SUPPLEMENTARY MATERIAL

One-dimensional computational circulatory models: a scoping review

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SEARCH STRATEGIES

Table 1S. Search strategies in different databases

Database	Search string	Articles retrieved
PubMed	("Hemodynamics"[MeSH Terms] OR "hemodynamics"[tw] OR "blood flow"[tw]) AND ("Cardiovascular System"[MeSH Terms] OR "arterial network"[tw] OR "vascular tree"[tw] OR "arterial system"[tw]) AND ("Models, Cardiovascular"[MeSH Terms] OR "data assimilation"[tw] OR "computational model"[tw] OR "mathematical model"[tw] OR "1D model"[tw] OR "one-dimensional model"[tw] OR "reduced-order model"[tw] OR "lumped parameter model"[tw]) AND ("Simulation"[MeSH Terms] OR "simulation"[tw])	3,757
Embase	('hemodynamics'/exp OR 'hemodynamics':ti,ab,kw,de,dn,df,mn,tn OR 'blood flow':ti,ab,kw,de,dn,df,mn,tn) AND ('cardiovascular system'/exp OR 'arterial network':ti,ab,kw,de,dn,df,mn,tn OR 'vascular tree':ti,ab,kw,de,dn,df,mn,tn OR 'arterial system':ti,ab,kw,de,dn,df,mn,tn) AND ('biological model'/exp OR 'data assimilation':ti,ab,kw,de,dn,df,mn,tn OR 'computational model':ti,ab,kw,de,dn,df,mn,tn OR 'mathematical model':ti,ab,kw,de,dn,df,mn,tn OR '1d model':ti,ab,kw,de,dn,df,mn,tn OR 'one-dimensional model':ti,ab,kw,de,dn,df,mn,tn OR 'reduced-order model':ti,ab,kw,de,dn,df,mn,tn OR 'lumped parameter model':ti,ab,kw,de,dn,df,mn,tn) AND ('simulation'/exp OR 'simulation':ti,ab,kw,de,dn,df,mn,tn)	3,061

Table 2S. Standard data collection form

Scoping Review Summary

Citation

Study objective

Model characteristics

Feature	Description
Model type	
Wall properties	
Geometry	
Numerical method	
Simulation fluid	

Model scope

- Number of segments:
- Includes venous system:

Validation method

Models validated by:

Key results from reviewed models

Model / Study	Findings

Conclusions

Table 3S. Data extraction and summary of the individual studies

Alastruey et al. (2011)

Citation

Alastruey J, Khir AW, Matthys KS, Segers P, Sherwin SJ, Verdonck PR, et al. Pulse wave propagation in a model human arterial network: assessment of 1-D visco-elastic simulations against *in vitro* measurements. J Biomech. 2011;44(12):2250-8.

Study objective

To assess the accuracy of nonlinear 1D viscoelastic equations of pressure and flow wave propagation in a realistic model of the human arterial system compared with *in vitro* measurements and to evaluate improvements over previous purely elastic models.

Model characteristics

Feature	Description
Model type	1D nonlinear time-domain viscoelastic formulation
Wall properties	Voigt-type viscoelasticity
Geometry	1:1 replica of 37 largest conduit arteries in the systemic circulation
Validation method	*In vitro* measurements with silicone tubes (no data fitting used)
Numerical scheme	Discontinuous Galerkin with spectral/hp discretization
Simulation fluid	65% water - 35% glycerol (mimics blood; ρ=1050kg/m^3, μ=2.5mPa·s)

Model scope

- Number of arterial segments: 37
- Includes venous system: No

Key results

Metric	Purely elastic model %	Viscoelastic model %	Improvement
Pressure error (EP)	3.0	2.5	$p<0.012$
Flow rate error (EQ)	15.7	10.8	$p<0.002$
Harmonic error (flow)	7.0	3.3	$p<10^{-6}$
Harmonic error (pressure)	0.7	0.5	p=0.107 (NS)

Conclusions

- Inclusion of the wall viscoelasticity significantly improved the accuracy of the 1D simulations
- This is especially relevant for reproducing the high-frequency components of pressure and flow waveforms
- Confirmed the clinical and research utility of 1D models when appropriate physical parameters are known
- 1D viscoelastic modeling achieved a good balance between accuracy and computational cost

Avolio (1980)

Citation

Avolio AP. Multi-branched model of the human arterial system. Med Biol Eng Comput. 1980;18(6):709-18.

Study objective

To develop a physiologically realistic, multibranched 1D computational model of the entire human arterial system—accounting for wave propagation, impedance, and pathological states such as arteriosclerosis and arterial stenosis.

Model characteristics

Feature	Description
Model type	1D transmission-line model (electrical analog principles)
Wall properties	Viscoelastic (with dynamic Young's modulus and phase shift)
Geometry	Multi-branched network of 128 arterial segments
Numerical method	FORTRAN code using transmission line theory with impedance calculations
Simulation fluid	Blood modeled using realistic viscosity and density

Model scope

- Number of arterial segments: 128
- Includes venous system: No

Validation method

Comparison of modeled vascular impedance and waveforms with *in vivo* human data:
- Experimental data from seven patients undergoing cardiac surgery
- Additional validation with data from previous studies on various arteries

Key results

Finding	Notes
Input impedance matched human data across frequency range	Especially accurate >2 Hz
Waveforms (pressure & flow) simulated in various segments	Realistic timing, amplitude, and shape
Pulse wave velocity matched theoretical Moens-Korteweg estimates	approximately 480 cm/s in descending aorta
Model captured effects of arteriosclerosis	Increased stiffness → ↑ systolic pressure, ↓ time delay
Simulated arterial stenosis in femoral artery	Detectable only at >65-70% diameter reduction
Pathological states analyzed: arteriosclerosis, stenosis, wave reflection	Enabled simulations of hypertension patterns and proximal/distal shifts

Conclusions

- The model realistically simulated pressure and flow dynamics in the human arterial tree
- Provided good agreement with experimental data across frequencies and vascular beds
- Particularly valuable for pathophysiological simulations, for example, assessing stenosis impact and pulse wave reflections
- Advantages over analog models—high fidelity to anatomical structures and greater flexibility for parameter changes

Bárdossy et al. (2010)

Citation

Bárdossy G, Halász G. Modeling blood flow in the arterial system. Periodica Polytechnica Mechanical Engineering. 2011;55(1):49-55.

