherosclerotic plaque formation. (2). One of the most effective implementation for atheroprone state and general cardiovascular health is calorie restriction (CR). In addition, lipid accumulation and subsequent metabolic disorders can be regulated by CR and longer lifespan can be achieved (3). In this study we aimed to determine the effect of different CR application types on liver TG2 levels of female mice fed up to 82 weeks old age.

Methods: For this purpose, female MMTV-TGF-α mice fed with different dietary regimes; *ad libitum* (AL), chronic CR (%15 restriction of AL group), intermittent CR (3 weeks AL (ICR-ReFeed)+1 week %60 restriction of AL (ICR-Restricted), between 10-week to 82-week old. Liver tissue was isolated at 10-week old (AL mice as baseline), 50 and 82 weeks. Then, liver tissue samples were homogenized for western blotting. Analysis made by ImageLab software and Glyceraldehyde-3-Phosphate Dehydrogenase used as housekeeping gene.

Results: TG2 levels were increased in CCR and ICR-R groups, decreased in ICR-RF compared to AL group. In addition, 82-week old AL mice had higher level of TG2 than 10-week old.

Conclusion: These results may provide future perspectives about TG2 levels depending on feeding protocols and ageing in kidney. TG2 levels in arteries of the same groups will be examined in further studies.

P.27

Mechanisms of NADPH oxidase participation in the regulation of diaphragm artery contractile responses

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Reactive oxygen species (ROS) produced by NADPH-oxidase (NOX) participate in vascular tone control, but their effects in the arteries of respiratory muscles is poorly understood. Possible targets of vasoregulatory ROS influence are NO-pathway in the endothelium and Rho-kinase pathway in smooth muscle cells. Therefore, the **aim** of this study was to evaluate the interaction of NOX-dependent control with NO- and Rho-kinase signaling pathways in rat diaphragm arteries (DA).

Methods. The segments of DA were isolated from male Wistar rats and mounted in wire myograph (DMT A/S). We studied the effects of NOX inhibitor VAS2870 (1 μM) on contractile responses to α_1-adrenergic agonist methoxamine in the absence and in the presence of NO synthase (L-NNA 100 μM) or Rho-kinase (Y27632, 3 μM) inhibitors as well as in the presence of NO donor DEA/NO.

Results. VAS2879 prominently attenuated the contractile responses of DA to methoxamine (30% decrease of the area under the concentration-response curve). L-NNA and Y27632 increased and decreased methoxamine-induced contraction of DA, respectively. L-NNA did not change the effects of VAS2870 and the sensitivity to DEA/NO did not differ in arteries with active and inhibited NOX. Along with that Y27632 eliminated the effects of VAS2879 on DA contractile responses to methoxamine.

Conclusions. We showed that NOX-produced ROS potentiate contractile responses of DA. ROS did not affect the activity of NO-pathway in either endothelial or smooth muscle cells of DA. However, ROS modulate the activity of the Rho-kinase pathway in DA smooth muscle cells. Supported by RSF (project № 19-75-00060).

P.28

COMPARISON OF REGIONAL VS LOCAL ARTERIAL PARAMETERS USING NEW US TECHNOLOGY

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ENVIRONMENT: It has been described that muscular arteries behaviour is different from aorta, and regional parameters like IMT, atherosclerotic plaques burden, PWV-c and endothelial function are related with age and risk factors and are powerful prognostic markers but it is not the case of local parameters like wall shear stress (WSS) , local PWV or beta index in muscular arteries like common carotid artery, only just recently available in the clinical practice.

OBJECTIVE: To analyze the relationship of regional and local arterial parameters with age and its potential use in the clinical practice.

METHODS: We evaluated 100 consecutive patients from April 2019 to February 2020 with a Resona 7 (Mindray) US device with tools to measure IMT, atherosclerotic load, PWV and endothelial function and by means of VFlow Doppler, an innovative multivectorial Doppler technology, we evaluated WSS and with radiofrequency edge detectors, stiffness parameters like PWV and beta index.

RESULTS: IMT remodelling, plaques burden, PWV correlated tightly with age and endothelial function did but inverserly (regression p >0.05). Local carotid parameters like wall shear stress, PWV and beta index were grouped within a range, independently of age. (regression p NS).

CONCLUSION: The evaluation of local parameters has been proposed as markers of arterial disease and they are independent of age which makes easier to detect abnormal values out o frange, early markers of vascular disease, even before atherosclerosis is present. WSS is used for the first time in the current clinical practice.

P.29

Involvement of cannabinoid receptors in regulation of MMPs, cell proliferation and apoptosis in vascular smooth muscle cells

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Objectives: Cannabinoid receptors CB1R and CB2R are expressed in the vascular smooth muscle cells (VSMCs) and may contribute to vascular remodeling process (O'Sullivan, 2015). This study aimed to investigate the implication of CB1R and CB2R in the regulation of matrix metalloproteases MMP2 and MMP9, cell proliferation and apoptosis.

Methods: Primary VSMCs of contractile type were derived from rat aorta. Following compounds were studied: the CB1R agonist arachidonyl-2-chloroethylamide (ACEA), the CB1R antagonist/inverse agonist rimonabant, the CB2R agonist JWH133, the CB2R antagonist/inverse agonist AM630. The cells were treated with compounds simultaneously with IL1α stimulation. MMP2 and MMP9 were analyzed 48h after treatment via gelatin zymography, Western blotting and immunofluorescence. Apoptotic markers FasL, Caspase-3 and TGFbeta1 were used. This experimental setup was repeated using IncuCyte cell imaging to evaluate cell proliferation and apoptosis.

Results: The CB2R agonist JWH133 decreased the activity of proMMP9 ($p<0.05$), abolished IL1α -induced up-regulation of proMMP9/MMP9 proteins, and decreased MMP2 activity by tendency (11%). JWH133 also decreased the number of apoptotic cells ($p<0.05$). Accordingly, CB2R antagonist AM630 did not prevent MMP9 release. CB1R antagonist Rimonabant reduced activity of proMMP9 (35%) and MMP2 (4%) and abolished protein up-regulation of proMMP9/MMP9. CB1R stimulation with ACEA had an ambiguous effect. JWH133 and Rimonabant increased cell proliferation ($p<0,05$) and decreased expression of apoptosis markers FasL and caspase-3.

Conclusions: The CB2R agonist JWH133 and CB1R antagonist Rimonabant prevented release of MMP9 and cell death of VSMCs. Therefore, stimulation of the CB2R or blockade of the CB1R may be favorable by vascular outward remodeling processes.

P.30

Angiotensin II Infusion Leads to Aortic Dissection in LRP8 Deficient Mice

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Background/Objectives: Myeloid cells are crucial for the development of vascular inflammation. Low-density lipoprotein receptor-related protein 8 (LRP8) or Apolipoprotein E receptor 2 (ApoER2), is expressed by macrophages, endothelial cells and platelets and has been implicated in the development of cardiovascular diseases. Our aim was to evaluate the role of LRP8, in particular from immune cells, in the development of vascular inflammation.

Methods. $LRP8^{+/+}$ and $LRP8^{-/-}$ mice (on B6;129S background) were infused with angiotensin II (AngII, 1 mg/kg/day for 7 to 28 day) using osmotic minipumps. Blood pressure was recorded using tail cuff measurements. Vascular reactivity was assessed in isolated aortic segments. Leukocyte activation and infiltration were assessed by flow cytometry of aortic tissue and intravital videomicroscopy imaging. Histological analysis of aortic sections was conducted using sirius red staining.

Results. AngII infusion worsened endothelial-dependent vascular relaxation and immune cells rolling and adherence to the carotid artery in both $LRP8^{+/+}$ as well as $LRP8^{-/-}$ mice. However, only $LRP8^{-/-}$ mice demonstrated a drastically increased mortality rate in response to AngII due to aortic dissection. Bone marrow transplantation revealed that chimeras with LRP8 deficient myeloid cells phenocopied $LRP8^{-/-}$ mice.

Conclusion. AngII-infused LRP8 deficient mice could be a useful animal model to study aortic dissection reflecting the lethality of this disease in humans.

P.31

Von Willebrand Factor Induces Vascular Smooth Muscle Cell Proliferation And Migration Through Low Density Lipoprotein-Related Receptor Protein 4 And αvβ3 Integrin

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Background and Objectives: Von Willebrand factor (VWF) is a plasma glycoprotein involved in primary hemostasis but recent data suggest additional roles beyond hemostasis in angiogenesis and potentially in vascular smooth muscle cell (VSMC) proliferation. Our aim was to investigate how VWF can modulate VSMC proliferation and identified the underlying mechanisms and the *in vivo* pathophysiological relevance.

Methods and Results: Cultured aortic VSMCs proliferation and migration were increased in the presence of VWF. VSMCs treatment with a siRNA targeting α_v integrin or the RGT-peptide blocking $\alpha_v\beta_3$ signaling completely inhibited proliferation. VWF did not bind directly to $\alpha_v\beta_3$ on VSMCs. We identified that VWF A2 domain was able to bind VSMCs. Since the low-density lipoprotein-related receptor protein (LRP) family are known to act as co-receptors we hypothesized the involvement of a member in the signaling pathway. Using the universal LRP-inhibitor (RAP), we confirmed LRP-mediated VSMC proliferation. siRNA experiments and proximity ligation assay staining identified LRP4 as the VWF-counterreceptor on VSMCs and showed co-localization between $\alpha_v\beta_3$ and LRP4. Carotid ligations were applied to VWF+/+ and -/- mice and intimal hyperplasia (IH) was measured. Less VWF-/- mice developed IH compared to VWF+/+ mice. Finally, the proliferative effect of VWF was confirmed in human atherosclerotic lesions from different vessels (aortas, carotids) showing a proximity between VWF and a-SM actin positive cells.

Conclusions: VWF mediates VSMC proliferation through its A2 domain binding to the LRP4 receptor and integrin $\alpha_v\beta_3$ signaling. The decreased IH following vascular injury suggests that targeting VWF-LRP4 interactions may contribute to limit remodeling.

P.32

Non-invasive measures of arteriosclerosis across childhood and adolescence: Insights into the natural history of disease

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Objectives: Non-invasive methodologies for assessing arteriosclerosis, including carotid intimal-medial thickness (cIMT) for assessing subclinical atherosclerosis and carotid-femoral pulse wave velocity (cfPWV) for measuring arterial stiffness, are well established and validated in adults [1, 2]. However, they are less well-described in children. Alternative methodologies, such as aortic IMT (aIMT), may be more appropriate in children provided the natural history of atherosclerotic disease [3]. Previous studies have predominantly applied these methodologies in a narrow age-range of children; methodological differences between studies make inter-study comparison of absolute values difficult. Therefore, we aimed to assess the severity of arteriosclerosis across childhood and adolescence using standardised application of age-appropriate and established methodologies.

Methods: We prospectively recruited 97 healthy children aged 2 to 20 (mean age = 11.2 ± 5.12 years old; stratified into five sex-balanced age groups). cIMT and aIMT were assessed via high-resolution B-mode ultrasonography. cfPWV was assessed via a semi-automated cuff-based device (Sphygmocor XCEL; AtCor Medical, Australia).

Results: aIMT increased with age (9 µm per year [95% CI: 6, 12], $P < 0.0001$), whereas cIMT did not meaningfully increase with age (2 µm per year [95% CI: -1, 5], $P = 0.14$). cfPWV remained relatively stable during early childhood, with an apparent increase from adolescence onwards.

Conclusions: Carotid and aortic atherosclerosis both increase throughout childhood, although this increase is greatest in the aorta. The aorta begins to stiffen during adolescence. Assessment of aortic arteriosclerosis is feasible in childhood and adolescence, and should be prioritised over assessment of carotid atherosclerosis in this age group.

P.33

Changes in blood pressure, pulse wave velocity and augmentation index induced by postural changes and exercise

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OBJECTIVES: To determine how orthostatic changes in body position alter BP and estimation of pulse wave velocity (PWV) and Augmentation Index (AIx), when changing from supine to the sitting position (1). Also, to analyze the effect of a short physical exercise and of physical training status on PWV/AIx in supine and sitting position.

METHODS: Cross-sectional, observational study in 63 voluntary healthy students. Age, height, weight, waist and smoking habits were assessed. We estimated peripheral and central BP, AIx and PWV (brachial oscillometry, AGEDIO, IEM®, Stollberg) after 5´ in supine position (SUP), then after 30´´ in sitting (SIT) and again in sitting position after 25 squats in 30´´ (EXE). A validated questionnaire (Vital sign, https://www.seh-lelha.org/wp-content/uploads/2017/03/GuiaEjercicioRCV.pdf) was implemented to assess chronic physical condition (CPC).

