



## Review

The case for the reservoir-wave approach <sup>☆</sup>John V. Tyberg <sup>a,\*</sup>, J. Christopher Bouwmeester <sup>a</sup>, Kim H. Parker <sup>b</sup>, Nigel G. Shrive <sup>c</sup>, Jiun-Jr Wang <sup>d</sup><sup>a</sup> Departments of Cardiac Sciences and Physiology/Pharmacology and Libin Cardiovascular Institute of Alberta, University of Calgary, Canada<sup>b</sup> Department of Bioengineering, Imperial College London, UK<sup>c</sup> Department of Civil Engineering, Schulich School of Engineering, University of Calgary, Canada<sup>d</sup> Department of Medicine, Fu Jen Catholic University, New Taipei City, Taiwan

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## ABSTRACT

The Reservoir-Wave Approach is an alternative, time-domain approach to arterial hemodynamics that is based on the assertion that measured pressure and flow can be resolved into their volume-related (i.e., reservoir) and wave-related (i.e., excess) components. The change in reservoir pressure is assumed to be proportional to the difference between measured inflow and calculated outflow. Wave intensity analysis of the excess components yields a pattern of aortic wave propagation and reflection in the dog that is novel and physiologically plausible: waves are reflected positively from a site in the femoral circulation and negatively from a site below the diaphragm, where the total “daughter-vessel” cross-sectional area exceeds the “mother-vessel” area. With vasodilatation, the negative reflection is augmented and with vasoconstriction, it is virtually eliminated. On the other hand, conventional hemodynamic analysis has been shown to yield a paradoxical “forward-going backward wave” and the impedance minimum, previously assumed to be an indicator of the source of wave reflection according to quarter-wave-length theory, has been shown to be due to the reservoir component. Clinical studies employing the Reservoir-Wave Approach should be undertaken to verify experimental observations and, perhaps, to gain new diagnostic and therapeutic insights.

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## 1. Introduction

The Reservoir-Wave Approach (RWA), first proposed by Wang et al. 10 years ago [1], is an alternative, novel, controversial approach to arterial hemodynamics. Essentially, it arose from the perception that  $\Delta P$ , the incremental change in pressure upon which wave intensity analysis (WIA) is based, should be “corrected” because pressure changes not only due to the passage of waves but also due to a change in volume of an elastic vessel or to a change in chamber elasticity, as occurs during ventricular contraction and relaxation. This is equivalent to the conclusion that measured pressure should be considered to be the sum of a volume-related pressure and a wave-related pressure, as illustrated in Fig. 1, a cartoon representing a canal lock (a “reservoir”) in which the level of the water rises and falls, independent of whatever wave motion may occur on the surface.

It has been claimed that the RWA has several intrinsic advantages and lacks several of the disadvantages [2] that limit conventional

“impedance analysis” [3,4]. On the other hand, it has also been claimed that the separation between reservoir pressure and “excess” or wave-related pressure is invalid and the implications of the RWA have been rejected, perhaps because they contradict some previously held postulates or because they do not agree with the results of some mathematical models.