Study objective

To develop a 1D unsteady viscoelastic model of blood flow in the human arterial network using the method of characteristics (MOC) and a novel Stuart viscoelastic material model capable of simulating pathologies, such as arteriosclerosis.

Model characteristics

Feature	Description
Model type	1D unsteady flow model using the method of characteristics (MOC)
Wall properties	Stuart viscoelastic model (Kelvin-Voigt + elastic spring)
Geometry	Network with 45 arterial segments, based on Avolio[11] and Wang et al[8]
Numerical method	Custom-built solver in Transient simulator software (FORTRAN)
Simulation fluid	Newtonian blood, density=1050kg/m^3

Model scope

- Number of arterial segments: 45
- Includes venous system: No

Validation method

Viscoelastic parameters empirically adjusted by comparing them with known pressure waveforms from the literature (Avolio, Wang et al). Additional validation of the Stuart model from previous bench tests with silicone tubes

Key results

Finding	Notes
Increasing systolic pressure along the arterial tree	Diastolic pressure remains relatively stable
Dicrotic notch observed in aortic pressure wave	Indicates high model fidelity
Backflow in iliac arteries during diastole captured	Reproduced known physiological behaviors
Simulated right external iliac stenosis	Diameter reduced from 8.0mm to 2.2mm
Peak pressure drop with stenosis: 122 → 79mmHg	Diastolic: 77 → 69mmHg
Flow velocity drop: 0.52 → 0.14 m/s	Backflow disappears with stenosis
Only 22% flow rate reduction despite 72.8% stenosis	Matches physician observations

Conclusions

- The model accurately reproduced physiological waveforms, including key features such as dicrotic notch and diastolic backflow
- Capable of simulating local stenosis and arteriosclerosis with segment-specific detail
- Stuart viscoelastic model adds significant realism over Hookean assumptions
- Future goals include automated parameter tuning, patient-specific adjustments, and comparison to *in vivo* measurements

Blanco et al. (2013)

Citation

Blanco PJ, Feijóo RA. A dimensionally-heterogeneous closed-loop model for the cardiovascular system and its applications. Med Eng Phys. 2013;35(5):652-67.

Study objective

To develop a closed-loop computational model of the entire human cardiovascular system using heterogeneous mathematical descriptions—including 1D arterial models, 0D lumped compartments, and embedded 3D geometries—to simulate both global and local hemodynamics under physiological and pathological conditions (*e.g.*, aortic regurgitation).

Model characteristics

Feature	Description
Model type	1D-0D-3D heterogeneous closed-loop model
Wall properties	Nonlinear viscoelastic model (includes damping term)
Geometry	128 systemic arterial segments (1D); 61 Windkessel terminals
Numerical method	Finite volume for 1D; Crank-Nicolson and ALE for 0D/3D models
Simulation fluid	Newtonian, ρ=1.04g/cm^3, μ=0.04 dyn·s/cm^2

Model scope

- Number of arterial segments: 128
- Includes venous system: Yes (full 0D compartments)

Validation method

Parameter tuning based on published physiological data. Compared pressure/flow waveforms and valve dynamics with known references

Key results

Simulation condition	Finding
Physiological state	Realistic pressure/flow waveforms, valve dynamics, chamber volumes
Aortic regurgitation (graded severity)	↓ Aortic pressure, ↑ LV end-diastolic volume, ↑ atrial pressure
Carotid and cerebral hemodynamics	Inverted diastolic flow and ↓ WSS in aneurysm under severe regurgitation
WSS/OSI/MRT in aneurysm	↓ WSS by 24%, ↑ OSI by 381%, ↓ MRT by 34% (severe case)
Backward flow during diastole	Captured in iliac arteries and aneurysms
System sensitivity	Large hemodynamic shifts emphasize the need for homeostatic controls

Conclusions

- Combined local and global modeling for a realistic simulation of cardiovascular physiology
- Suitable for simulating complex pathologies and their impact on specific regions (e.g., aneurysms)
- Highlights the role of arterial-venous-cardiac-pulmonary coupling
- Model supports patient-specific diagnostics and therapeutic planning
- Suggests future integration of autonomic control mechanisms (*e.g.*, baroreflex)

Blanco et al. (2014)

Citation

Blanco PJ, Watanabe SM, Dari EA, Passos MA, Feijóo RA. Blood flow distribution in an anatomically detailed arterial network model: criteria and algorithms. Biomech Model Mechanobiol. 2014;13(6):1303-30.

Study objective

To define physiologically accurate criteria for blood flow distribution in a detailed 1D arterial model (ADAN - anatomically detailed arterial network) and develop a numerical calibration algorithm to compute terminal resistances, enabling realistic simulation of regional perfusion across 144 vascular beds (specific organs and distributed territories).