RESULTS: 52,4% were women, mean age was 23.2 years. Systolic, diastolic and median BP rose from 115/67/89 (SUP) to 118/72/93 (SIT) and to 122/67/93 (EXE) mmHg ($p<0.001$ for all comparisons to SUP), PWV was 4.9 (SUP), 4.9 (SIT) and 5.0 (EXE) m/s, with no significant difference. Variables associated with PWV were central BP ($p<0.001$), age ($p<0.001$), gender ($p<0.001$) and AIx ($p=0.04$), but not CPC. Predictors of AIx were heart rate ($p=0.003$), BMI ($p=0.03$) and CPC ($p=0.03$). The latter became more significant in the transition from SUP over SIT to EXE (R^2 of multivariate analysis 0.23, 0.55 and 0.68, respectively).
CONCLUSIONS: Although peripheral BP significantly changed from supine, sitting and post-exercise sitting, PWV remained constant. Chronic physical condition did not affect PWV, but was associated with wave reflection.

P.34

Preeclampsia leads to the delayed development of sympathetic control of the cardiovascular system in the offspring

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Background
Preeclampsia is a common pregnancy disease characterized by hypertension and kidney failure. Recent studies have shown that even in the case of successful delivery, preeclampsia could induce long-term effects in the offspring. Nevertheless, the effects of preeclampsia on the cardiovascular system of the offspring are poorly studied.

Methods
We induced preeclampsia in pregnant rats by L-NAME (nitric oxide synthase inhibitor) supplementation (250 mg/l in drinking water from gestation day 10 to delivery, daily dose 39 mg/kg). The model was verified by the dam's blood pressure (BP) elevation (tail-cuff) and creatinine clearance reduction (metabolic cages) compared to control dams. Male offspring 16-18-day old were used for BP recording (catheter technique under urethane anesthesia); their isolated arteries were studied by wire myography. Adrenergic nerve plexus was visualized by glyoxylic acid staining.

Results
Offspring of dams with preeclampsia had reduced body weight compared to control. They also demonstrated decreased BP (43.3±1.9 vs 50.5±1.4 mmHg) and diminished response to the ganglionic blocker chlorizondamine (2.5 mg/kg); heart rate didn't differ between the groups. The density of the sympathetic innervation of the right atrium and the saphenous artery was reduced in the preeclampsia offspring. Saphenous arteries from preeclampsia offspring had smaller diameter (276±5 vs. 296±7 micron) and maximal contraction force (9.4±0.4 vs. 10.6±0.4 mN) compared to control.

Conclusions
Preeclampsia is followed by the delay in sympathetic nervous system development in the offspring, which is accompanied by structural and functional alterations in the cardiovascular system.
The research was supported by Russian Science Foundation (Grant N 19-15-00210).

P.35

TASK-1 channels play an anticontractile role in rat septal coronary artery under pharmacological blockade of endothelium

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Purpose/Background/Objectives. TASK-1 (TWIK-related acid-sensitive potassium) channels conduct background K^+ currents in smooth muscle cells of pulmonary arteries suppressing vasocontraction. However, the vasomotor role of TASK-1 channels in systemic arteries as well as their regulation by the endothelium are poorly understood. Therefore, we investigated the impact of TASK-1 channels on the regulation of arterial contraction in the presence or absence of the relaxing influence of the endothelium in the brain and the heart, important regions of the systemic circulation.

Methods. Segments of basilar and septal coronary arteries (2-mm long) were isolated from male Wistar rats and mounted in a wire myograph. The functional impact of TASK-1 channels was assessed by the effect of their blocker AVE1231 (1 μM) on contractile responses to the thromboxane A2 receptor agonist U46619. Experiments were performed first on endothelium-intact arteries and then after combined blockade of NO-synthase (L-NNA, 100 μM), cyclooxygenase (indomethacin, 10 μM) and EDHF (TRAM34, 1 μM and UCL1684, 0.1 μM).

Results. AVE1231 did not change basal tone and contractile responses to U46619 of either basilar or septal coronary arteries with intact endothelium. However, incubation with AVE1231 after pharmacological blockade of the endothelium led to the development of basal tone and augmented contractile responses to U46619 in septal coronary arteries, but not in basilar arteries.

Conclusions. TASK-1 channels may play a protective anticontractile role in the coronary circulation under conditions of endothelial dysfunction.

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Carotid artery correlates with aorta reactivity to sympathetic stimulation in healthy individuals and patients with abdominal aortic aneurysm

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Background
Sympathetic stimulation of coronary and carotid arteries leads to vasodilation in healthy subjects, but to vasoconstriction in those with cardiovascular disease. This response has an independent prognostic value for future cardiovascular events. Whether the aorta demonstrates a similar responsiveness is yet unknown. Therefore, this study investigated the relation between the carotid artery and the abdominal aorta diameter response to sympathetic stimulation using the cold pressure test (CPT).

Method
Subjects underwent CPT, while performing simultaneously an ultrasound scan to measure the diameter of the abdominal aorta and the carotid artery. Spearman rho's correlation coefficient was calculated to investigate whether and to what extend the maximal responses of both arteries correlated.

Results
In this interim analysis, 20 healthy young participants, 15 patients with an AAA and 14 healthy older participants were included. Overall, a trend for a positive correlation was found between the carotid artery and the abdominal aorta diameter change (r=0.255, p=0.092) Both arteries demonstrated the same response for 80%, 57% and 36% of respectively the young healthy group, older healthy group and the AAA group, where respectively 70%, 47% and 18% demonstrated dilatation of both arteries.

Conclusion
A correlation exists between the carotid artery and abdominal aorta responsiveness. This supports the potential use of the carotid artery reactivity test to reflect abdominal aorta vascular health in AAA patients.

P.37

An assessment of potential sources of error that may arise in the measurement of carotid-femoral pulse wave velocity

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Objectives: Carotid-femoral pulse wave velocity (cfPWV) approximates aortic stiffness **and** is a predictor of cardiovascular events. Despite the literature highlighting the clinical relevance of cfPWV, there is minimal integration of this parameter in clinical assessments. An underlying reason may stem from potential measurement errors. This paper investigates the potential sources of error in the measurement of cfPWV.

Methods: Participants (n=15, age 30±15 years, 12 female) had supine cfPWV measured using the SphygmoCor XCEL device. Sources of error investigated included: 1) operator experience; 2) poor carotid waveform acquisition; 3) low placement of the leg cuff; and 4) tape measurement of distance. True cfPWV was obtained by averaging twenty cfPWV measurements (regression to the mean). Comparisons were made with regression analysis and Bland-Altman plots.

Results: All cfPWV measurements, for both the experienced and less experienced operator, were within ±0.5 m/s of the true cfPWV when three (but not two) measurements were averaged. cfPWV acquired with a poor carotid waveform and lower placed leg cuff did not significantly differ from the measured cfPWV ($p > 0.05$), however, there were some physiological meaningful errors (cfPWV error > ±0.5 m/s). Excluding four distance measurements, three of which made by the same operator for a single individual, all distance measurements were within 5% of the true distance.
Conclusions: Irrespective of the operators' experience, with good cuff placement and carotid waveform acquisition, three measurements quantifies cfPWV accurately. Measurement error should not be a factor in the lack of clinical uptake of cfPWV.

Key words: arterial stiffness, measurement, pulse transit time, pulse wave velocity.

P.38

Comparison of arterial hemodynamics in early vascular aging (EVA), average vascular aging (AVA) and healthy vascular aging (HVA)

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Background
Large artery stiffening, as indexed by carotid-femoral pulse wave velocity (cfPWV), may vary substantially among individuals. The present study aimed to characterize the arterial mechanical properties of the macro- and microvasculature in subjects with early or healthy vascular aging (EVA, HVA).

Methods
Carotid and femoral pressure and central flow waveforms were noninvasively acquired in a total of 873 community residents (aged ≥50 years, mean age 66.9 years, 69.2% female). They were classified as EVA and HVA, according to the highest and lowest 10% of the cfPWV stratified by 5-year intervals. The remaining 80% were defined as average vascular aging (AVA). Macrovascular and microvascular functions were characterized by aortic input impedance, systemic vascular resistance and wave reflection indices.

Results
EVA subjects had significantly higher prevalence of hypertension and diabetes. In multivariable analysis adjusting for sex, height, weight and mean arterial pressure (MAP), EVA had significantly increased characteristic impedance and reduced arterial compliance. By contrast, for the microvascular functions, systemic vascular resistance (not adjusted for MAP), amplitude of the reflected wave (Pb) and excess pressure integral (XSPI) derived from the reservoir-wave analysis were significantly increased in EVA when compared with HVA or AVA (all $P<0.05$). Primary determinants of HVA included female, lower value of XSPI and SVR, whereas determinants of EVA included male, elevated BP, metabolic syndrome, increased Pb and SVR.

Conclusions
Systemic microvasculature play an important role in interacting with macrovasculature, as evidenced from increased or reduced systemic resistance and wave reflection, in subjects with HVA and EVA.

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THE ROLE OF ADVANCED GLYCATION END PRODUCTS IN VASCULAR AGEING. WHICH PARAMETER IS THE MOST SUITABLE AS BIOMARKER?

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Background Advanced glycation end products (AGEs) are involved into several pathophysiologic processes in vascular diseases, including progressive loss of elasticity of vessel wall (arterial stiffness). Circulating soluble receptor for AGEs (sRAGE) act as a decoy and counterbalanced the harmful properties of AGEs as the natural protective factor (1,2). We compared the role of circulating or skin-deposed AGEs and sRAGE regarding natural course of arterial stiffening.

Methods In a prospective cohort study, we longitudinally followed 536 general-population-based subjects (subsample of Czech post-MONICA study). Aortic pulse wave velocity (PWV) was measured twice (at baseline and after ~8 years of follow-up) using a SphygmoCor device (AtCor Medical Ltd.), and the intraindividual change in PWV per year (ΔPWV/year) was calculated. Concentrations of sRAGE and carboxylmethyl lysine (circulating AGEs) were assessed at follow-up visit by ELISA, while skin AGEs were measured using autofluorescence-based device AGE Reader.

Results Using multiple regressions, we found significant association between ΔPWV/year as dependent variable and both, sRAGE and skin AGEs as independent ones (each on its own model). However, the closest association to ΔPWV/year were found for ratio of these two factors (skin AGEs/sRAGE) [coeff=0.0747 (SE 0.0189), p<0.0001]. In categorized manner, subjects with skin AGEs/sRAGE ratio ≥3.3 showed about two-fold higher risk having PWV/ year ≥0.2 m/sec [adjusted odds ratio was 2.09 (95%CI:1.35-3.22), p=0.001]. In contrast neither circulating AGEs nor circulating AGEs/sRAGE showed any significant relation to PWV/ year.

Conclusions Skin AGEs/sRAGE ratio seems to be more sensitive biomarker of vascular ageing than these single factors themselves or circulation status of AGEs (3).

P.40

Ambulatory Measurement of Carotid Stiffness with a Novel Accelerometric System

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Purpose: Arterial stiffness is a well-established marker for cardiovascular health assessment. Current methods rely on expensive imaging systems and specialised operators to perform local stiffness evaluation from the common carotid artery (CCA) and cannot be used for ambulatory monitoring in chronic disease management. In this study, we compare the performance of a novel accelerometric system, which performs ambulatory non-invasive CCA stiffness measurement, against an ultrasound-based stiffness monitor.

Methods: The accelerometric system with a wearable patch probe derives CCA's diameter parameters from the skin surface vibrations arising out of the arterial wall displacement wave propagated to the skin surface. A subject-specific one-time calibration procedure is used to ensure accuracy, reliability and repeatability. Inbuilt pressure measurement unit of the system estimates blood pressure at CCA. Simultaneously obtained pressure and diameter parameters are used to evaluate various clinically accepted stiffness indices. An in-vivo study was performed on 36 subjects (20-50 years). Measured stiffness indices were compared against those obtained sequentially from an imaging system analysed using Carotid Studio.

Results: Accelerometric-derived diameter waveform was comparable to that acquired using the reference device. Measured group-average end-diastolic diameter and distension values were 5.81±0.53mm and 0.51±0.15mm, respectively. Diameter and stiffness indices (β, Ep, AC, PWV, etc.) were repeatable over continuous cycles (variability<12%). These measures significantly correlated (r^2>0.88, p<0.001) with an acceptable agreement. The one-time calibration remained valid for more than 12 days (error<13%).

Conclusion: The study demonstrated the feasibility and essential functionality of a cost-effective method for long-term stiffness monitoring with potential applications in ambulatory healthcare devices.

P.41

Measurement of pressure-dependent intra-beat changes in carotid pulse wave velocity using image-free fast ultrasound

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Background: The clinical significance of pressure-dependent intra-beat changes in local pulse-wave velocity (C) has recently come to light (1). While reported methods require arterial pressure and diameter measurements from a single site to assess intra-beat changes in C, we present an image-free fast ultrasound device that performs this by capturing diameter waveforms from two proximal locations on an artery.

Methods: The functionality was assessed on eight normotensive participants (26±4 years). By perturbing blood pressure through a short duration moderate lower body negative pressure intervention (2), C_D and C_F pulse wave velocities corresponding to diastolic and 80% of peak pressure were measured from the carotid artery. Human NIBP system (ADInstruments, India) was used for monitoring continuous pressure.

