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LETTER

Anatomy of arterial systems reveals that the major function of the heart is not to emit waves associated with the axial blood motion

The CrossTalk articles by Tyberg *et al.* (2013) and Westerhof & Westerhof (2013) and the comments about the forward and backward pressure waves in the arterial system reveal that most researchers depict the left ventricle as a wave emitter, and all the wave models arise from the axial momentum equation or the Navier–Stokes equations applied to the blood. These models not only have difficulty in defining the reflection sites, they also cannot explain many physiological observationssuch as that the heart rate is inversely proportional to the size of the animal (Milnor, 1989), that the large arterial vessels have a high distensibility and are subjected to substantial longitudinal stretching *in vivo*, and that the organs of most mammals have similar structures and connect perpendicularly to the aorta at similar relative positions. Anatomical designs are important elements in the mechanism of blood deliveryand therefore a realistic haemodynamic model should be able to provide insight into the underlying physiological effects.

Recently, taking those physiological observations into account, we integrated some haemodynamic theories(Lin Wang *et al.* 2004*a*,*b*, 2008) to construct a multi-rank distributed-cyclic-hydraulic-press (DCHP) model (Lin Wang & Wang, 2013) to describe the ventricular–arterial system and provided a quantitative method for exploring its overall behaviour. We proposed that the transportation of blood from the heart to the peripheral arterioles is via hydraulic devices of four different ranks. Since pressure is a continuous parameter, instead of taking the branching points or the side-openings as the reflection sites for waves, we treated them as sites for offering both hydraulic pressure forces and blood flow to the connecting lower rank systems. In this model, the left ventricle is depicted as the hydraulic pressure supplier of the first rank. As the blood is ejected from the left ventricle, it encounters the arch of the aorta, changes its axial velocity dramatically in both magnitude and direction. According to Newton's second law, as the blood changes its momentum, it exerts a force on the arterial wall. Thus, the periodic burst of blood from the left ventricle provides a pulsatile force to the main aorta. This pulsatile force will cause the aorta to conduct a distributed radial oscillatory motion which can be represented and measured either by the distributed radius pulse of the artery $R(z,t)$ or the accompanying pressure pulse $P(z,t)$.

The gradient of the pulse pressure $\partial P/\partial z$ also induces an inevitable axial oscillatory motion $Q(z,t)$ of the blood. However, this is not the purpose of the ventricular output. Skalak *et al.* (1966) and Milnor (1989) reported that most of the energy supplied by the left ventricle is dissipated in the friction of viscous flow, while almost all of the amount of work done in distending the arteries is returned later in each cycle of the heart beat because of the relatively small viscous behaviourof the vascular wall. Along large arteries, there is little attenuation of the pressure pulse; hence the ventricular energy input is mainly delivered via the radial pulsatile motion. To reduce energy dissipation, the axial flow in the large arteries should be as small as possible. The 180 deg bend near the arch of the aorta greatly reduces its axial kinetic energy; the ends of the ulnar artery and the radial artery join together as a loop at the palmar arch. Similarly, the deep plantar artery unites with the termination of the lateral plantar artery to complete the plantar arch (Marieb & Hoehn, 2009). At these arches, the head-on collision between blood approaching from opposite directions eliminates their axial momentum and helps to distribute blood more uniformly to all of the perpendicularly attached side-branches. These arterial structures have the effect of reducing the kinetic energy associated with the axial flow Q to only a few per cent of the energy associated the pressure P. The flow Q is a vector parameter, and is more posture dependent compared with the scalar parameter P; hence downgrading the role of Q in large arteriesmay also give mammals more freedom to change their postures.

Two important concepts are utilized in the multi-rank DCHP model(Lin Wang & Wang, 2013). First, the pressure pulse is not governed by the axial momentum equation or the Navier-Stokes equations, but by the radial momentum equations for the blood and the arterial wallor the low dissipated*PR* wave equation (Lin Wang *et al.* 2004*b*). Second, resonant behaviours between the heart and the main aorta (Lin Wang *et al.* 2004*a*, 2008), or between the aorta and different organs (Lin Wang *et al.* 1991) are plausible. Here we share our model in the expectation that itcan be used for physiological and pathological studies of arterial systems in the future.

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References

Lin Wang YY, Chang SL, Wu YE, Hsu TL & Wang WK (1991). Resonance. The missing phenomenon in hemodynamics. *Circ Res* **69**, 246–249.

Lin Wang YY, Jan MY, Shyu CS, Chiang CA & Wang WK (2004*a*). The natural frequencies of the arterial system and their relation to the heart rate. *IEEE Trans Biomed Eng* **51**, 193–195.

Lin Wang YY, Jan MY, Wang GC, Bau JG & Wang WK (2004*b*). Pressure pulse velocity is related to the longitudinal elastic properties of the artery. *Physiol Meas* **25**, 1397.

Lin Wang YY, Sze WK, Bau JG, Wang SH, Jan MY, Hsu TL & Wang WK (2008). The ventricular-arterial coupling system can be analyzed by the eigenwave modes of the whole arterial system. *App Phys Letters* **92**, 153901–153901.

Lin Wang YY & Wang WK (2013). A hemodynamics model to study the collective behaviour of the ventricular-arterial system. *J Appl Phys* **113**, 024702–024702.

Marieb EN & Hoehn K (2009). *Human Anatomy and Physiology*, 8th edn. Benjamin Cummings, San Francisco.

Milnor WR (1989). Hemodynamics, 2nd edn. Lippincott, Williams and Wilkins, Baltimore, MD, USA.

Skalak R, Wiener F, Morkin E & Fishman AP (1966). The energy distribution in the pulmonary circulation, II: Experiments. *Phys Med Biol* **11**, 437.

Tyberg JV, Bouwmeester JC, Shrive NG & Wang JJ (2013). CrossTalk opposing view: Forward and backward pressure waves in the arterial system do not represent reality. *J Physiol* **591**, 1171–1173.

Westerhof N & Westerhof BE (2013). CrossTalk proposal: Forward and backward pressure waves in the arterial system do represent reality. *J Physiol* **591**, 1167–1169.

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