Model characteristics

Feature	Description
Model type	1D arterial network with 144 terminal locations (closed loop)
Wall properties	Nonlinear viscoelastic wall with elastin, collagen, and smooth muscle
Geometry	2,142 arteries, including 1,598 named segments + 544 perforator arteries
Numerical method	Newton-based optimization, least squares FEM with finite differences
Simulation fluid	Newtonian; μ=4 cP (arteries), 1 cP (perforators); ρ=1.04g/cm^3

Model scope

- Number of segments: 2,142 arteries (1,598 named + 544 perforators)
- Includes venous system: No

Validation method

Anatomical calibration using vascular territories based on Taylor and other atlases. The waveforms and pressures were validated against the literature. Jacobian-based resistance was optimized using Newton's method

Key results

Component	Finding
Blood flow to 28 specific organs	64.7% of cardiac output
Blood flow to 116 vascular territories	35.3% of cardiac output
Total arteries modeled	2,142 vessels (unprecedented resolution for 1D model)
Terminal resistances optimized	*Via* Newton method solving 144-equation nonlinear system
Backflow & waveform fidelity	Captured retrograde flows and realistic pressure/flow profiles
Coronary arteries	Time-varying terminal pressures from ventricular pressure profile
Perforator artery resolution	Mapped each vascular territory to source arteries with area-volume flow laws

Conclusions

- Provides a high-resolution arterial network (ADAN) with validated blood flow distribution criteria
- Model accounts for specific and distributed perfusion using advanced anatomical data
- Introduced an efficient calibration algorithm for terminal resistance estimation
- Suitable for regional hemodynamics studies, surgical planning, and spinal cord flow assessments
- Model and database are publicly available at: http://hemolab.lncc.br/adan-web.

Blanco et al. (2015)

Citation

Blanco PJ, Watanabe SM, Passos MA, Lemos PA, Feijóo RA. An anatomically detailed arterial network model for one-dimensional computational hemodynamics. IEEE Trans Biomed Eng. 2015;62(2):736-53.

Study objective

To present the ADAN model—a detailed 1D computational model of the entire arterial system of an average adult man—integrating vascular anatomy, morphometry, wall mechanics, and hemodynamics, validated against both generic physiological data and patient-specific measurements. This model aimed to advance cardiovascular research and clinical planning.

Model characteristics

Feature	Description
Model type	1D model with over 2,000 arterial vessels (ADAN model)
Wall properties	Nonlinear viscoelastic model (including collagen contribution)
Geometry	2,142 vessels (1,598 named + 544 perforators), 3D space with anatomical realism
Numerical method	Finite difference method with Windkessel outflow models
Simulation fluid	Newtonian incompressible fluid

Model scope

- Number of arterial segments: 2,142 (1,598 named + 544 perforators)
- Includes venous system: No
- Blood supply to 28 specific organs and 116 vascular territories

Validation method

Comparison of pressure and flow waveforms with published *in vivo* data
Comparison of cardiovascular indices (CO, ABI, AI, PWV, and PPA) with reference values
Patient-specific case: Invasive catheter-based pressure waveforms used for calibration (32-year-old male). Sensitivity analysis: perturbation of arterial stiffness across regional territories

Key results

Evaluation component	Finding
Waveforms in brain, limbs, abdomen	Match *in vivo* recordings across central and peripheral arteries
Patient-specific calibration	Accurately predicted 6 pressure waveforms along arterial tree
Impedance analysis	Matches Type B curve, Z_0=1205 dyn·s/cm^5 (close to literature)
Cardiovascular indices	HR=60 bpm, CO=6.7 L/min, ABI=1.11 (all within normal range)
Sensitivity analysis	Aortic arch and iliac stiffness greatly impact pressure pulse form
Comparison with simplified model (55 vessels)	ADAN performs better, especially in the presence of stenoses
ICA stenosis simulation	ADAN predicts 1.5% ↓ cerebral flow *versus* 12.96% ↓ in simplified model
Subclavian steal syndrome simulation	Accurately captured retrograde flow in VA and redistribution

Conclusions

- ADAN is the most anatomically realistic 1D model till date, integrating geometry, physiology, and pathology
- Capable of simulating both generic and patient-specific hemodynamics
- Demonstrated superior performance *versus* simplified models in pathological conditions (*e.g.*, stenosis, SSS)
- Useful in research, education, and potentially in clinical diagnostics and planning
- Publicly available: http://hemolab.lncc.br/adan-web

Blanco et al. (2020)

Citation

Blanco PJ, Müller LO, Watanabe SM, Feijóo RA. On the anatomical definition of arterial networks in blood flow simulations: comparison of detailed and simplified models. Biomech Model Mechanobiol. 2020;19(5):1663-78.

Study objective

To compare the predictive capacity of the anatomically detailed ADAN model (2,142 arteries) with its simplified version (ADAN-86) in both healthy and pathological conditions, particularly for evaluating collateral blood flow during common carotid artery (CCA) occlusion and in the Circle of Willis (CoW) anatomical variations.

Model characteristics

Feature	Description
Model type	1D closed-loop model (ADAN *versus* ADAN-86)
Wall properties	Nonlinear viscoelastic (elastin, collagen, smooth muscle contributions)
Geometry	ADAN: 2,142 arteries (4041 1D segments); ADAN-86: 86 vessels (with CoW)
Numerical method	Finite volume (local time stepping), 10 cardiac cycles simulated
Simulation fluid	Newtonian; same inflow conditions for both models

Model scope

- Number of segments: ADAN=2,142 arteries; ADAN-86=86 arteries
- Includes venous system: No

Validation method

Global cardiovascular indices (CO, PWV, ABI)
Impedance and wave intensity analysis
Comparison of pressure/flow waveforms in central and peripheral arteries
Simulation of left CCA occlusion, with and without anterior communicating artery (ACoA)
Benchmarking against values from previous ADAN studies and literature

Key results

Comparison aspect	ADAN *versus* ADAN-86 findings
Healthy scenario	Similar pressure waveforms centrally; large peripheral flow differences
Carotid occlusion (w/ACoA)	ADAN preserves cerebral flow better; lower pressure drop across occlusion
Carotid occlusion (wo/ACoA)	ADAN predicts extracranial → intracranial (ECA-to-ICA) steal; more realistic hemodynamics
Wave intensity analysis	ADAN shows richer forward/backward wave interaction near occlusion
Flow redistribution	ADAN features extracranial collaterals (thyroid, facial arteries)
Pressure drop across occlusion	ADAN: 4.0×10^4 dyn/cm^2; ADAN-86: 10.6×10^4 dyn/cm^2
CBF reduction in occlusion (wo/ACoA)	ADAN: -11.7%; ADAN-86: -25.7%
Steal phenomena	ADAN shows ICA-to-ECA (with ACoA) and ECA-to-ICA (without ACoA)