Results: The device captured dual-diameter waveforms and evaluated C_D and C_F, with a beat-to-beat variation <8% during baseline. C_D was smaller than C_F ($p<0.001$), 4.2±0.5 m/s versus 4.6±0.7 m/s during baseline and 3.7±0.6 m/s versus 3.9±0.7 m/s during intervention. Concomitant to the drop in group-average diastolic (17%) and systolic (18%) pressures during the intervention, C_D and C_F dropped by 14% and 16%, respectively. The statistically significant correlation ($r>0.6$, $p<0.001$) of C_D and C_F with the diastolic and systolic pressures for each individual was preserved even after adjusting for heart-rate.

Conclusions: The device demonstrated its functionality and reliably measured the incremental nature of C. Its pressure-dependent intra-beat variations and inter-beat dynamics during the intervention concurred with literature. Further studies are underway to demonstrate the potential use of the device in vascular research and clinical applications.

P.42

The effects of chemotherapy on arterial stiffness in patients with Hodgkin lymphoma

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Introduction:
Malignancies and cardiovascular disease are the two leading causes of mortality worldwide[1]. While there is extensive literature describing the cardiotoxic effects of chemotherapy on left ventricular systolic function[2,3], there is only little evidence regarding chemotherapy effects on a vascular functional parameters.
Purpose:
Our aim was to investigate the effect of chemotherapy in aortic stiffness in patients with Hodgkin lymphoma (HL), a malignancy with known high metabolic burden.

Methods:
Thirty two patients (mean age 65 years) with HL underwent therapy with Doxorubicin, bleomycin, vinblastine and dacarbazine (ABVD). The interim of their treatment was set at 1 to 3 days prior to initiating the 3rd chemotherapy cycle. All patients were reassessed six months after chemotherapy completion. Blood pressure (BP) and carotid-femoral pulse wave velocity (c-f PWV) as an index of aortic stiffness were measured at baseline, interim and after completion of chemotherapy.

Results:
Figure illustrates c-f PWV changes from baseline to interim and 6 months after completion of chemotherapy in patients with HL and patients with NHL. As figure shows, c-f PWV decreased at treatment interim (by 0.34 m/s) and remained significantly decreased at 6 months after chemotherapy completion (by 0.52 m/s (overall $P<0.001$, by ANOVA) The progressive decrease in c-f PWV remained statistically significant after adjustment for age, systolic BP and diabetes ($F=4.853$, $P=0.005$). Changes in systolic and diastolic BP from baseline, to interim and 6 months post therapy were insignificant (decrease by 4 mmHg and 1 mmHg respectively, compared to baseline, all $P>0.05$).

Conclusion:
Carotid-Femoral PWV decreased during and post chemotherapy in patients with Hodgkin lymphomas, suggesting that arterial stiffness improves with chemotherapy in these patients. Considering that systemic inflammation influences changes in aortic systems and that HL is an inflammatory rather than a solid tumor, the significant improvement in arterial stiffness after ABVD therapy imply that the metabolic burden of the malignancy may play a significant role in the arterial stiffness progression.

P.43

The association between early vascular aging and cyclothymic affective temperament

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Objectives: Affective temperaments (depressive, anxious, irritable, hyperthymic, cyclothymic) are regarded as the biologically stable core of personality, and accumulating data implies their relationship with cardiovascular diseases. There are currently limited data on the association of affective temperaments and early vascular aging. The aim of our study was to assess the potential relationship of affective temperaments and vascular age, as assessed by coronary CT.

Methods: In our current cross-sectional study, 209 patients referred to coronary computed tomography angiography (CCTA) due to suspected coronary artery disease (CAD), were included. After the evaluation of medical history and demographic parameters, all patients completed the Temperament Evaluation of Memphis, Pisa, Paris, and San Diego Autoquestionnaire (TEMPS-A) and the Beck Depression Inventory (BDI). Vascular age was estimated using coronary artery calcium (CAC) score and we calculated its difference from chronological age for each patient. Linear regression analysis was applied to identify predictors of early vascular aging in the entire cohort and in male and female sub-populations, separately.

Results: The independent predictors of early vascular aging were female sex (B = -10.82 [95%CI: -15.30 – -6.33]), diabetes mellitus (B = 7.16 [95%CI: 1.20 – 13.12]) and dyslipidemia (B = -8.28 [95%CI: 3.94 – 12.62]). Further assessing gender differences, cyclothymic temperament score proved to be an independent predictor of early vascular aging in women (B = 0.89 [95%CI: 0.04 – 1.75]), while this association was absent in men.

Conclusions: Our results suggest that cyclothymic affective temperament contribute to early vascular aging in women.

P.44

Application of an algorithm developed for measuring gastrointestinal motility to the assessment of arterial mechanical properties.

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Background: GIQuant (Motilent, London) is an algorithm for analysing cine-MRI, using the displacement fields generated by registration of indivisdual ciné frames. It produces a summary of how much movement occurs ("motility") for anatomical locations within a reference image [1]. Although it is used clinically for quantifying gut motility [2], it can process any ciné-MRI. In this study, we explore the hypothesis that it can be used to examine the functionality of blood vessels.

Methods: Using ciné-MRI of the abdomen, obtained in initially to assess small-bowel motility in patients with Crohn's disease, we tested the feasibility of using GIQuant to examine the mechanical properties of blood vessels. In such data, coronal slices often intersect the abdominal aorta and the common iliac arteries. The reference images were manually segmented (see Fig1A) for statistical analysis of the motility scores.

Results: We compared the mean motility score in the common iliac arteries, finding that the inter-subject, is greater than the intra-subject variability (Fig1B). Additionally, motility in the right and left common iliacs is not correlated with that of the abdominal aorta (Fig1C). When assessed spatially along the abdominal aorta, the motility score is correlated with position in a physiologically plausible manner, showing a general decrease in the caudal direction (Fig1D-E).

Conclusions: It appears that GIQuant can be used to provide biologically meaningful information about blood vessel properties. Further validation work will use this technique to examine function in patients with bicuspid aortic valves, and to examine its predictive value for aortic aneurysm prognosis.

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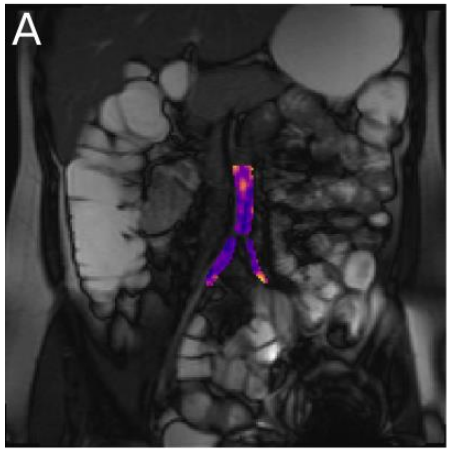
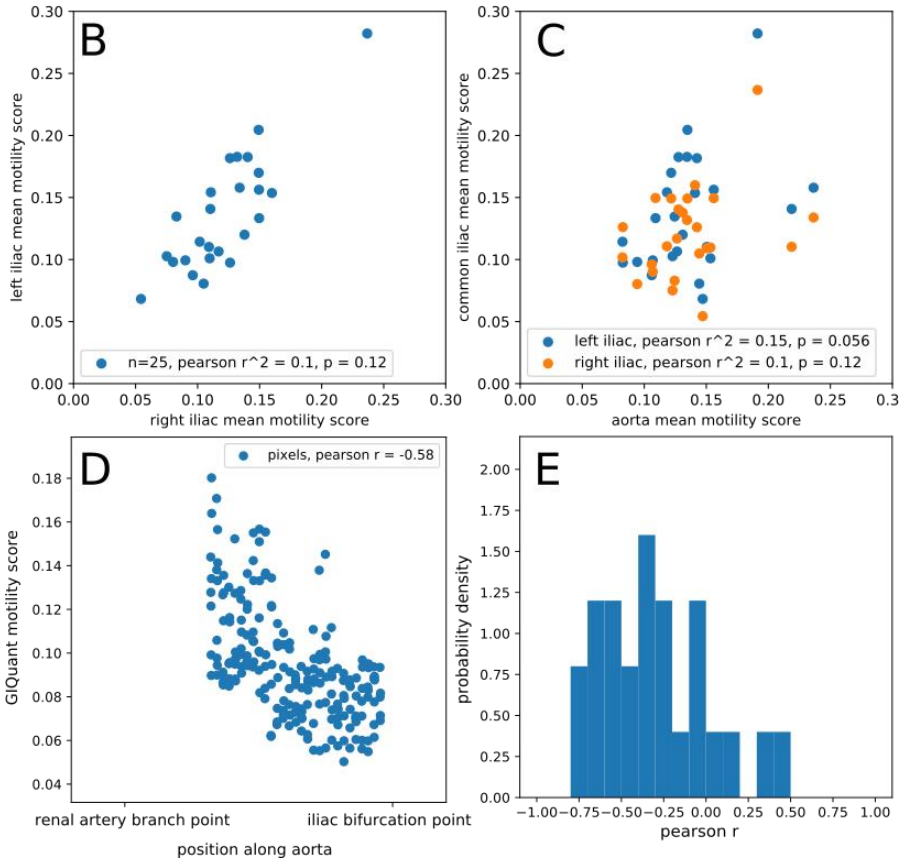


A. Example reference image, with abdominal aorta and common iliacs false-coloured according to GIQuant motility score.
B-C. Scatter plots showing mean motility scores for both common iliacs (B) and comparisons with aorta (C).
D. Scatter plot showing pixelwise motility score against craniocaudal position, for the image shown in (a)
E. r- values from n=25 subjects treated as per (D)



P.45

Characterization of the microcirculatory response to gravity-induced changes using thermal imaging

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[1]School of Medical Engineering, Afeka Tel-Aviv Academic College of Engineering, [2]School of Electrical Engineering, Afeka Tel-Aviv Academic College of Engineering, [3]Department of Industrial Engineering, Tel-Aviv University, [4]Yazmonit ltd. , [5]Department of Vascular Surgery, Sheba Medical Center

Objective: The goal of this study was to characterize the changes in the palm's blood distribution in response to a decrease in blood pressure due to gravity-induced changes, using thermal imaging.

Methods: Thermal hands images were taken from ten healthy volunteers, without any known vascular pathologies, in three different stages: baseline, gravitation and recovery. In the baseline stage the hand was set on a table, at heart height. During the gravitation stage one hand was placed 40 cm above the table for 10 minutes, while the second hand was stayed on the table. The recovery stage, in which both hands were placed back on the table, was recorded for 10 minutes. Thermal images of both hands were taken every ten seconds throughout the experiment.

Results: Mean skin temperatures were increased during hand elevating in both the palm center and the distal phalanx of the middle finger by 2.57 ˚C and 3.33 ˚C, respectively. This increase was significant and remained high during the recovery period ($p<0.01$). A similar effect was also observed with the other hand, which remained on the table.

Conclusions: The temperature increase of the palm during gravity conditions reflects blood perfusion compensation due to high local oxygen consumption during decrease in local blood pressure. The bilateral effect indicates the central nervous system involvement. Thermal imaging allows characterization of the palm's blood distribution under gravitational conditions. Since this technique is noncontact and safe, it could be useful for assessment of blood supply during physical effort.

KEYWORDS
blood flow; gravitational effect; thermal imaging

P.46

Assessment of intraplaque hemorrhage by photoacoustics imaging (PAI): first in-vivo human validation study

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Aim: To validate a photoacoustic imaging (PAI) system, for the identification of intraplaque hemorrhage, comparing it with MRI and histology (gold standard).

Methods: 25 patients with carotid stenosis>70% and clinical indication to tromboendoarterectomy were recruited. Angio-MRI for intraplaque hemorrhage assessment (Cube sequence) was performed. PAI clips (5 seconds, Frame rate 1000/sec, 3 to 15 per patient) were acquired. Each clip was scored for the presence of PAI signal by means of an integrated scoring system (semiquantitative, from 0 to 12). Semiquantitative grading scales were used to assess plaque histological features of hemorrhage and vulnerability.

Results: 18 patients had no missing MRI, PAI and histology data and were included in this analysis. Mean age was 73 ± 8 years, 60% men, 80% Caucasians, 92% hypertensives, 60% with a previous stroke. At histology, only 3 plaques out of 21 showed no signs of intraplaque hemorrhage, 4 showed small hemorrhage, while 14 (67%) showed large hemorrhages. PAI score (best cut-off $\geq$4) correctly classified 14 out of 18 patients (Sensitivity=73.3%, specificity=100%, AUC=0.867). MRI performance was substantially similar, with 12 patients correctly classified (sensitivity=60%, specificity=100%, AUC=0.800), with a non-significant difference in AUC compared to PAI (p=0.420).

