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Review

Wave potential: A unified model of arterial waves, reservoir phenomena and their interaction☆

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Abstract Models of haemodynamics play a central role in current research directed to understanding and addressing cardiovascular disease. Although conventional windkessel and wave models are very useful, they are incompatible due to conflicting assumptions and neither comprehensively explain the basis and interdependencies of pressure/flow waves, mean pressure and reservoir filling/discharge phenomena. The hybrid reservoir-wave model was proposed to address this gap, but is not widely accepted due to theoretical inconsistencies and negative results from validation studies. We recently described a unified model of haemodynamics based on the concept of 'wave potential', which identifies physically meaningful information from the absolute values of the forward/backward components of pressure and flow. Within this paradigm, hydraulic power may also be separated into forward/backward components, thus allowing study of time-dependent cardiac and vascular effects that influence hydraulic power output and efficiency. Based on in vivo and numerical experiments, it has been shown that 1) absolute values of the pressure/flow/power components represent wave potential, spatial gradients of which produce waves that transfer hydraulic energy, 2) mean pressure is generated by waves, 3) wave potential is a measure of local conduit arterial reservoir function and stored hydraulic energy, and 4) the diastolic pressure decay and associated 'self-cancelling' diastolic waves can be explained purely on the basis of wave reflection and distal leakage of wave potential. Wave potential provides a unified and analytically simple

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paradigm of arterial haemodynamics that extends and is fully compatible with conventional wave separation, while overcoming the difficulties encountered with the reservoir-wave paradigm.

Contents

The need for a unified model of haemodynamics

Models of haemodynamics play a foundational role in cardiovascular research, influencing the conceptual framework within which hypotheses are generated and haemodynamic variables interpreted. Desirable properties of a model include broad scope (explaining much), power (simplicity without compromising scope), internal consistency, soundness of assumptions, quantifiability (expressed mathematically and produces useful indices) and validity (accurate model predictions). Two models currently dominating the field of haemodynamics are the windkessel model and wave model. Although conflicting in terms of certain assumptions (e.g. infinite vs. finite wave speed), they are also complementary in their respective strengths for describing haemodynamics in systole (wave model) and diastole (windkessel model).

However, this conflict and complementarity in the prevailing models highlights the need for a single unified model that would likely aid investigations of cardiovascular physiology and disease. Such a model would provide an integrated analytical framework and conceptual understanding of pulse waveform features (e.g. pressure augmentation, the diastolic pressure decay and the spatial evolution of the pulse), mean pressure, blood volume storage/discharge effects in large arteries (reservoir function) and ventricular-vascular coupling dynamics. This review summarises the strengths and weaknesses of current models and provides an overview of recent efforts towards developing a unified model of haemodynamics.

Windkessel model

Windkessel models represent the arterial system with several lumped (or zero-dimensional, 0D) parameters. The original two-element windkessel, formally proposed by Otto Frank in 1899,[1] elegantly describes the reservoir function of large arteries, whereby part of the blood volume ejected by the ventricle is stored in a large artery compliance (C_{art}) during systole and is discharged through a peripheral resistance (R_p) during diastole. This model explains the exponential decay of pressure during diastole as arising from the discharge of reservoir volume, and predicts the time constant (R_pC_{art}) governing this pressure decay. Mean arterial pressure (MAP) is also explained as arising from the resistance to cardiac output (CO), i.e. $MAP = CO \times R_p + CVP$, where CVP is central venous pressure.

The key limitation of the two-element windkessel, however, is that wave speed is assumed to be infinite. The consequent rather poor prediction of systolic pressure/flow waveforms led to the development of a number of modified windkessel models with additional elements, e.g. the three-element windkessel and others.[1] Although improving the prediction of systolic haemodynamics, these models retain the assumption of infinite wave speed and hence key wave-related phenomena, such as the systolic pressure inflection and the spatial evolution of the pressure/flow pulses, fall outside their scope.

Wave model

With a rich history that will not be reviewed here (see Nichols and O'Rourke[2] and Parker[3]), wave models describe