Conclusions

- Detailed anatomical modeling (ADAN) improved prediction in pathological scenarios, for example, carotid occlusion
- Simplified models performed adequately in healthy or global hemodynamic studies, but failed to capture collateral pathways
- The degree of anatomical detail critically determines the accuracy of simulation in disease and variation
- ADAN enabled investigation of complex hemodynamic effects such as posterior steal, wave reflections, and contralateral compensation

Liang et al. (2009)

Citation

Liang F, Takagi S, Himeno R, Liu H. Multi-scale modeling of the human cardiovascular system with applications to aortic valvular and arterial stenoses. Med Biol Eng Comput. 2009;47(7):743-55.

Study objective

To develop a multi-scale closed-loop model of the cardiovascular system by integrating a 1D arterial tree with a 0-D lumped parameter model for the heart, pulmonary, and peripheral circulations. This model was used to study the global hemodynamic effects of the aortic valve (AV) and arterial stenoses in various regions of circulation.

Model characteristics

Feature	Description
Model type	Multi-scale model: 1D arterial tree + 0-D heart, veins, pulmonary circulation
Wall properties	Elastic with nonlinear pressure-area relation (viscoelasticity for heart)
Geometry	1D: 55 large arteries; 0-D: complete heart and venous blocks
Numerical method	1D: Lax-Wendroff; 0-D: Runge-Kutta; Interface: ghost-point + Newton-Raphson
Simulation fluid	Newtonian; ρ=1.06g/cm^3, μ=4.43s/cm^2

Model scope

- Number of segments: 55 large arteries
- Includes venous system: Yes (as 0-D compartments)

Validation method

Comparison of simulated pressure and flow waveforms at multiple anatomical locations using physiological data from literature and clinical standards. Assessment of heart-vascular interactions using an elastance-based cardiac model

Key results

Scenario	Findings
Normal circulation	Realistic pressure/flow waveforms with proper systolic amplification
AV stenosis (85%)	Prolonged aortic pressure rise, ↑ LV pressure, ↓ SV by approximately 10.5%, ABI unchanged
Thoracic/abdominal aortic stenosis	Proximal pressure overshoot, ↓ distal pressure, moderate LV impact
Renal/femoral stenosis	Minimal LV impact, ↓ ankle pressures, reduced ABI<0.92
Ankle-Brachial Index (ABI)	AV and renal stenoses preserve ABI; others reduce it
Wave reflections	Significant upstream reflections with aortic stenosis; distal flattening

Conclusions

- Stenosis effects are highly location-dependent; aortic lesions impact the heart whereas distal ones affect ABI
- Closed-loop integration enables the study of heart-artery coupling and peripheral changes in the same framework
- The model reproduces clinical findings and offers insight into noninvasive pulse-based diagnostics
- Useful tool for diagnostic research, studying combined cardiac-vascular pathology, and wave reflection analysis

Müller et al. (2014)

Citation

Müller LO, Toro EF. A global multiscale mathematical model for the human circulation with emphasis on the venous system. Int J Numer Methods Biomed Eng. 2014;30(7):681-725.

Study objective

To present a global closed-loop multiscale model of the human circulation with a detailed 1D description of both the arterial and venous systems, complemented by 0-D models of the heart, microcirculation, and pulmonary compartments. A special emphasis is placed on modeling venous hemodynamics, especially for the head and neck veins, motivated by links to neurodegenerative diseases.

Model characteristics

Feature	Description
Model type	Multiscale closed-loop model (1D + 0D)
Wall properties	Variable mechanical properties, nonlinear viscoelastic tube laws
Geometry	85 major arteries and 92 veins modeled as 1D; full 0D model for capillaries, heart, and pulmonary system
Numerical method	High-order ADER method; DOT Riemann solver for 1D
Simulation fluid	Newtonian incompressible fluid, ρ=1.06g/cm^3

Model scope

- Number of segments: 85 arteries, 92 veins
- Includes venous system: Yes (1D venous network, including cerebral veins)

Validation method

Comparison with MRI-derived flow waveforms in the head and neck veins (patient-specific)
Literature-based data for arterial system waveforms and pressure-flow relationships.
In vitro and physiological data for wave speed, pressure-area relations, and venous collapse dynamics

Key results

Component	Findings
Arterial waveforms	Realistic wave propagation; compliant with literature
Venous modeling (head/neck)	Captures physiological waveforms; collapse and backflow during upright posture
Patient-specific simulation	Head/neck venous network calibrated from MRI geometry and flow data
Numerical scheme	ADER scheme achieved 2nd-5th order accuracy; robust to venous collapse
Wave speed and stiffness	Veins had wave speeds of 1-3 m/s; stiffness derived from *in vivo* data
Global hemodynamics	Captures redistribution of flow owing to postural changes
Riemann problem test	Demonstrated accurate capture of wave reflections and elastic jumps

Conclusions

- This is the first global closed-loop model to feature a detailed 1D representation of the venous system
- Capable of modeling collapsible veins, transcritical flow, and gravitational effects
- Patient-specific applications demonstrated using MRI-derived venous geometry and flow
- Useful for studying neurovascular conditions, e.g., chronic cerebrospinal venous insufficiency
- The model forms a robust basis for future venous hemodynamic studies and integrated simulations

Müller et al. (2023)

Citation

Müller LO, Watanabe SM, Toro EF, Feijóo RA, Blanco PJ. An anatomically detailed arterial-venous network model. Cerebral and coronary circulation. Front Physiol. 2023;14:1162391.