Conclusions: In this first in-vivo human study, PAI is able to identify histological intraplaque hemorrhage with an excellent specificity and acceptable sensitivity, equivalent to MRI. The very high specificity, with a low number of false positives, make PAI a good candidate for evaluation of plaques prior to surgery to i.e. reinforce the decision to perform surgery.

P.47

Feasibility evaluation of imaging-free ultrasound technology to measure diameters of brachial and radial arteries for assessment of endothelial function

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Background
Ultrasonographic imaging to record changes in peripheral arterial diameter associated with Flow mediated dilatation or Low flow mediated constriction is routinely used to assess various facets of vascular endothelial function. Imaging poses many challenges including requirement of costly ultrasound machines, trained manpower to perform imaging and effort-intensive steps to analyse the images subsequently using manual or automated methods. We tested the feasibility and validity of using an imaging-free technology to record resting arterial diameters of brachial and radial arteries.

Methods
Eight healthy volunteers initially underwent ultrasonographic imaging (M7, Mindray; Shenzhen, P.R. China) of brachial artery and proximal radial artery. The brachial and radial artery 'zones' thereby identified through imaging were surface marked on subject's arm. Imaging-free ARTSENS® Pen device (1) (Healthcare Technology Innovation Centre, IIT Madras, India) consisting of highly integrated hardware for operating a single element broadband ultrasound transducer (centre-frequency = 5 MHz, spatial half angle < 1.3 degrees, diameter = 5 mm) in pulse-echo mode was used to track detectable arterial wall motion and measure end-diastolic diameters from previously identified brachial and radial 'zones'.

Results
End-diastolic diameters measured by ARTSENS® Pen decreased significantly on moving from brachial to radial zone identified by imaging (4.34 ± 1.07 mm vs 2.05 ± 0.43 mm; P <0.0001) and correlated strongly with imaging-based measurements (r = 0.93; P<0.0001).

Conclusion
ARTSENS® Pen device offers feasible and valid imaging-free solution to measure peripheral arterial diameters which could potentially be employed for assessment of vascular endothelial function.

P.48

Ultrasound-based velocity and acceleration of the carotid atheromatous plaque in asymptomatic patients with moderate and severe stenosis

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Purpose
The purpose of this study was to investigate differences in ultrasound-based velocities and accelerations of the carotid atheromatous plaque between asymptomatic patients with moderate and severe stenosis, based on the assumption that plaque motion features are sensitive to cardiovascular health status.

Methods
The dataset used consists of 38 sequences of B-mode images (videos) of carotid atheromatous plaque of asymptomatic patients. Among the examined carotid arteries, 27 had severe stenosis degrees (>70%) and 11 had moderate stenosis degrees (<70%). Plaque motion estimation was based on an adaptive block matching methodology which incorporates Kalman filtering update strategies. Plaque velocity and acceleration were calculated by differentiating displacement and velocity, respectively. The two-tailed t-test was used to assess statistically significant differences.

Results
Figure 1 shows examples of plaque velocity and acceleration waveforms. Radial and longitudinal plaque velocities were similar between moderate and severe cases (0.43±0.15 mm/s and 0.73±0.18 mm/s, respectively, vs. 0.37±0.21 mm/s and 0.65±0.20 mm/s, respectively; p-values 0.35 and 0.27, respectively). Radial and longitudinal plaque accelerations were significantly higher in moderate compared to severe stenosis cases (7.07±4.64 mm/s^2 and 10.08±5.22 mm/s^2, respectively, vs. 3.17±2.25 mm/s^2 and 5.05±2.96 mm/s^2, respectively; p-values=0.00 for both cases). In moderate stenoses, longitudinal velocities, but not accelerations, were significantly higher than radial ones, whereas in severe stenoses, longitudinal velocities and accelerations were significantly higher than radial ones.

Conclusions
Ultrasound-based velocities and accelerations can characterise biomechanical phenomena of the carotid plaque. Accelerations can differentiate between moderate and severe plaque.

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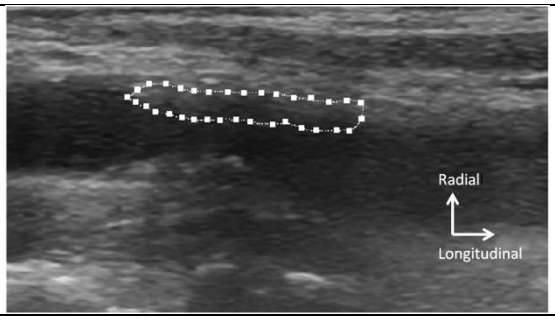
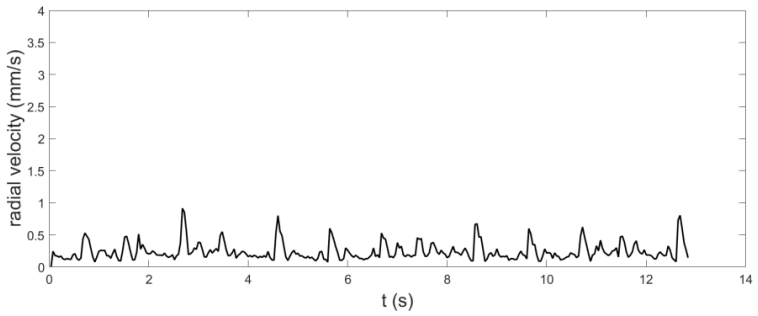
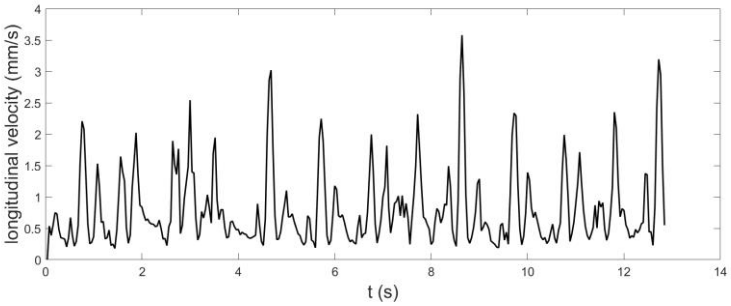


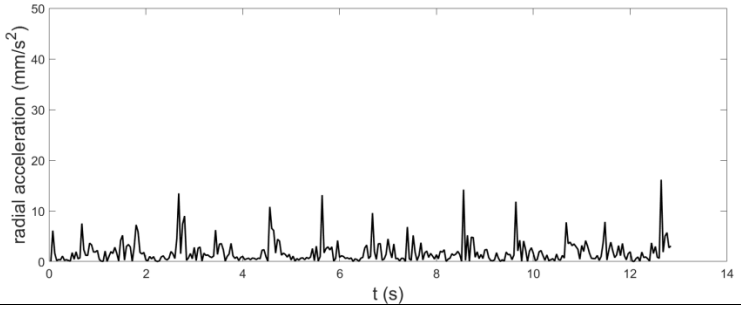
(a)



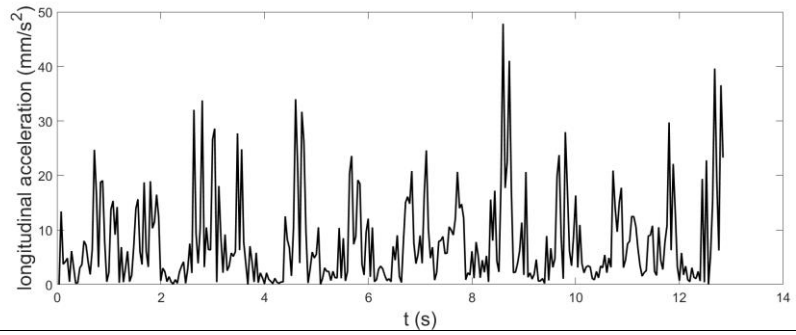
(b)



(c)



(d)



(e)

Figure 1. (a) Example of ultrasound image of the carotid artery with the atheromatous plaque outlined in white. (b), (c) waveforms of radial and longitudinal velocities, respectively, of the plaque in (a). (d), (e) waveforms of radial and longitudinal accelerations, respectively, of the plaque in (a).

P.49

Aortic root longitudinal strain by speckle-tracking echocardiography: comparison with cardiac magnetic resonance and predictive value in Marfan syndrome patients

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Background
Low longitudinal strain of the ascending aorta (AAo) by cardiac magnetic resonance (CMR) predicts dilation and aortic events in Marfan syndrome (MFS) [1], possibly reflecting aortic stiffness [2]. Speckle-tracking is established for cardiac deformation, but proximal aorta applications are challenging due to wall thickness and substantial motion. We aimed to validate a purpose-specific speckle-tracking tool for root longitudinal strain analysis by comparison with CMR-derived AAo longitudinal strain and as predictor of dilation in MFS patients.

Methods
CMR feature-tracking [1] and echocardiography speckle-tracking where applied to 25 MFS patients free from previous aortic surgery by a single observer blind to clinical data. For echocardiography, two regions of interests were manually created covering both walls in a parasternal long-axis view and tracked along the cardiac cycle. Longitudinal strain was computed as the average of maximum increase in relative distance of several sub-regions covering both walls. Aortic diameter was measured on CMR images.

Results
Both techniques were successfully applied to all patients. Aortic root longitudinal strain by echocardiography was linearly related to CMR-derived AAo longitudinal strain (R=0.573, p-value=0.003, Figure 1A) and was higher (20.4±8.4 vs 10.5±3.8), especially at higher absolute values (Figure 1B). After a mean follow up of 45±13 months, aortic root diameter growth rate was 0.27±0.3 mm/year. In multivariable analysis corrected for root diameter and heart rate (p-value=0.083 and 0.005, respectively), baseline longitudinal strain by echocardiography was independently related to progressive dilation (B=-0.017, p=0.005).

Conclusions
Aortic root longitudinal strain by echocardiography is related to CMR-derived AAo longitudinal strain and is an independent predictor of progressive dilation in MFS patients.

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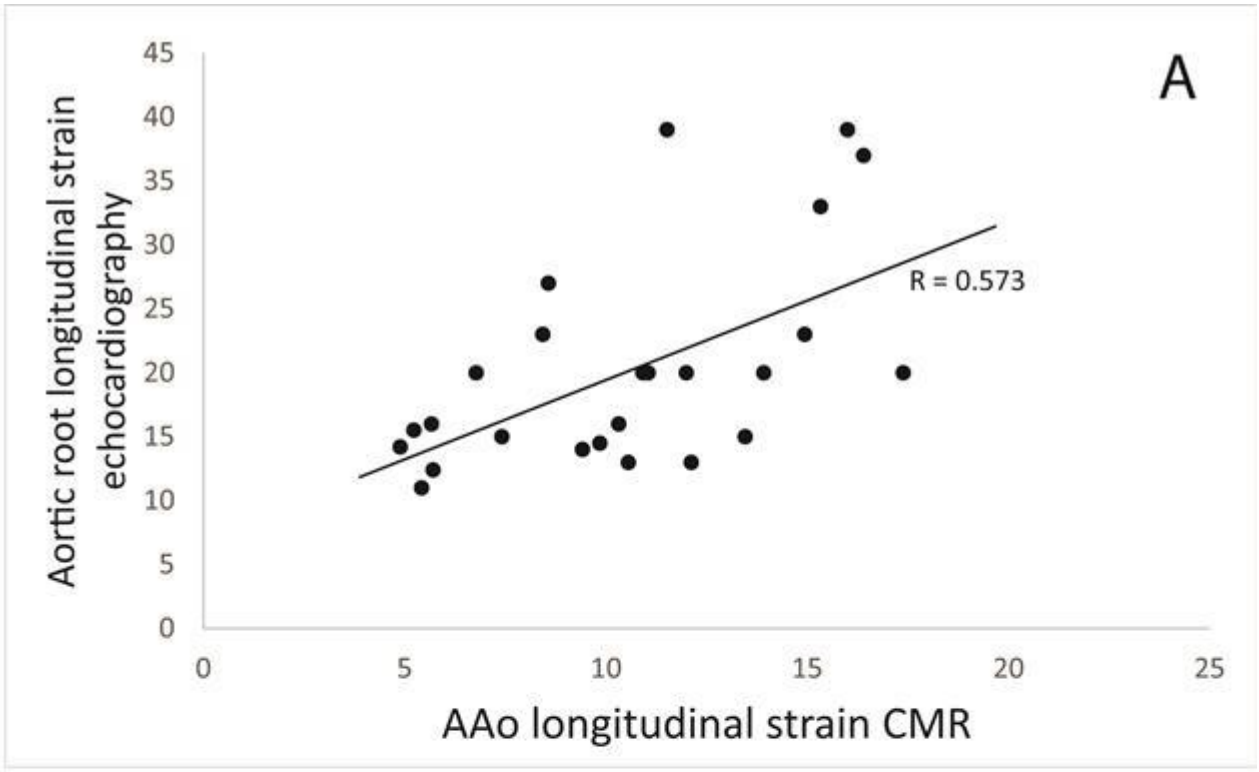
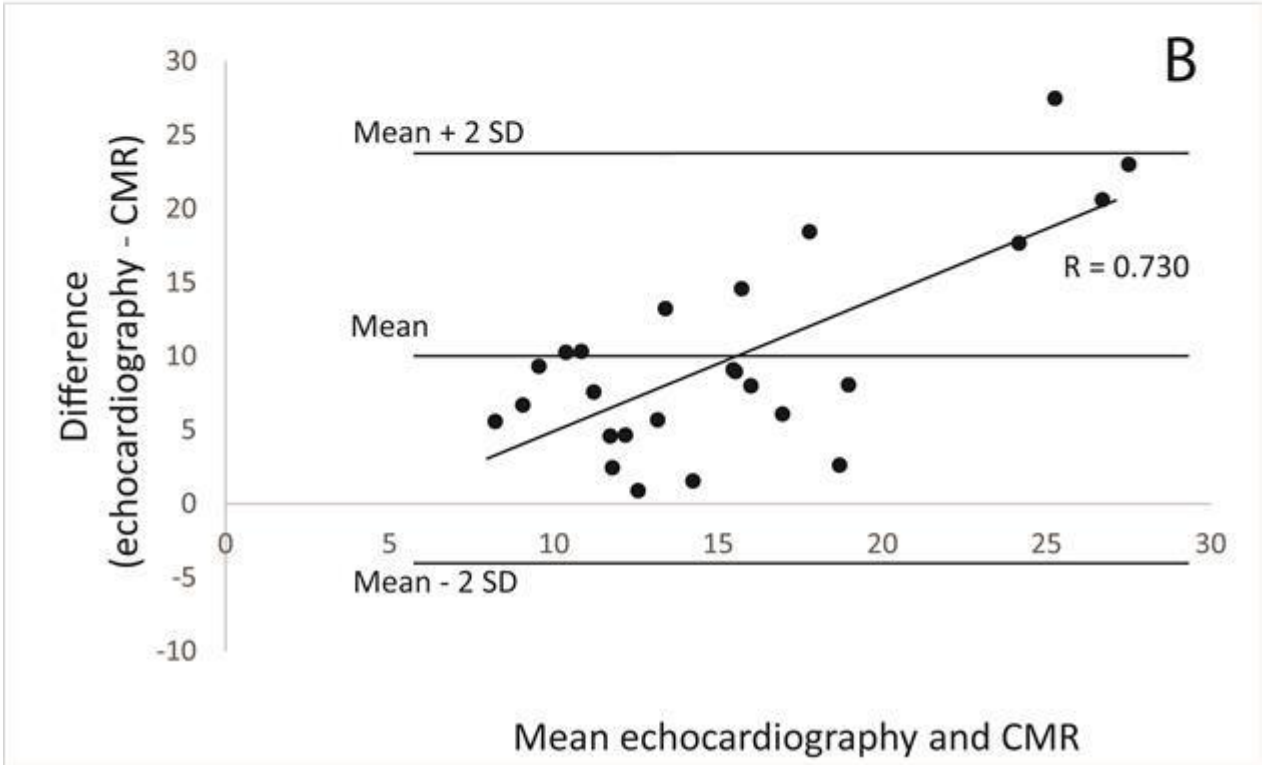