## 2. What does “wave” mean?

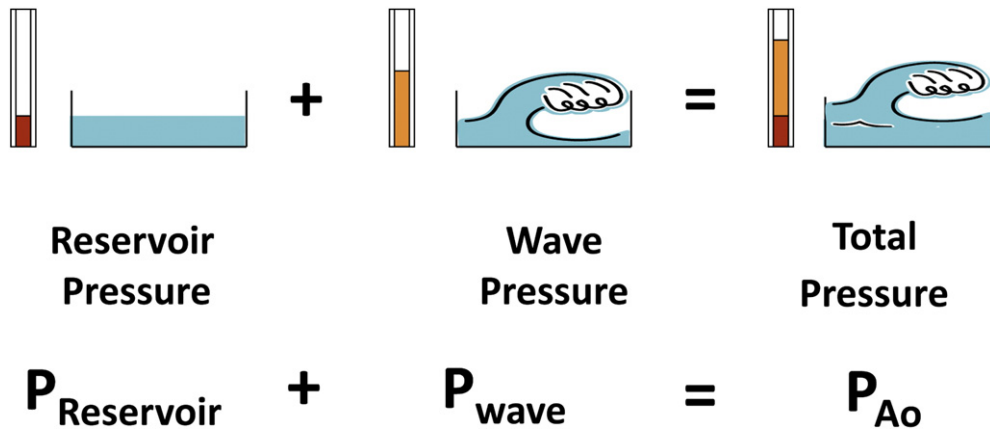
Most importantly, “wave” is defined differently by different investigators. Wang et al. [1] defined wave narrowly and designated the early systolic, forward-going compression (i.e., pressure-increasing) wave (FCW) generated by the left ventricle (LV) as the prototype wave. There are several other definitions of wave. The pressure and flow waveforms measured in a vessel or the heart are frequently called waves [5]. In the context of impedance analysis, all the constituent sinusoids are called waves [6]. Also, wave has been applied to the theoretical components of diastolic pressure or flow which, when added together, equal the measured diastolic pressure or diastolic flow (zero at the aortic root) [7]. Finally, in theoretical discussions of wave propagation and reflection, a number of infinitesimal waves have been postulated [8], waves that are undetectable by WIA but, nonetheless, in the aggregate, have been identified as the basis of reservoir pressure. [The definition of a wave is especially important and may, indeed, be the basis of much of the current controversy. The proponents of the RWA think that reservoir pressure is important and, indeed, critical because, by

*Abbreviations:* BCW, backward-going compression wave; BDW, backward-going decompression wave; FCW, forward-going compression wave; FDW, forward-going decompression wave; LV, left ventricle (ventricular); Mtx, methoxamine; NP, (sodium) nitroprusside; RWA, reservoir-wave approach; WIA, wave intensity analysis.

<sup>☆</sup> Each author takes responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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**Fig. 1.** A cartoon illustrating the concept that measured pressure is the sum of reservoir pressure and excess pressure, which is wave-related. The analogy is one of a canal lock, the level of which can rise or fall but, at any level, superimposed wave activity will be accompanied by the additional pressure required to support the wave. (Courtesy of Jacqueline Flewitt McQuaker.)

difference, it defines the change in pressure due to the passage of waves. Others believe that all pressure changes – including reservoir pressure itself – can and must be explained in terms of waves alone.]