Study objective

To develop the ADAVN model (anatomically detailed arterial-venous network)—a novel 1D closed-loop cardiovascular model integrating an ultradetailed arterial system (ADAN) with a newly constructed venous network emphasizing the cerebral and coronary territories, incorporating cerebrospinal fluid (CSF) and cardiac mechanics interactions—to simulate normal and pathological hemodynamics.

Model characteristics

Feature	Description
Model type	Multiscale closed-loop 1D model (arterial + venous + lumped heart and lungs)
Wall properties	Nonlinear viscoelastic tube laws (different for arteries and veins)
Geometry	2,185 arterial vessels + 189 veins (including 79 cerebral and 14 coronary)
Numerical method	Hyperbolized system + ADER finite volume method + local time stepping
Simulation fluid	Newtonian; ρ=1.04g/cm^3; μ=0.04 P (0.01 P in perforators)

Model scope

- Number of segments: 2,185 arteries, 189 veins
- Includes venous system: Yes (79 cerebral veins, 14 coronary veins, dural sinuses, venous valves, Starling resistors)

Validation method

Comparison with published *in vivo* measurements of hemodynamic variables
Patient-specific geometries and physiological data (*e.g.*, MRI venous flow)
Local sensitivity analysis to assess the venous system impact on cardiac output, ICP, and other parameters
Physiological accuracy tested across cardiac and vascular indices

Key results

Component	Findings
Hemodynamic waveforms	Realistic in arteries and veins, reproducing physiological patterns
Cerebral venous modeling	Includes collapse dynamics and ICP regulation through Starling resistors
Coronary circulation	Incorporates cardiac muscle compression and myocardial perfusion layers
Arterio-venous coupling	Flexible multi-capillary connections between arterioles and venules
Cardiac indices	HR=75 bpm, CO=6.1 L/min, MAP=97.4mmHg (within physiological range)
ICP regulation	Implemented through dynamic CSF model (Ursino model)
Venous sensitivity	Venous compliance significantly affects stroke volume and pulse pressure
Computation	Whole-loop simulation approximately 15 min per cycle with high spatial resolution

Conclusions

- The ADAVN model is the most anatomically and functionally complete 1D arterial-venous model till date
- Demonstrated the potential for simulating normal and pathological scenarios, with special attention to the brain and heart
- Incorporated biophysical mechanisms such as collapsible veins, venous valves, ICP, and coronary perfusion
- Serves as a platform for future investigations in neurovascular diseases, heart-brain interactions, and clinical applications

Mynard et al. (2008)

Citation

Mynard JP, Nithiarasu PA. 1D arterial blood flow model incorporating ventricular pressure, aortic valve and regional coronary flow using the locally conservative Galerkin (LCG) method. Commun Numer Methods Eng. 2008;24(5):367-417.

Study objective

To develop a comprehensive 1D model of systemic and coronary circulation that integrates ventricular pressure, a dynamically modeled aortic valve, and regional coronary flow using the Locally Conservative Galerkin (LCG) method and to test its behavior under normal, exercise, and pathological conditions.

Model characteristics

Feature	Description
Model type	1D systemic and coronary arterial model with ventriculo-vascular coupling
Wall properties	Nonlinear elastic law with variable stiffness and taper
Geometry	Systemic arterial tree with approximately 40 segments; LCA and RCA branch into subendocardial and subepicardial vessels
Numerical method	Locally Conservative Galerkin (LCG) finite element method
Simulation fluid	Newtonian; constant density and viscosity assumptions

Model scope

- Number of segments: approximately 40 arterial segments (systemic + coronary)
- Includes venous system: No

Validation method

Verification of the waveforms against published *in vivo* pressure and flow patterns
Simulation of rest and exercise conditions
Comparison of the 1D and 3D velocity profiles in patient-specific carotid bifurcation geometries

Key results

Component	Findings
Systemic waveforms	Realistic pressure and flow waveforms at multiple sites
Coronary dynamics	Flow suppression in subendocardium during systole; diastolic dominance
Aortic valve	Opens and closes based on pressure/velocity thresholds; includes RVOT/RVCT
Afterload sensitivity	Ventricular pressure adjusted by reflected waves and downstream impedance
Terminal elements	Tapering vessels reproduced realistic input impedance better than Windkessel
LCG method	Accurate, locally conservative, and efficient with natural branching
Pathological simulations	Disease features (*e.g.* stenosis) induced changes matching clinical patterns
1D *versus* 3D	Good agreement in velocity profiles in carotid bifurcation comparisons

Conclusions

- The model provides a more physiologically accurate framework by integrating realistic aortic valve mechanics, ventriculo-vascular interaction, and regional coronary perfusion
- LCG method supports complex branching and conserves local flow
- Offers potential for clinical simulation of disease, exercise hemodynamics, and coronary flow modulation

Mynard et al. (2015)

Citation

Mynard JP, Smolich JJ. One-dimensional haemodynamic modeling and wave dynamics in the entire adult circulation. Ann Biomed Eng. 2015;43(6):1443-60.

Study objective

To develop a comprehensive 1D closed-loop model of the entire adult cardiovascular system, including detailed representations of systemic, pulmonary, coronary, and portal circulations, coupled with a lumped-parameter heart model incorporating chamber interactions. The model aimed to explore wave propagation, wave intensity, and ventricular-vascular interactions under normal and pathological conditions.