A
Aortic root longitudinal strain echocardiography
AAo longitudinal strain CMR
R = 0.573
0
5
10
15
20
25
30
35
40
45


B
Difference (echocardiography - CMR)
Mean echocardiography and CMR
Mean + 2 SD
Mean
Mean - 2 SD
R = 0.730
30
25
20
15
10
5
0
-5
-10

P.50

Radial artery phenotyping in fibromuscular dysplasia through ultra-high frequency ultrasound: a radiomic approach

Miss Federica Poli[1], **Miss Rosa Maria Bruno[1,2]**, Mr Francesco Faita[3], Mr Hakim Khettab[2], Mr Michel Azizi[4], Mr Saverio Vitali[5], Mr Mirco Cosottini[1,5], Mr Davide Caramella[1,5], Mr Lorenzo Ghiadoni[1], Mr Stefano Taddei[1,5], Mr Pierre Boutouyrie[6], Mr Alexandre Persu[7], Mr Xavier Jeunemaitre[4], Mr Aurélien Lorthioir[6]
[1]Università Di Pisa, [2]INSERM, U970, Paris Cardiovascular Research Center –PARCC, [3]Istituto di Fisiologia Clinica, CNR Pisa, [4]Université Paris-Descartes, [5]Azienda Ospedaliero Universitaria Pisana, [6]APHP, Hôpital Européen Georges Pompidou, [7]Université Catholique de Louvain

Rationale and aim: This study is aimed at identifying possible patterns of vascular wall disarray and remodeling in radial arteries of patients with fibromuscular dysplasia (FMD), by means of ultrahigh frequency ultrasound (UHFUS).

Methods: UHFUS scans of the radial arteries and of 30 FMD patients and 30 healthy controls were obtained by VevoMD (70 MHz probe, FUJIFILM, VisualSonics, Toronto, Canada). 10 end-diastolic frames for each subject were analyzed. 74 radiomic features and 4 engineered parameters were extracted: intima-media thickness (IMT) and adventitia thickness (AT), an adjunctive acoustic interface for each layer (IMT and AT triple signal). The extracted parameters were used to train classification models, using Support Vector Machine Linear (SVM), K-Nearest Neighbors (KNN), Logistic Regression, Linear Discriminant Analysis (LDA). The models were then tested on an independent validation population (38 FMD patients and 28 healthy subjects).

Results: IMT (185±46 vs 168±37, p=) and AT (104±34 vs 96±35, p=0.004) were significantly higher in FMD than in controls. IMT and AT triple signal were also more frequent in FMD than in control images (p< for both). The most accurate classification models were LDA (sensitivity=0.67, specificity=0.76, accuracy=0.71, AUC=0.71) and Logistic Regression (sensitivity=0.71, specificity=0.72, accuracy=0.71, AUC=0.71). The models showed and accuracy of about 70% when tested on the validation population.

Conclusions: Wall ultrastructure of radial arteries of FMD patients is extensively altered: IMT and AT are thickened and the first and/or second layer of the arterial wall is splitted, showing a triple signal feature. Radiomic descriptors combined with engineered parameters allow to distinguish between radial images from FMD patients and controls with a 70% accuracy.

P.51

Development of a diagnostic prediction score to estimate the probability of intraplaque haemorrhage on carotid magnetic resonance imaging in patients with recent cerebrovascular disease: the Plaque At RISK (PARISK) study.

This abstract has been withdrawn by the author.

P.52

Simultaneous investigation of structural and functional parameters of carotid artery plaque using routine B-mode ultrasonography: the Plaque At Risk (PARISK) study.

This abstract has been withdrawn by the author.

P.53

Ascending aorta diameter and pulse wave velocity are increased and local hemodynamic is disrupted in patients with blunt traumatic thoracic aortic injury treated by TEVAR.

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Background
Thoracic endovascular aortic repair (TEVAR) is becoming the preferred treatment option to repair the proximal descending aorta after rupture following blunt traumatic injury. However, hemodynamic and mechanic implications of this intervention are poorly understood. Exploiting the possibilities of 4D flow magnetic resonance imaging, hemodynamics, stiffness and local dilation in the ascending aorta in patients following aortic repair by TEVAR are studied.

Methods
Fifteen apparently healthy individuals who underwent TEVAR implantation after traumatic descending aortic injury and 44 healthy volunteers (HV) underwent 4D flow-MRI. Ascending aorta pulse wave velocity was computed [1]. Moreover, at eight planes equally distributed in the ascending aorta systolic flow reversal ratio, i.e. relative amount of backward flow during systole, and in-plane rotational flow, measuring the strength of helical flow, were computed [2,3].

Results
TEVAR patients and HV did not differ in terms of age, sex, BSA and blood pressure (Table 1). However, compared to HV, TEVAR patients showed reduced in-plane rotational flow in the distal ascending aorta in patients with TEVAR and increased backward systolic flow in the whole ascending aorta (Figure 1). Patients with TEVAR had a stiffer ascending aorta, with pulse wave velocity higher compared control (7.8±4.2 vs 5.3±1.9, p=0.004). Finally, aortic root and ascending aorta diameters were larger in TEVAR patients compared to HV (Table 1).

Conclusions
The implantation of TEVAR in apparently healthy individuals after traumatic rupture of the proximal descending aorta is associated with altered hemodynamics, higher stiffness and larger aortic diameter in the region proximal to the TEVAR.

(figures on next page)

	Healthy volunteers	TEVAR patients	p-values
N	44	15	
Age [years]	40±12	43±10	0.392
Sex [% male]	66	80	0.075
Years from intervention [years]	N/A	10.3±6.4	N/A
Body surface area [m^2]	1.87±0.15	1.96±0.23	0.088
Systolic blood pressure [mmHg]	127±19	131±15	0.389

Diastolic blood pressure [mmHg]	70±11	74±9	0.198
Aortic root diameter [mm]	31±4	34±4	0.026
Ascending aorta diameter [mm]	28±4	32±3	0.003
Ascending aorta PWV [m/s]	5.3±1.9	7.8±4.2	0.004

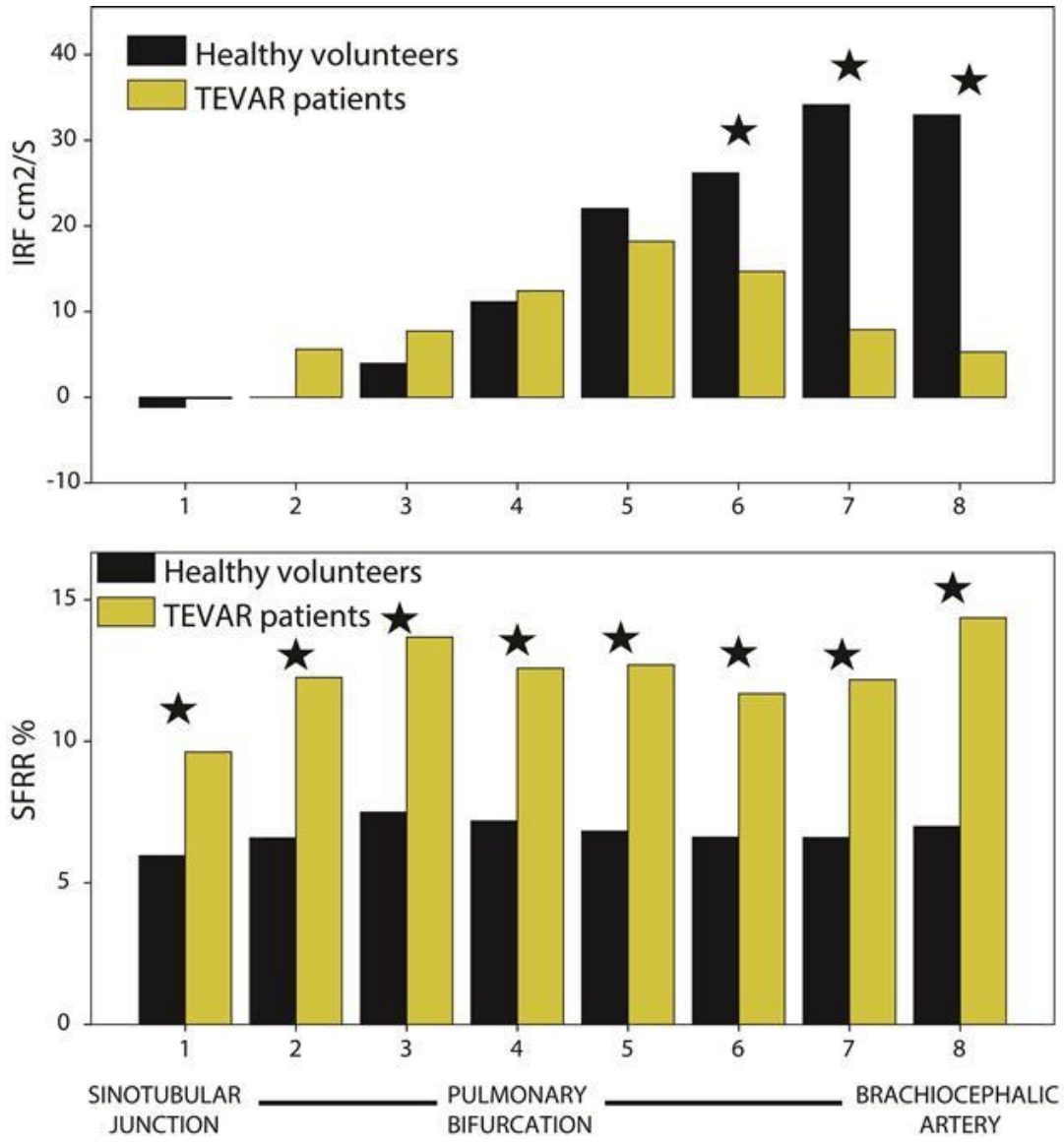

Figure 1: In-plane rotational flow (IRF, top) and systolic flow reversal ratio (SFRR, bottom) in healthy volunteers (black) and TEVAR patients (yellow) in the proximal (planes 1 to 4) and distal (planes 5 to 8) ascending aorta. Stars show statistically-significant differences ($p<0.05$).

