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Chemeca 2026 and Hazards Australasia
28 – 30 September, Melbourne, Australia

Fluid Mechanics of Cardiovascular Disease: Integrating Bernoulli, Poiseuille, and Blood Rheology

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ABSTRACT

Cardiovascular disease is typically framed as a biochemical disorder, yet its initiation and progression are also fundamentally governed by fluid mechanics. This work integrates Bernoulli's equation, Poiseuille's law, and non-Newtonian blood rheology to describe how vascular geometry and flow patterns drive endothelial dysfunction, plaque formation, and aneurysm evolution.

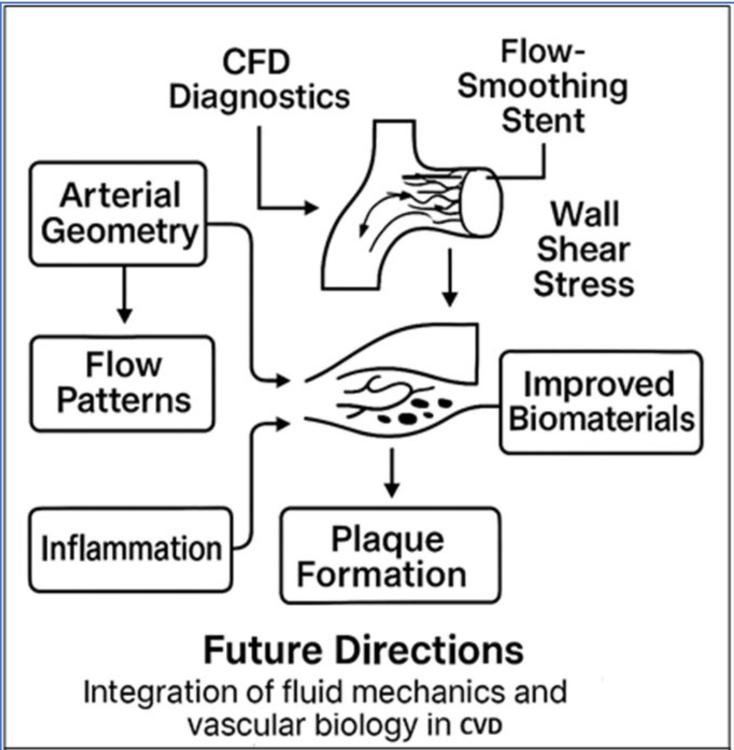
Arterial stenosis produces a characteristic redistribution of pressure and velocity. As cross-sectional area decreases, conservation of energy along a streamline requires

$$P + \frac{1}{2}\rho v^2 + \rho g h = constant$$

so velocity v increases and static pressure P decreases at the stenotic throat. Upstream, the reduced radius r increases hydraulic resistance according to Poiseuille's law,

$$Q = \frac{\pi \Delta P r^4}{8\mu L}$$

forcing the heart to raise ΔP to maintain flow Q. Downstream, rapid flow expansion generates turbulence, high shear gradients, and energy loss, all of which amplify endothelial injury.



Illustrates how arterial geometry influences flow patterns, which determine wall shear stress distributions. These mechanical cues activate endothelial mechanotransduction pathways, leading to inflammation, plaque formation, or aneurysm progression. It also highlights engineering interventions such as flow-smoothing stents, CFD-based diagnostics, and rheology-modifying therapies.

Plaque formation is shown to localize at bifurcations, curvatures, and flow-separation zones where

shear stress is low, oscillatory, or multidirectional. These disturbed shear fields alter endothelial mechanotransduction, increasing permeability, inflammatory signalling, and lipid infiltration. Conversely, aneurysms represent the geometric inverse of stenosis: dilation reduces velocity, increases static pressure, and elevates wall tension via Laplace's law,

$$T = P.r$$

while recirculating low-shear flow promotes thrombus deposition and wall weakening.

Blood's shear-thinning rheology further modulates these phenomena. High shear rates in stenotic jets reduce apparent viscosity, whereas low shear in recirculation zones increases viscosity and stagnation, reinforcing pathological flow patterns.

By unifying classical fluid mechanics with vascular biology, this work highlights new engineering-driven opportunities for intervention, including flow-smoothing stent geometries, shear-normalizing vascular implants, rheology-modifying therapeutics, and CFD-based risk prediction tools. Also treating cardiovascular disease as a fluid-dynamics problem opens a complementary pathway for prevention and device innovation.

KEY WORDS

Hemodynamics, Bernoulli's equation, Poiseuille's law, Wall shear stress, Endothelial mechanotransduction, Atherosclerosis, Aneurysm biomechanics, Non-Newtonian blood rheology, Computational fluid dynamics (CFD), Vascular engineering, Turbulence and recirculation, Biomedical transport phenomena

BIOGRAPHY

Adjunct Professor Michael Akindeju is a distinguished engineering leader whose work spans engineering biology, advanced process design, and innovation in complex industrial systems. He serves as Principal Consultant at MKPro Group, where he delivers high-impact solutions across biotechnology, energy, and manufacturing sectors; and an Adjunct Professor with Federation University, Australia. A Fellow of IChemE and RACI, Chartered Engineer, and Registered Professional Engineer, he is recognised for integrating scientific insight with practical engineering to drive sustainable, technology-enabled transformation. Professor Akindeju is committed to strengthening industry capability, fostering research translation, and mentoring the next generation of engineers through his academic and professional contributions.

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