Model characteristics

Feature	Description
Model type	Closed-loop 1D model of full circulation + 0-D heart and microvasculature
Wall properties	Nonlinear viscoelastic (power law elastic + Voigt viscous)
Geometry	396 1D segments (arteries + veins) in systemic, pulmonary, coronary, portal
Numerical method	Finite element method with operator splitting
Simulation fluid	Newtonian; ρ=1.06g/cm^3; μ=0.035

Model scope

- Number of segments: 396 1D vessels, 5359 nodes, 188 junctions
- Includes venous system: Yes (systemic, pulmonary, portal, coronary)

Validation method

Comparison with *in vivo* human flow and pressure waveforms
Reproduction of waveforms across the aorta, pulmonary arteries, portal system, vena cava, and coronary arteries/veins
The hemodynamic variables were validated against published data (CO, SVR, and chamber volumes)

Key results

Component	Findings
Global waveform reproduction	Close match with *in vivo* data for pressure and flow waveforms
Wave intensity profiles	Simulated wave intensity closely resembled available *in vivo* results
Aortic/coronary wave behavior	Captured key wave reflection and compression/expansion waves
Venous wave analysis	Novel insight into wave reflections in vena cava and pulmonary veins
Cardiac-vascular coupling	Changes in ventricular function influenced waveforms in remote vessels
Regional differences	Wave dynamics varied across brain, limbs, myocardium, lungs, liver
Mechanical interactions	Included pericardial pressure, AV plane motion, septal interaction
Sensitivity analysis	Showed effect of changes in chamber elastance, preload, interaction

Conclusions

- Provides the first anatomically detailed 1D model of full circulation, including systemic, pulmonary, portal, and coronary systems
- Capable of reproducing global wave dynamics, ventricular-vascular interactions, and regional hemodynamics
- Offers novel insights into wave reflection, forward/backward wave behavior, and mechanical effects of chamber interactions
- Useful for investigating cardiovascular disease, wave-based diagnostics, and physiology simulations

Olufsen (1999)

Citation

Olufsen MS. Structured tree outflow condition for blood flow in larger systemic arteries. Am J Physiol. 1999;276(1):H257-68.

Study objective

To improve the physiological accuracy of the 1D models of large arteries by introducing a structured tree model at the distal ends of the arterial system, allowing wave propagation effects to persist beyond the truncated computational domain. This approach provides a frequency-dependent outflow boundary condition that better represents the downstream vasculature than traditional lumped models.

Model characteristics

Feature	Description
Model type	1D nonlinear model of large arteries + structured tree as outflow condition
Wall properties	Linearly elastic wall with exponential stiffness-radius relation
Geometry	21 large arteries + structured binary tree per terminal (~17 generations)
Numerical method	Lax-Wendroff scheme; semi-analytical Fourier-based impedance for terminal trees
Simulation fluid	Newtonian, axisymmetric, laminar flow

Model scope

- Number of segments: 21 large arteries + structured tree outflow (~17 generations each)
- Includes venous system: No

Validation method

Comparison of simulated flow and pressure against measured data from human arteries, Windkessel, and pure resistance boundary models. Evaluated impedance spectra, wave reflections, and pressure-flow phase lag

Key results

Component	Findings
Impedance profiles	Structured tree preserves high-frequency impedance oscillations; better match with human data
Pressure-flow relation	Maintains physiologic phase lag; avoids artificial reflections
Comparison with Windkessel	Windkessel fails to model wave reflections and phase shifts
Reflected waves	Dicrotic notch and wave shape changes captured better with structured tree
Spatial pressure distribution	Progressive amplification and steeper slopes distally, as *in vivo*
Outflow impedance	Frequency-dependent convolution relation derived analytically

Conclusions

- The structured tree outflow boundary condition provides a physiologically consistent and computationally feasible alternative to lumped models (e.g., Windkessel)
- It accounts for wave propagation, impedance spectra, and arterial-tissue coupling at the terminal level
- Suitable for studies of wave reflection, arterial stiffening, and peripheral resistance
- Offers flexibility to simulate vasodilation/vasoconstriction by adjusting tree geometry or mechanical parameters
- Represents a paradigm shift in boundary condition modeling for 1D arterial networks

Reymond et al. (2009)

Citation

Reymond P, Merenda F, Perren F, Rüfenacht D, Stergiopulos N. Validation of a one-dimensional model of the systemic arterial tree. Am J Physiol Heart Circ Physiol. 2009;297(1):H208-22.

Study objective

To build and validate a comprehensive 1D model of the human systemic arterial tree, including heart-vascular coupling, cerebral and coronary circulation, nonlinear viscoelastic wall properties, and realistic boundary conditions. The model was validated *in vivo* using flow and pressure data obtained from healthy young volunteers.

Model characteristics

Feature	Description
Model type	1D nonlinear arterial model + 0-D heart + Windkessel terminals
Wall properties	Nonlinear viscoelastic (Holenstein/Bergel)
Geometry	Detailed full-body arterial tree including coronaries and cerebral circulation
Numerical method	Implicit finite difference + Newton-Raphson
Simulation fluid	Newtonian; ρ=1050kg/m^3; μ=0.004 Pa·s

Model Scope

- Number of segments: 103 arterial segments
- Includes venous system: No

Validation method

Comparison with *in vivo* pressure and flow waveforms in
- Ascending, thoracic, and abdominal aorta
- Iliac, femoral, carotid, radial, and temporal arteries
- Middle cerebral, vertebral, and internal carotid artery

Flow *via* PC-MRI and ultrasound Doppler; pressure *via* applanation tonometry

Key results

Component	Findings
Waveform match	Good agreement in pressure and flow waveform shape and amplitude
Cerebral circulation	Detailed model avoids backflow artifacts, improves physiological pulsatility
Validation metrics	Mean flow error approximately 12%; pressure error <10% in most locations
Viscoelasticity	Significant in distal arteries; affects flow and pressure wave shape
Convective acceleration / WSS	Witzig-Womersley method improves prediction *versus* Poiseuille approximation
Boundary conditions	3-element Windkessel with impedance matching at terminals
Coronary modeling	Simplified, coupled to time-varying elastance of LV

Conclusions

- This model is among the most anatomically complete 1D arterial models till date
- Includes heart-vascular coupling, nonlinear viscoelasticity, and cerebral/coronary branches
- Validated against real-world measurements, showing high fidelity in capturing physiological wave dynamics
- Well-suited for simulating wave propagation, pathologies, and clinical interventions

Safaei et al. (2016)

Citation

Safaei S, Bradley CP, Suresh V, Mithraratne K, Muller A, Ho H, et al. Roadmap for cardiovascular circulation model. J Physiol. 2016;594(23):6909-28.