P.54

Biomarkers and haemodynamic Predictors of Left Atrial Strain in Early Hypertension

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Background: In the early stages of atrial remodeling, increased aortic stiffness might indicate atrial fibrosis. Objective: This study aimed to investigate the association between left atrial (LA) mechanical function, assessed by two-dimensional speckle tracking echocardiography and aortic stiffness and whether this association is medicated by circulating biomarkers in early hypertension.

Methods: We studied 34 patients, mean age 47.5±11.6 years, 56% females. All parameters for arterial stiffness including 24 hour central systolic pressure, augmentation index and pulse wave velocity were measured non–invasively using Mobil-O-graph PWA. All patients underwent standard two - dimensional echocardiography with Spackle tracking analysis for left atrium (LA). Circulating biomarkers including renin, aldosterone and morning cortisol were measured using ELISA.

Results: LA strain showed a significant relationship with central aortic systolic pressure; 24-hr($r=-0.46$, $p<0.05$), daytime($r=-50$, $p<0.05$) and nighttime ($r=-55$, $p<0.01$) with no relationship with central diastolic or brachial pressures. There was a significant positive association between LA strain and aldosterone ($r=0.49$, $p<0.05$), aldosterone-renin ratio($r=0.30$, $p<0.05$) and cortisol ($r=0.66$, $p<0.01$) with no relationship observed with plasma renin activity. In a stepwise regression model, central systolic pressure, aldosterone and cortisol emerged as predictors of LA strain independent of age, gender, brachial pressure and LAVi.

Conclusions: In mild hypertension, central but not brachial systolic pressure determines LA remodeling which in turn may be mediated by circulating aldosterone and cortisol. This study highlights the important role of circulating biomarkers in mediating aortic stiffness and LA fibrosis in early hypertension.

P.55

Dietary nitrate prevents progression of carotid subclinical atherosclerosis through BP-independent mechanisms in patients with or at risk of type 2 diabetes mellitus: results from the double-blind, randomized-controlled, factorial Vasera trial

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Background: Epidemiological and animal studies suggest the potential of dietary nitrate (NO3-) to inhibit atherogenesis. Spironolactone may improve arterial stiffness. We tested if 6 months' intervention with dietary nitrate and spironolactone could affect carotid subclinical atherosclerosis and stiffness versus placebo/doxazosin, to control for blood pressure (BP), in a population with or at risk of type 2 diabetes [1].

Methods: A subgroup of participants in our double-blind, randomized-controlled, factorial VaSera trial were randomized to nitrate-containing beetroot juice or nitrate-depleted juice, and spironolactone or doxazosin. Ultrasound for carotid diameter (CD, mm) and intima-media thickness (CIMT, mm) was performed at baseline, 3- and 6-months. Carotid stiffness (CS, m/s) was estimated from aortic pulse pressure (Arteriograph®) and carotid lumen area. Data was analysed by modified intention-to-treat and mixed-model effect, adjusted for confounders.

Results: 93 participants had a baseline evaluation; 86% had follow-up data. No statistical interactions occurred between the juice and drug arms. BP was similar between the juices and between the drugs. CIMT was significantly lower following nitrate-containing, compared with placebo juice [-0.06 (95% Confidence Interval -0.12, -0.01), p=0.022], with no effect on CD. CS reduction was similar between juices [-0.38(-0.67, -0.10) with placebo, -0.13 (-0.42, 0.16) with active juice] and the drugs [-0.30(-0.58, -0.02) with doxazosin, -0.21(-0.51, 0.09), with spironolactone]. No differences were detected between spironolactone or doxazosin on CIMT and CD.

Conclusion: 6 months' intervention with dietary nitrate influences vascular remodelling, but not carotid stiffness or diameter. Neither spironolactone nor doxazosin had a BP-independent effect on carotid structure and function.

P.56

Differences in vascular effects between one session of moderate-intensity continuous physical exercise and high-intensity interval physical exercise in individuals with high blood pressure

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[1]*InCor HC FM USP*

Purpose: To compare augmentation index (AIx) between one Moderate-intensity continuous physical exercise (MICPE) and one High-intensity interval physical exercise (HIIPE) session in normal/high normal blood pressure (BP) (120-140 for systolic and 80-90mmHg for diastolic). Additionally, to compare two AIx methods (SphygmoCor® and Arteriograph®)[1].

Methods: Exercise intensity and energy expenditure (equalizing) were according to the cardiopulmonary stress test. Individuals were randomized to exercise sessions, performed as cross-over. AIx were analyzed at baseline, immediately after and 24hours after MICPE and HIIPE session and compared among all times. ΔAIxHIIPE (AIxHIIPE - AIxBaseline) and ΔAIxMICPE were calculated. Correlation and agreement analysis was performed between AIx methods.

Results: Individuals (n=23; 78% women; 48 ± 1 years; systolic/diastolicBP = 125 ± 2/84 ± 1mmHg) had lower AIxSphygmoCor® at MICPE compared to baseline and to 24hours MICPE (27.2 ± 2.2 vs 32.8 ± 1 and 31.0 ± 2.5%; p<0.01). AIxSphygmoCor® was lower in HIIPE than other times (23.2 ± 2.4 vs baseline 32.8 ± 1.9 p<0.01; vs MICPE 27.2 ± 2.2; p=0.039; vs 24hours MICPE 31.0 ± 2.5; p<0.01 and vs 24hours HIIPE 32.2 ± 2.0%; p <0.01). AIxArteriograph® was lower in HIIPE (16.0 ± 3.7%) than baseline (28.9 ± 3.4%; p=0.001), 24hours MICPE (25.7 ± 4.0 %; p=0.008) and 24hours HIIPE (29.5 ± 3.9%; p=0.005). ΔAIxHIIPE was greater than ΔAIxMICPE (-9.37 vs -5.15; p=0.028). AIxArteriograph® showed a positive correlation with AIxSphygmoCor® (r=0.793; p<0.01) and showed agreement.

Conclusion: Regardless of intensity, one exercise session improves AIx. The effect seems to be greater after HIIPE than MICPE.

P.57

Acetylsalicylic acid reduces passive aortic wall stiffness and cardiac remodelling in a mouse model of advanced atherosclerosis

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Background: Acetylsalicylic acid (ASA) is used in secondary prevention of cardiovascular disease (CVD) because of its antithrombotic effects. We investigated whether ASA has additional therapeutic value by preventing the progression of inflammation and cardiovascular remodelling in mice with stable atherosclerosis (*ApoE*$^{-/-}$) and in a model of arterial stiffness with advanced unstable atherosclerotic plaques (*ApoE*$^{-/-}$*Fbn1*$^{C1039G+/-}$ mice).

Methods: Female *ApoE*$^{-/-}$ and *ApoE*$^{-/-}$*Fbn1*$^{C1039G+/-}$ mice were fed a Western diet (WD). At 10 weeks WD, the mice were divided in 2 groups receiving ASA (5mg/kg/day) via the drinking water or plain water (control) for a period of 15 weeks. Echocardiograms were performed at 10, 17 and 25 weeks WD. At the end, blood pressure was measured via tail-cuff and blood samples were taken. The aorta and heart were collected for histology.

Results: *ApoE*$^{-/-}$*Fbn1*$^{C1039G+/-}$ mice showed an increased neutrophil-lymphocyte ratio (NLR), an important inflammatory biomarker, which was decreased by ASA treatment (Table 1). Wall thickness of the proximal ascending aorta was reduced and elastin/collagen ratio was increased in ASA-treated *ApoE*$^{-/-}$*Fbn1*$^{C1039G+/-}$ mice, resembling values measured in *ApoE*$^{-/-}$ mice (Table 1). Systolic blood pressure, cardiac fibrosis and hypertrophy (Table 1, Figure 1) were reduced after ASA treatment in *ApoE*$^{-/-}$*Fbn1*$^{C1039G+/-}$ mice.

Table 1: Summary of main results

		ApoE$^{-/-}$				*ApoE*$^{-/-}$*Fbn1*$^{C1039G+/-}$			
		Control		**ASA**		**Control**		**ASA**	
		Mean ± SEM	N	Mean ± SEM	N	Mean ± SEM	N	Mean ± SEM	N
Blood	Neutrophils (%)	10.7±2.1	15	9.1±1.5	12	26.4±6.2**	10	13.2±2.1§	7
	Lymphocytes (%)	79.2±3.0	15	80.0±4.3	13	58.2±7.9**	10	86.4±2.4§§	7
	NLR	0.12±0.02	14	0.12±0.02	12	0.45±0.13***	9	0.16±0.03§§	7
Proximal aorta	Wall thickness (µm)	64.9±1.5	9	61.7±1.8	10	70.0±2.4	13	60.3±2.9§§	15
	Elastin wall (%)	24.1±1.2	9	28.0±1.2	10	19.9±1.1*	13	22.8±1.5	15
	Collagen wall (%)	44.4±4.5	9	39.4±3.2	10	63.9±3.9**	13	47.1±3.3§§	15
	Elastin/collagen ratio (%)	58.8±5.9	9	75.1±6.9*	10	32.5±2.6***	13	49.5±2.1§§§	15
Heart	Heart weight/body weight (%)	0.50±0.02	15	0.49±0.02	14	1.16±0.12***	10	0.76±0.05§§§	8
	Total fibrosis (%)	5.7±0.2	15	6.1±0.4	14	9.0±0.7***	16	6.1±0.5§§§	15
BP	Systolic BP (mmHg)	88±4	15	80±4	12	83±6	7	67±4§	8
	Diastolic BP (mmHg)	63±4	15	56±3	12	59±6	7	47±4	8

2-way ANOVA followed by a simple main effects analysis including a Bonferroni correction for multiple comparisons: *p<0.05, **p<0.01, ***p<0.001 vs. *ApoE*$^{-/-}$ control and §p<0.05, §§p<0.01, §§§p<0.001 vs. *ApoE*$^{-/-}$*Fbn1*$^{C1039G+/-}$ control. NLR=neutrophil-lymphocyte ratio, BP=blood pressure.

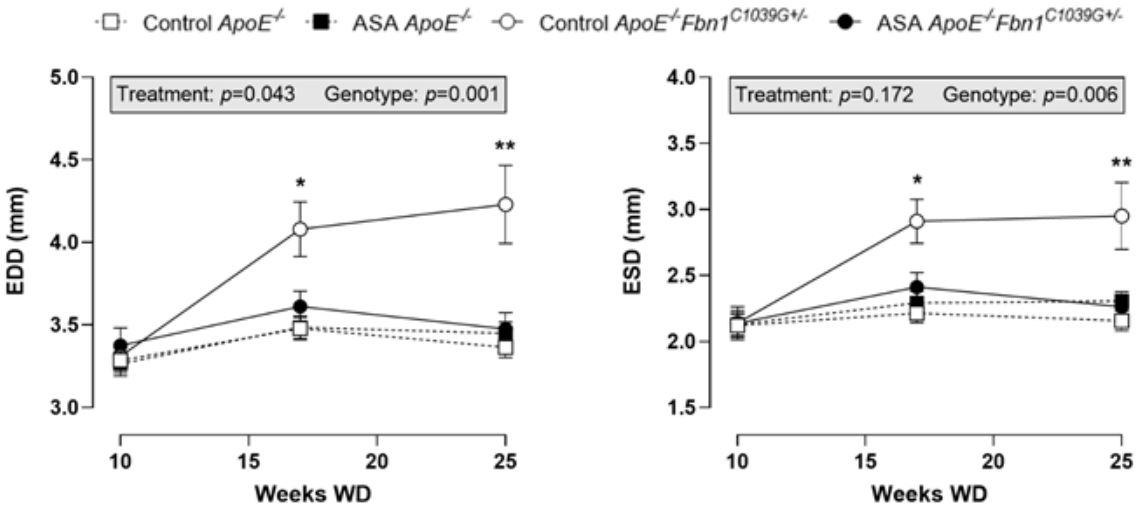

Figure 1: Left ventricular end-diastolic (EDD) and end-systolic diameter (ESD). 3-way mixed ANOVA followed by a simple main effects analysis including a Bonferroni correction for multiple comparisons: *$p<0.05$, **$p<0.01$ vs. control *ApoE*$^{-/-}$*Fbn1*$^{C1039G+/-}$ mice at the respective timepoint.

Conclusion: We showed that ASA is able to decrease the NLR, passive aortic wall stiffness and cardiac remodelling in mice with advanced atherosclerosis to levels observed in mice with smaller and more stable atherosclerotic plaques. These data point towards an additional benefit of ASA in the prevention of CVD beyond its classical use.

P.58

Genetic Background Dictates Aortic Fibrosis in Hypertensive Mice

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Background
Many genetic mutations affect aortic structure and function in mice, but little is known about the influence of background strain. We compared the biomechanical, structural, and gene expression responses of C57BL/6J and 129SvEv aortas to angiotensin II (AngII)-induced hypertension.