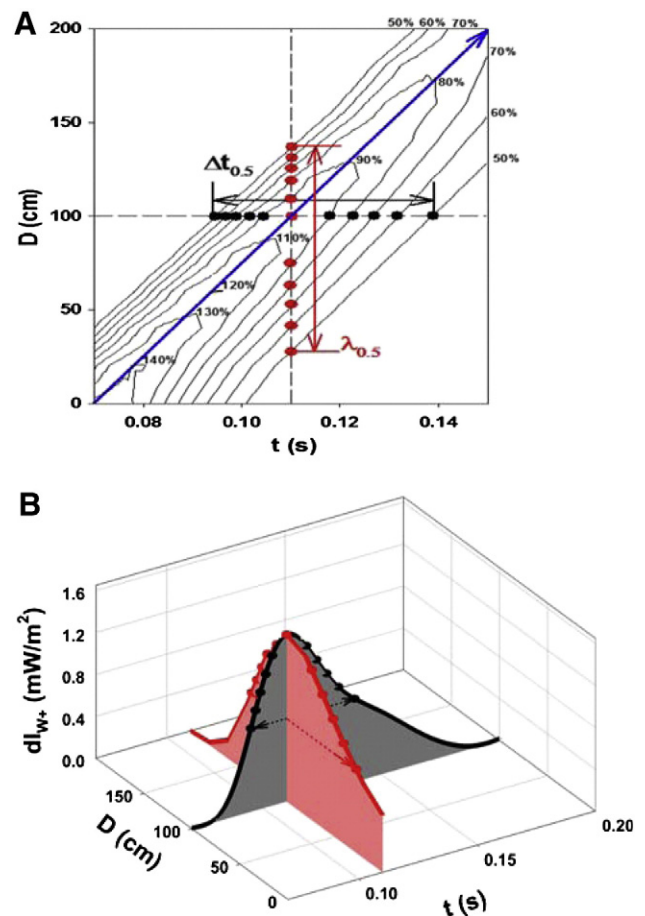
Using bench-top apparatus with which they reproducibly generated a decompression (i.e., pressure-decreasing) wave and observed its propagation and reflection in a 2-m, closed-end, plastic tube (pressure and flow were measured every 20 cm), Wang et al. defined the ensuing wave, both in its 1-dimensional spatial and temporal extents (see Fig. 2) [9]. (Because it was difficult to identify the “feet” of the wave, they referred instead to points where wave intensity was 50% of its maximal value.) The concepts of a spatial ( $\lambda_{0.5}$ ) and temporal ( $\Delta t_{0.5}$ ) “wave width” might be understood more easily with the aid of a commonplace analogy: a truck that passes an observer who stands beside a roadway. How long is the “truck”? In the context of Fig. 2A, imagine standing by the side of a “road” at the 100-cm point on the y axis as the truck passes. At 0.11 s, the front of the truck is indicated by the top red dot and the back of the truck, by the bottom red dot. Thus, the length of the truck is 110 cm [ $110 \text{ cm} = \lambda_{0.5} = (140 \text{ cm} - 30 \text{ cm})$ ]. How long does it take for the truck to pass? At the 100-cm point, the front of the truck would appear at the time indicated by the black dot furthest to the left and the back of the truck would pass the observer at the time indicated by the black dot furthest to the right. Thus, the time it takes for the truck to pass is 0.047 s [ $0.047 \text{ s} = \Delta t_{0.5} = (0.140 \text{ s} - 0.093 \text{ s})$ ]. Fig. 2B represents the shape of the wave in 3 dimensions (intensity, distance, time). Thus, according to the RWA Wang et al., have defined the spatial and temporal extents of the wave [9].

### 3. The classical 3-element Windkessel re-interpreted

Fig. 3 illustrates the re-interpretation of Westerhof’s classical 3-element Windkessel [4] according to Tyberg et al. [10].

$P_{\infty}$ . The fundamental assumption of the RWA is that during diastole arterial pressure declines exponentially and asymptotically toward a value ( $P_{\infty}$ ) that may be greater than zero. That it declines exponentially would seem plausible if it is stipulated that diastolic arterial pressure should be related to the contained blood volume and that arterial outflow is driven by the changing pressure during diastole. That pressure might not decline to zero [11] seems entirely consistent with Burton’s work on critical closing pressure [12] and Magder’s work on the Starling Resistor [13]. All these observations suggest that  $P_{\infty}$  is the pressure at which arterial outflow stops. (At  $P_{\infty}$ , there is no change in pressure;  $dP/dt = 0$ . If there is no change in pressure, there is no change in volume. If there is no change in volume, at the aortic root there is no outflow, because there is no inflow during diastole.) Wang et al. suggested that the value of  $P_{\infty}$  might be related to myogenic tone in that

$P_{\infty}$  decreased intermittently and progressively during the course of a diastolic pause that lasted several tens of seconds; myogenic tone might have decreased as the tissues became more ischemic [1].



**Fig. 2.** A. Detailed contour plot of a forward-going decompression wave as a function of distance ( $D$ ) and time ( $t$ ). Contour lines represent 10% differences from the maximum value of intensity (i.e.,  $1.6 \text{ mW/m}^2$ ) at the intersection of the black ( $D = 100 \text{ cm}$ ) and red ( $t = 0.111 \text{ s}$ ) lines. The red, double-headed arrow indicates  $\lambda_{0.5}$ . The black, double-headed arrow indicates  $\Delta t_{0.5}$ . The blue dashed arrow indicates the distance–time progression of the peak of the FDW. B. 3-D plot of the intensity of the FDW at  $D = 100 \text{ cm}$  (black) and  $t = 0.111 \text{ s}$  (red). Red and black points respectively represent 90%, 80%, 70%, 60%, and 50% of the maximum-intensity values in time and distance. See text. (From Wang et al. [9], with permission of Elsevier).

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