Study objective

To propose a comprehensive, open-source computational framework for simulating full-body cardiovascular circulation—integrating 1D, 0D, and 3D models—and enabling multiscale coupling with organ physiology and biomechanics. This paper outlines the mathematical formulation, software architecture, and validation strategies for large-scale simulations.

Model characteristics

Feature	Description
Model type	Multiscale: 1D arterial model + 0D Windkessel + support for 3D coupling
Wall properties	Elastic, viscoelastic (Voigt, Maxwell models), with generalized Young's modulus
Geometry	ADAN-86: 86 arteries including cerebral, coronary, visceral, limb, and abdominal branches
Numerical method	Finite element (Galerkin), Crank-Nicolson, CellML-FieldML integration
Simulation fluid	Newtonian; ρ=1050kg/m^3; ν=4 cP (typical)

Model scope

- Number of segments: 86 arterial vessels (ADAN-86 subset), 230 elements, 457 nodes
- Includes venous system: Partial support for venous and arteriolar models
- Coupling to capillaries and tissue beds *via* RCR and CellML-based 0D models

Validation method

The input flow rate and terminal impedance were modeled using previously published data
Terminal impedance was modeled using the RCR Windkessel
Validation using pressure/flow waveforms across segments and comparison with published data

Key results

Component	Findings
Pressure/flow waveforms	Physiologic waveforms reproduced across full arterial tree
Viscoelastic modeling	Maxwell + Voigt + generalized modulus accurately capture wall behavior
0D-1D coupling	Achieved *via* Riemann invariants; convergence tolerance $\varepsilon<10^{-6}$
Field standards	Implements CellML (ODEs) and FieldML (spatial PDEs) with OpenCMISS
Vascular territories	Blood flow distribution informed by anatomical perfusion domains
3D-1D interface	Coupling *via* stress and flow continuity (mass flux, traction)
Transmission line modeling	Applied in parallel for fast simulations using complex impedance
Computation time	12 h (1 core); 1.5 h (8 cores); goal=near real-time with future methods

Conclusions

- Presents a blueprint for full-body cardiovascular model incorporating multiscale physics
- Emphasizes interoperability *via* open standards (CellML, FieldML) and open software (OpenCMISS)
- Enables simulation of organ-specific perfusion, biomechanical effects, and patient-specific models
- Sets the stage for future real-time or interactive simulations for clinical or research applications

Schaaf et al. (1972)

Citation

Schaaf BW, Abbrecht PH. Digital computer simulation of human systemic arterial pulse wave transmission: a nonlinear model. J Biomech. 1972;5(4):345-64.

Study objective

To develop a comprehensive nonlinear 1D model of arterial pulse wave transmission incorporating finite radial wall displacements with improved realism over linear and lumped-parameter models. The model uses a branching arterial tree and captures wave reflections and propagation across systemic circulation.

Model characteristics

Feature	Description
Model type	1D nonlinear arterial model; solved numerically *via* method of characteristics
Wall properties	Linear elastic (finite radial strain); no viscoelastic terms included
Geometry	Branching network of approximately 28 major arteries (head, arms, trunk, legs) and 47 arterial segments
Numerical method	Method of characteristics with finite differences
Simulation fluid	Newtonian; incompressible; laminar, axisymmetric

Model scope

- Number of segments: 47 arterial segments
- Includes venous system: No
- Fourteen output locations mapped to clinically relevant sites

Validation method

The simulated pressure and flow waveforms at 14 locations were compared with clinical data
Fourier analysis was used for the input impedance assessment at the aortic root and femoral artery

Key results

Component	Findings
Waveform fidelity	Good reproduction of clinical pulse wave shapes, inflection points, and amplifications
Pulse pressure amplification	approximately 70% from aortic root to iliac; within range of clinical data
Impedance comparison	Better agreement with clinical data than linear models
Convective acceleration	Shown to be negligible for large vessels
Wall friction effects	Unsteady friction shown to have minor influence on overall dynamics
Linear *versus* nonlinear comparison	Linear models produce more oscillatory artifacts; nonlinear model more realistic
Terminal models	Purely resistive loads used at distal ends
Computational results	Rapid convergence to physiologic steady oscillations within three cycles

Conclusions

- The nonlinear 1D model captures key pulse dynamics more accurately than linear models
- Wall distensibility (nonlinear compliance) is crucial in modeling wave propagation, especially in central arteries
- Convective acceleration and fluid friction contribute little to global wave dynamics
- Recommended for detailed analysis of arterial wave transmission, especially in elastic vessels such as the aorta
- Demonstrated that nonlinear modeling provides improved physiological realism

Stergiopulos et al. (1992)

Citation

Stergiopulos N, Young DF, Rowe TR. Computer simulation of arterial flow with applications to arterial and aortic stenoses. J Biomech. 1992;25(12):1477-88.

Study objective

To develop a fully nonlinear 1D computer model of the systemic arterial circulation and use it to investigate the hemodynamic effects of arterial and aortic stenoses. This model aimed to simulate the pressure and flow waveforms under both normal and pathological conditions and compare the predictions with *in vivo* data.