Methods
After AngII infusion (14-day, 1000 ng/kg/min) and euthanasia, excised thoracic aortas were characterized functionally using isobaric vasoactive and cyclic passive stiffness tests. Immunohistochemistry quantified medial/adventitial composition and infiltration of pan-inflammatory $CD45^+$ cells. RNA sequencing-based gene ontology, wall stress analyses, and growth and remodeling (G&R) simulations were performed to complement our mechanical findings.

Results
Baseline aortic geometry, composition, and biomechanical properties, as well as AngII-induced blood pressure increases (+34% vs. +32%, systolic), were similar across strains. Yet, AngII-induced aortic remodeling differed dramatically, with gross maladaptive, fibrotic remodeling (exuberant medial/adventitial thickening) in C57BL/6J but not in 129SvEv mice (+89% vs. +12% thickness increase, p=0.022). $CD45^+$ cell density was markedly higher in hypertensive C57BL/6J than 129SvEv aortas (p=0.001), while vasoconstrictive responses to AngII (causing a wall stress decrease $\Delta\sigma$) were greater in 129SvEv than C57BL/6J mice, both before ($\Delta\sigma$=-8 vs. -24%, p=0.023) and after ($\Delta\sigma$=-24 vs. -46%, p<0.001) hypertension. Gene expression, stress analyses, and G&R simulations reinforced the emergent hypothesis that mechanical stress-mediated immune processes promote maladaptive remodeling while smooth muscle contractile processes reduce wall stress and thereby protect against fibrosis (Figure).

Conclusions
Differentially expressed mechano-sensitive genes play key roles in the distinct hypertensive aortic remodeling in C57BL/6J and 129SvEv mice and must be considered when comparing studies in different background strains.

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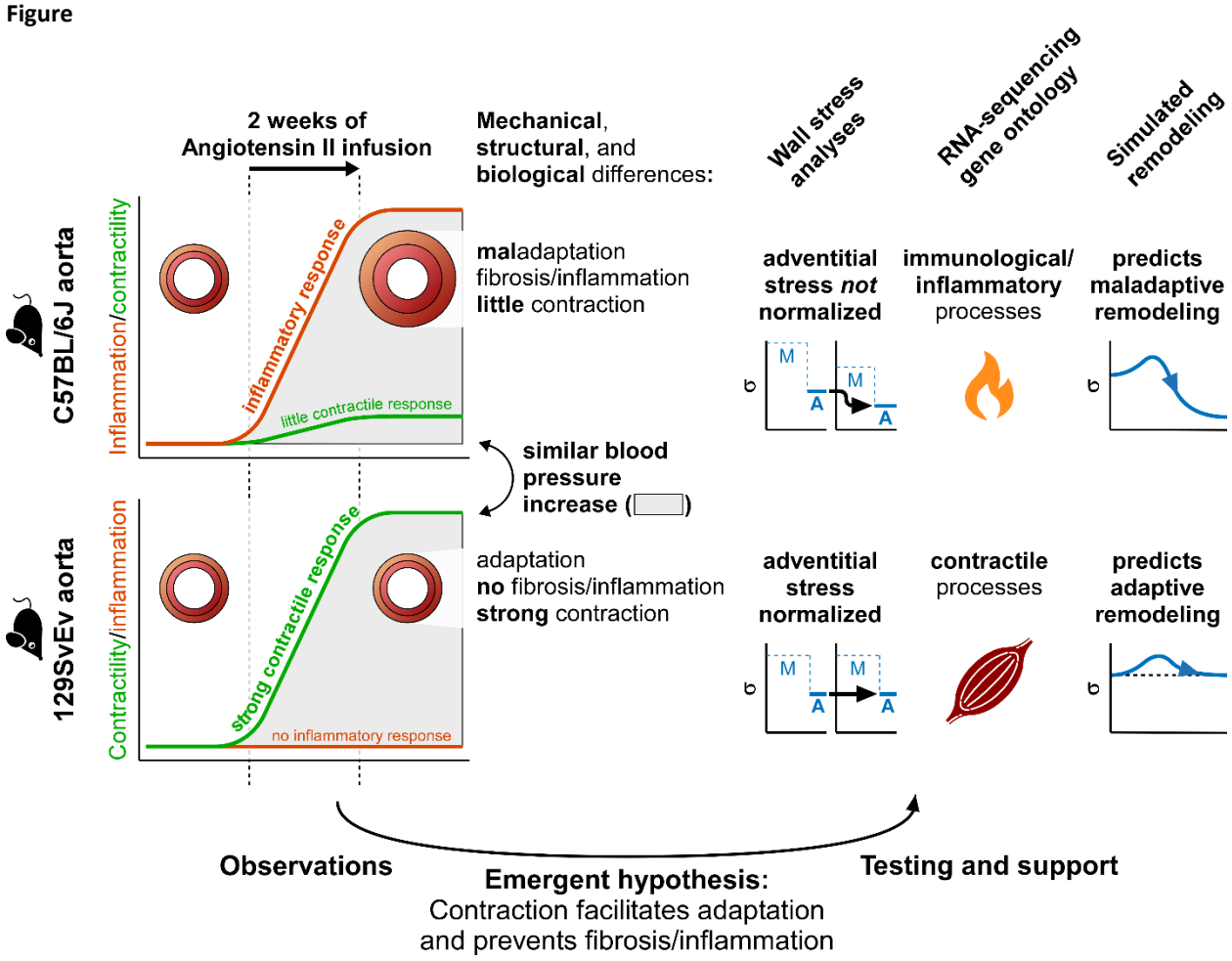
Figure


2 weeks of
Angiotensin II infusion
Mechanical, structural, and biological differences:
Wall stress analyses
RNA-sequencing gene ontology
Simulated remodeling
C57BL/6J aorta
Inflammation/contractility
inflammatory response
little contractile response
maladaptation
fibrosis/inflammation
little contraction
adventitial stress not normalized
immunological/ inflammatory processes
predicts maladaptive remodeling
similar blood pressure increase ( )
129SvEv aorta
Contractility/inflammation
strong contractile response
no inflammatory response
adaptation
no fibrosis/inflammation
strong contraction
adventitial stress normalized
contractile processes
predicts adaptive remodeling
Observations
Testing and support
Emergent hypothesis:
Contraction facilitates adaptation
and prevents fibrosis/inflammation

P.59

The influence of the aortic root in the assessment of cardiovascular function: a new wave separation analysis method

This abstract has been withdrawn by the author.

P.60

Improvement in muscular strength within one year is associated with increased arterial stiffness in young male soccer players

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Purpose
The adaptation process of the cardiovascular system to exercise and muscular strength in young athletes is unclear. Therefore, we investigated the influence of changes in muscular strength and weekly training load within one year on arterial stiffness in young male soccer players.

Methods
30 male soccer players were examined twice (age t0: 13.3±2.0 years, age t1: 14.2±2.0 years). cSBP and aPWV were measured oscillometrically (Mobil-O-Graph®) and z-scores were calculated. The MoMo physical activity questionnaire recorded the weekly training load and the hand dynamometer measured muscular strength. Height and weight were examined and body surface area (BSA) was computed.

Results
The investigated parameters and the converted z-scores of cSBP and aPWV did not change over time, but handgrip strength (t0: 25.2±10.2 kg, t1: 29.5±10.2, $p<0.001$) and training load (t0: 7.1±1.5 hours/week, t1: 8.3±2.4 hours/week, $p=0.005$) increased significantly. 13.3% and 16.7% had cSBP >90th percentile at t0 and t1 respectively. The prevalence of aPWV >90th percentile was 26.7% at both times. Regardless of age and BSA, improvement in handgrip strength was significantly associated with higher values of cSBP ($\beta=1.66$, $p=0.009$, $R^2=0.42$) and aPWV ($\beta=0.09$, $p=0.001$, $R^2=0.58$) at t1. An improvement in weekly training load was not associated with cSBP and aPWV at t1.

Conclusion
cSBP and aPWV are negatively influenced by an improvement in muscular strength in young male soccer players within one year. Therefore, in addition to the recommended pre-participation screening, arterial stiffness in young athletes should be monitored annually to detect possible negative outcomes of exercise on vascular health.

P.61

Impact of kidney transplantation on arterial reservoir-wave analysis

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Purpose/background/objective: According to reservoir-wave approach (RWA) arterial pressure is the sum of a reservoir pressure (RP) accounting for dynamic storage and release of blood from arteries, and an excess pressure (XSP) analogous to flow. RP is the minimal left ventricular work required to generate aortic flow, while XSP corresponds to surplus cardiac workload. We have previously shown that kidney transplantation (KTx) improves aortic stiffness [1], however, by adding renal vessels to existing vascular network, KTx may increase cardiac output. Thus, we aimed to examine whether XSP increases after KTx.

Methods: Before and 3 months after KTx, carotid pressure waves were recorded using arterial tonometry, calibrated using brachial diastolic and mean blood pressure. Using pressure only approach, reservoir-wave analysis was used to derive RP, XSP and their integrals (RPI, XSPI). RWA parameters were compared with Wilcoxon non-parametric test using SPSS 26.0.

Results: 75 patients (69% male, mean age 51±13 years) were assessed. Three months after KTx, both carotid RP (121.2±20.7 vs 103.5±15.7, P<0.001) and RPI (11192.52±2763.11 vs 9531±1978, P<0.001) decreased significantly, but carotid XSP and XSPI remained unchanged. Carotid systolic (131.0±23.2 vs 114.1±15.5, P<0.001) and diastolic (83.4±11.9 vs 72.8±9.93, P<0.001) blood pressures were also reduced.

Conclusion: KTx decreased reservoir pressure, suggesting a decrease in minimal cardiac workload. However, we did not see an increase in excess pressure or its integral, suggesting that addition of a donor renal artery does not significantly alter cardiac outflow and excess workload 3 months after KTx.

P.62

Assessment of isoflavone and ethanolic extract of Inonotus obliquus on experimentally induced diabetes.

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Purpose: Studies support beneficial effects of isoflavones, but antidiabetic effects of these agents remains unconfirmed[1,2]. This pilot study investigates isoflavones and *Inonotus obliquus* (chaga) extract effects on diabetes.

Methods: Diabetes was induced (streptozotocin 65 mg/kg, nicotinamide 110 mg/kg) in 9 male Wistar rats (12 weeks old). 9 additional rats were healthy controls. After 4 weeks animals were treated for 4 weeks with vehicle, isoflavone (200 mg/kg/day), or *Inonotus obliquus* (100 mg/kg/day). Blood pressure and metabolic caging were measured weekly. Glucose tolerance, renal function (serum creatinine, blood urea nitrogen (BUN) level, creatinine clearance rate) and heart, kidney and body weight were assessed at the end-point.

Results: The diabetes group had 1 death (ketoacidosis). Untreated diabetic rats showed glucose intolerance (area under curve (AUC)= 64.87±9.71min×mmol/L), ameliorated with isoflavone (AUC=14.78±1.1min×mmol/L, $p<0.001$) and chaga extract (AUC=30.4 ±13.5 min×mmol/L, $p<0.001$). Body weight was lower but not significantly different in untreated (491.3±35.3g) versus isoflavone (521.0±7.0g, $p >0.05$) and chaga treatment (552.0±91.9 g, $p >0.05$). Kidney mass index was higher in untreated diabetic rats (0.51±0.06) compared to isoflavone (0.36±0.02, $p< 0.05$) but not significantly different in chaga (0.39±0.06, $p >0.05$) treatment. Food and water intake and 24 hr urine output was not significantly different. No difference in serum creatinine, BUN, or creatinine clearance rate were found.

Conclusions: Initial results indicate renal benefits of isoflavone and chaga extract in an animal model of diabetes but without other cardiovascular impact. The study was underpowered to detect all differences and further work, including translating results to humans, is required.

P.63

Sarcopenia and atherosclerotic occlusive disease: how much we know and what we need to know about this association?

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Purpose/Background: Sarcopenia (decrease of muscle mass and function) has been linked with atherosclerosis[1]. The EWGSOP2 updated consensus, uses low muscle strength as the primary indicator of sarcopenia[2]. It is acknowledged that strength is better than mass for predicting adverse outcomes[2]. Handgrip strength (HGS) is a simple assessment to estimate overall muscular strength[3]. and is associated with cardiovascular mortality[4].

Objective: Analyze the relationship between HGS and atherosclerotic disease (carotid artery disease + lower extremity artery disease).

Methods: Prospective observation study was conducted from January to December 2019. The clinical and demographic data was recorded. Isometric HGS was measured with an adjustable handheld dynamometer (Jamar). The higher value of each arm was used to classify the patient as sarcopenic or non-sarcopenic. Definition of sarcopenia: HGS <30kgf in men and <20 kgf in women[5].

Results: 94 patients (aged 44-86 years) were analyzed: 64 sarcopenic and 30 non sarcopenic. Groups differed in the prevalence of diabetes and smoking status (Table I). No differences were found in the carotid parameters analyzed (Table I). There was, a difference in the prevalence of chronic limb-threatening ischemia (CLTI) in sarcopenic versus non-sarcopenic group (23.44% *versus* 6.67% p=0.046). Importantly, binary logistic regression showed that diabetes (p=0.014), and HGS (p=0.027) have a significant effect on CLTI (Table II).

Conclusions: No relationship was found between sarcopenia (measured by HGS) and carotid atherosclerosis, differing from other authors[1,6]. In this study, sarcopenic had a higher incident of diabetes and CLTI. Sarcopenia and diabetes are reciprocally related and may share a similar pathogenetic pathway[7,8,9].

(Figures on next page)

Table I.

	Sarcopenia (n=64)	No sarcopenia (n=30)	p
Age(years)	69.81±8.79	62.6±8.61	p=0.889
Male	47 (73.44%)	27 (90.00%)	p=0.067
Hypertension	51 (79.69%)	21 (70.00%)	p=0.301
Dyslipidemia	47 (73.43%)	18 (60.00%)	p=0.189
Smoking load(UMA)	24.42±33.14	37.76±31.8	p=0.748
Smoker/ Ex-smoker	33 (51.56%)	24 (80.00%)	**p=0.013***
Diabetes	28 (43.75%)	7 (23.33%)	**p=0.049***
Coronary disease	11 (17.19%)	4 (13.33%)	p=0.613
History of stroke	11 (17.19%)	3 (10.00%)	p=0.347
Total cholesterol(mg/dL)	158.16±39.82	159.6±30.72	p=0.22
LEAD	43 (67.19%)	17 (56.67%)	p=0.275
Claudicants	28 (43.75%)	15 (50.00%)	p=0.615
CLTI	15 (23.44%)	2 (6.67%)	**p=0.046***
ABI right	0.83±0.24	0.78±0.29	p=0.287
ABI left	0.81±0.28	0.77±0.23	p=0.671
Right carotid artery stenosis			
50-70	4 (6.25%)	2 (6.67%)	p=0.952
>70%	58 (90.63%)	27 (90.00%)	p=0.702
Left carotid artery stenosis			
50-70%	3 (4.79%)	1 (3.33%)	p=0.787
>70%	4 (6.25%)	2 (6.67%)	p=0.903
Area right carotid plaque(mm^2)	21.22±19.81	20.01±17.04	p=0.622
Average IMT- right(mm)	0.96±0.41	0.88±0.24	p=0.159
Area left carotid plaque(mm^2)	21.46±18.73	21.47±22.06	p=0.948
Average IMT- left(mm)	0.93±0.25	0.88±0.29	p=0.861

Table II

Independent variables	Categories	β	95% CI	p
CLTI	Diabetes	1.488	1.34-14.60	0.014
	Higher HGS	-0.088	0.846-0.990	0.027

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Active vitamin D treatment does not improve arterial stiffness and markers of cardio-renal risk in patients with type 2 diabetes and stage 3 chronic kidney disease: a randomised controlled trial.

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Background and aims

Active vitamin D [1,25(OH)2D3] deficiency is a potential modifiable risk factor for cardiovascular (CVD) and renal disease in patients with type 2 diabetes (T2DM) and stage 3/4 chronic kidney disease (CKD). Exact mechanisms are unclear.Arterial stiffness is an independent predictor of CVD. There is limited data on the effect of active vitamin D treatment on arterial haemodynamics in this patient population.

Materials and methods

We performed a 48 week duration single centre randomised double blind placebo controlled trial on the impact of calcitriol 0.25 mcg od in patients with T2DM and stage 3 CKD . Primary endpoint was change in Ao-PWV (index of arterial stiffness) measured by applanation tonometry (Sphygmocor system). Secondary endpoints included albuminuria (albumin excretion rate-AER) and changes in other indices of central haemodynamics .

Results

127 (male 70%) patients were randomised to calcitriol (n=64) or placebo (n=63). Baseline, mean ±SD, values were: age 64.2 ± 7.7, eGFR 43.2 ±20.2 ml/min, SBP 146.2 ±19.9 mmHg , Ao-PWV 11.6±3.3 m/s, and AER median (IQR) 50.51 (11.5 to 188.6) mcg/min. There was no significant mean (95% CI) change in Ao-PWV as compared to placebo of 0.05 m/s (-0.68 to 0.78) vs 0.23 m/s (-0.46 to 0.93) with a between treatment mean (95% CI) difference for Ao-PWV of 0.19 (-0.81 to 1.19) m/s (p=0.71). No significant effect of calcitriol treatment observed on augmentation index or albuminuria. .

Conclusion

In T2DM patients with stage 3 CKD, 48 week treatment with calcitriol as compared to placebo does not improve Ao-PWV, albuminuria or other indices of central haemodynamics.

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Increased biomarkers of endothelial dysfunction and thrombotic microenvironment in patients with autoimmune rheumatic disorders free from cardiovascular comorbidities

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Purpose/Background/Objectives

Cardiovascular risk is increased in patients with autoimmune rheumatic disorders (1). Endothelial and platelet MVs (EMVs, PMVs) are small vesicles (0.1-1 μm) released from plasma membrane and represent novel markers of endothelial dysfunction and thrombosis. Their levels increase substantially in patients with cardiovascular diseases (2,3). We tested whether EMVs and PMVs are increased in patients with autoimmune rheumatic disorders in the absence of cardiovascular comorbidities.

Methods

Consecutive patients with rheumatoid arthritis or systemic lupus erythematosus were studied, provided they were free from cardiovascular comorbidities (hypertension, diabetes, heart disease, history of cardiovascular or cerebrovascular events). We additionally used (a) a control group consisting of healthy volunteers and (b) a reference group including patients with stable coronary artery disease (CAD). MVs were measured by a standardized flow cytometry protocol (2,3).

Results

We studied 74 participants: 17 patients with autoimmune rheumatic diseases; 34 healthy volunteers, and 23 stable CAD patients. Patients with rheumatic diseases presented increased levels of both EMVs (283.3±195.0/μL vs 168.5±54.8/μL, p=0.029) and PMVs (374.0±275.3/μL vs 225.7±101.1/μL, p=0.046) compared to controls. In addition, they presented similar levels of EMVs compared to CAD patients (283.3±195.0/μL vs 297.0±211.8/μL, p=0.846), whereas PMVs were substantially elevated in the latter (374.0±275.3/μL vs 1034.8±374.0/μL, p=0.029).

Conclusions

Endothelial dysfunction and thrombotic predisposition, shown by increased levels of EMVs and PMVs, respectively, may be evidenced in patients with autoimmune rheumatic diseases, even in the absence of cardiovascular comorbidities and before the establishment of clinically evident cardiovascular complications. In these patients, levels of EMVs appear to be comparable with those of stable CAD patients.

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Radial artery systolic-diastolic pulse transit time after kidney transplantation

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Purpose/background/objective:
We have previously shown that restoration of kidney function through kidney transplantation (KTx) is associated with improved aortic stiffness. In this study, we aim to examine whether this change in aortic stiffness translates into improvement of radial artery systolic-diastolic pulse transit time.

Methods: Before and three months after KTx, we obtained radial pressure waveforms using applanation tonometry, in a group of 61 patients with restored renal function (eGFR > 45 ml/min/1,73 m^2). Radial waveforms were recorded over a 10 seconds period and ensemble-averaged (using in house-MATLAB program) to obtain a single waveform and then modelled using two Gaussian functions, was then determined as the transit time between the first systolic peak T1 and the early diastolic peak T2.

Results: 61 patients (66% male, mean age: 48±14 years, mean eGFR 3 months after Ktx: 66.0±17.1) were assessed. After KTx, there was a significant reduction in central systolic (125,266±21,848 to 108,994±14,407, p<0.001) and diastolic BP (84,718□11,679 to 74,092□9,774 , p<0.001), carotid-femoral PWV (11,444□2,626 to 10,235□1,890, p<0.001) and carotid-radial PWV (9,350□1,485 to 8,831□1,291, p= 0,003). While T1 declined (0.184 [0.173-0.198] to 0.180 [0.168-0.194], p = 0.018), there were no significant changes in T2 (0.322 [0.295-0.360] to 0.318 [0.283-0.355], p = 0.169) and in dT1-2 (0.135 [0.119-0.161] to 0.134 [0.117- 0.167], p=0.457).

Conclusions: Contrary to our expectation, three months after KTx, we did not observe a significant change in radial systolic-diastolic pulse transit time after kidney transplantation, despite an improvement of BP, aortic and brachial stiffness.

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The effects of chemotherapy on arterial inflammation assessed by 18FDG PET-CT in patients with Lymphoma

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Introduction: Anti-cancer treatment can lead to increased cardiovascular morbidity among lymphoma survivors[1]. This may be the result of direct effect of treatment on heart function, or indirect acceleration of atherosclerosis. ^{18}F-fluorodeoxyglucose (FDG) uptake is a sensitive and robust marker for assessment of atherosclerotic inflammation[2,3,4].

Purpose: To investigate the effects of chemotherapy on arterial inflammation using FDG-PET CT in patients with lymphoma.

Methods: Fifty nine (mean age 58±17 years) patients with Hodgkin (n=39) or non-Hodgkin lymphomas (n=20) underwent ^{18}FDG PET-CT imaging at baseline, interim and after completion of chemotherapy as part of their routine protocol. Arterial inflammation was assessed by arterial target to background ratio (TBR) of the aortic wall along the entire aorta. The index vessel TBR (the vessel with the higher value at baseline) was used for assessment of arterial inflammation. Patients with Hodgkin Lymphomas (HL) underwent therapy with Doxorubicin, bleomycin, vinblastine and dacarbazine (ABVD). The interim of their treatment was set at 1 to 3 days prior to initiating the 3rd chemotherapy cycle. Patients with non Hodgkin Lymphomas (NHL) underwent therapy with cyclophosphamide, doxorubicin, vincristine, and prednisone+rituximab (R-CHOP). The interim of their treatment was set at 2 weeks post the 4th chemotherapy cycle. All patients we reassessed 6 weeks after chemotherapy completion.

Results: There were no differences in age and atherosclerotic risk factors (hypertension, diabetes, dyslipidemia and smoking), between the two groups (all P>0.05). Similarly, there were no differences in mean (±SD) index vessel TBR between HL and NHL patients (2.4 ± 0.7 vs 2.7 ± 0.9, respectively, *P*=0.65). In the whole study population the index vessel TBR progressively decreased after the end of therapy (by 0.53±0.11, from baseline to 6 weeks following the end of therapies) (F=10.94, P<0.001, ANOVA). The index vessel TBR decreased in both HL and NHL patients at 6 weeks after therapy compared to baseline level (all P<0.01, ANOVA, figure). The decrease at the interim scan was more pronounced in NHL compared to HL patients, however at 6 weeks after chemotherapy completion the index vessel TBR decreased further in patients with HL, while it increased slightly compared to interim levels in NHL patients (figure).

Conclusion: Arterial inflammation is reduced during and post-chemotherapy in patients with lymphoma. The index vessel TBR changes at the interim phase and 6 weeks after therapy completion indicate a different effect of specific treatment regimes in arterial inflammation between HL and NHL patients.

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WaveGraft – a novel endovascular device concept for restoring the natural arterial cushioning effect

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Background and Objectives
The cushioning effect of large, healthy arteries reduces pulsatile afterload to the heart, reduces pulsatility in the microvasculature of target organs, and promotes coronary/cerebral perfusion.(1) With age, large arteries become stiffer, which increases both pulse wave velocity (PWV) and pulse pressure (PP). This results in isolated systolic hypertension(2), which is characterized by increased systolic blood pressure (SBP) with normal or low diastolic blood pressure (DBP) leading to left ventricular afterload. This study aims to replicate, experimentally, arterial stiffness of hypertensive patients, and proposes a novel thoracic endograft for restoring the arterial cushioning function.

Methods
The experimental setup comprised of two stiff descending aorta silicone replicas (DASR 1&2), pulse duplicator, heated blood mimicking fluid, pressure and flow sensors.
An internal annular viscoelastic cushion, referred to as the 'WaveGraft', was deployed in DASR2. Firstly, pulsatile flow was directed through DASR1, (stiff vessel only), then it was diverted through DASR2 (vessel including WaveGraft).

Results
When compared to the stiff vessel replica (DASR1), the WaveGraft (DASR2) data showed significant reduction in PWV(-60%), SBP(-9%) and PP(-35%), while DBP increased by 10%. An increase in diastolic perfusion by up to 150%, was observed from the recorded data.

Conclusions
This work successfully replicated, key features of aortic hemodynamics in hypertension, linked to the increase in arterial stiffness. The WaveGraft concept showed great potential in altering blood pressure, flowrates and PWV, which may become an important clinical tool in the management of isolated systolic hypertension, heart failure, chronic kidney disease and other chronic conditions.