Model characteristics

Feature	Description
Model type	1D nonlinear model with stenosis module and Windkessel outflows
Wall properties	Nonlinear compliance using quadratic pressure-area relationship
Geometry	55 arterial segments (head, arms, trunk, legs)
Numerical method	Explicit finite difference; upwind differencing; stability $\Delta t<\Delta x/c$
Simulation fluid	Newtonian, laminar, axisymmetric

Model scope

- Number of segments: 55 arterial segments
- Includes venous system: No

Validation method

Comparison with experimental data from the literature (pressure/flow waveforms)
Pulsatility index (PI) compared with clinical ultrasound data
Segmental systolic pressure index validated against vascular occlusive disease patient data

Key results

Component	Findings
Waveform validation	Good match with clinical waveforms in shape, timing, and amplification
Pulse pressure amplification	Captures increase from aorta to femoral (approximately 70%), consistent with data
Effect of stenosis on flow	Critical stenosis severity approximately 85% (normal), approximately 60% (vasodilated state)
Pulsatility index (PI)	Drops significantly beyond 60% stenosis
Systolic pressure index	Significant drop in systolic pressure with stenosis >60%; validated in comparison with other studies
Aortic stenosis simulation	Reproduces delayed rise, reduced amplification, and plateau in periphery
Stenosis model	Nonlinear pressure drop across lesion modeled using empirical Q-based formula
Boundary conditions	Three-element Windkessel with realistic impedance matching

Conclusions

- Nonlinear 1D models accurately simulate systemic hemodynamics and pathologic states (arterial and aortic stenosis)
- Reproduced clinically observed features such as pressure dampening, pulse shape deformation, and wave reflection changes
- Useful for studying diagnostic markers such as PI and systolic pressure ratios
- Reinforces the role of computational models in noninvasive diagnostics and physiological interpretation

Wang et al. (2004)

Citation

Wang JJ, Parker KH. Wave propagation in a model of the arterial circulation. J Biomech. 2004;37(4):457-70.

Study objective

To explore the role of wave reflections and re-reflections in the systemic arterial system using a linearized 1D model of 55 large arteries. This study isolated the effect of arterial geometry on wave dynamics while simplifying the cardiac input and eliminating nonlinearities to better understand pressure and velocity waveforms in health and disease.

Model characteristics

Feature	Description
Model type	1D linearized model of systemic arteries (55 segments)
Wall properties	Linear elastic (Moens-Korteweg-based wave speed)
Geometry	Bifurcating tree based previously published data with modified radii
Numerical method	Method of characteristics with 'tree of waves' algorithm
Simulation fluid	Newtonian; incompressible; U << c assumed (quasi-linear)

Model scope

- Number of segments: 55 arterial segments
- Includes venous system: No

Validation method

Waveforms compared with *in vivo* measurements and classic studies
Simulated conditions included the heart as absorber *versus* reflector, aortic valve closure, aortic occlusion at four sites, changes in terminal resistance, and coronary flow effects on the reflection coefficients

Key results

Component	Findings
Wave reflection dynamics	Complex re-reflections at bifurcations are main contributors to waveform
Pulse pressure amplification	Observed distal amplification consistent with clinical data
Heart reflection effects	Increased pressure in diastole; mimicked aortic regurgitation
Aortic occlusion modeling	Wave reflections matched experimental canine data previously published data
Terminal resistance effects	Lower Rp reduced diastolic pressure and reversed flow magnitude
Coronary circulation	Lowered post-valve reflection coefficient; altered waveform slope
Transfer function insights	Segment-wise transfer functions contain full info about wave propagation
Backward wave "trapping"	Reflections attenuated by poorly matched bifurcations on return path

Conclusions

- Wave reflections in a realistic 1D arterial model explained most of the observed pressure and velocity waveform features
- The "tree of waves" algorithm effectively tracks forward and backward waves with high precision
- Demonstrated that distal waveform changes and pathological alterations (*e.g.*, occlusion, regurgitation) can be explained by wave behavior alone
- Highlights the value of transfer functions and their potential clinical applications, although direct human measurement remains challenging

Westerhof et al. (2020)

Citation

Westerhof BE, van Gemert MJ, van den Wijngaard JP. Pressure and flow relations in the systemic arterial tree throughout development from newborn to adult. Front Pediatr. 2020;8:251.

Study objective

To develop a distributed hemodynamic model of the human arterial tree that accounts for developmental changes from newborns to adults and to use this model to examine pressure-flow relationships and the applicability of Windkessel models across different ages

Model characteristics

Feature	Description
Model type	1D distributed model with 3-element Windkessel terminals
Wall properties	Elastic and viscoelastic; wall stiffness and thickness vary with age
Geometry	121 arterial segments adapted by growth curves of body parts
Numerical method	Frequency-domain impedance analysis using circuit theory
Simulation fluid	Newtonian; parameters age-adjusted (flow, heart rate, vessel radius)

Model scope

- Number of segments: 121 arterial segments
- Includes venous system: No

Validation method

The simulated pressure and flow waveforms were compared with *in vivo* data from population studies
The input impedance and pulse-wave velocity were matched the developmental trends
Brachial pressures were validated against clinical centile datasets

Key results

Component	Findings
Pressure amplification	Peripheral pressure in children <5 years ≈ central pressure; amplification increases with age
Pulse wave velocity	Gradual increase with age; consistent with clinical observations
Transfer functions	Adult-like shape only from approximately 10 years onward; children <10 years have flatter transfer curves
Impedance modeling	3- and 4-element Windkessel approximations valid across age range
Wall shear stress	Preserved constant by scaling radius to tissue perfusion (approximately r ∝ Q^1/3)
Model calibration	Required adjustments to wall stiffness and peripheral resistance to avoid hypertensive output
Clinical relevance	Caution advised when using adult transfer functions for children <10 years

Conclusions

- This model provides a comprehensive simulation framework for evaluating arterial hemodynamics from newborns to adults
- Peripheral-to-central pressure differences are negligible in young children but become significant after approximately 10 years
- Windkessel models remain applicable if properly scaled by age, height, and body composition
- This model may aid in interpreting pediatric pressure data, estimating cardiac output, and analyzing the effects of vascular disease or anomalies during development