Peripheral Augmentation Index and Wave Reflection in the Radial Artery

To the Editor:

We were interested to read the recent article by Munir et al and congratulate them on making a valuable contribution to the field of pulse wave analysis. They and Payne and Webb, in their accompanying editorial, ponder why the late systolic shoulder (SBP₂) corresponds so closely with central systolic blood pressure and suggest that selective modification of specific frequency components of the waveform may account for this. We agree with this (indeed, it is implicit in the widespread use of a transfer function to derive central blood pressure), but we consider that the physiological basis of this relationship is not readily apparent from this frequency-based interpretation. We concur with the belief by Payne and Webb that wave separation in the time domain may provide a clearer insight into the mechanisms involved, and we have undertaken such an analysis of the radial arterial waveform based on wave intensity analysis and data collected previously.

Our findings are shown in the Figure. The data indicate that the characteristic peaked waveform seen in the radial artery is largely because of an early backward-traveling (reflected) compression wave that merges with the forward traveling compression wave attributable to cardiac ejection. This reflected wave elevates pressure and depresses flow. The early return of this wave is because of the relatively high wave speed in the radial and digital arteries and the proximity to the sites of impedance mismatching in the hand circulation. From the Figure, it is apparent that the SBP₂ corresponds with a time in systole when the intensity of the backward compression wave has declined and before the arrival of a large forward expansion wave generated by deceleration of the rate of the ventricular contraction before closure of the aortic valve. The absence of significant wave intensity at the time of SBP₂ means that pressure in the radial artery at this time should correspond relatively closely with aortic or central blood pressure, which is what Munir et al observed.

Our findings underline the value of wave intensity analysis and wave separation based on concurrent measurement of pressure and flow in understanding the hemodynamic events responsible for pressure wave morphology in the arterial tree and, we believe, provide a readily comprehensible explanation for the close correspondence between central systolic blood pressure and SBP₂ reported by Munir et al.

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Alun D. Hughes
Justin E. Davies
Darrel Francis
Jamil Mayet

International Centre for Circulatory Health
National Heart and Lung Institute Division
Imperial College London and Imperial College Healthcare National Health Service Trust
London, United Kingdom

Kim H. Parker
Department of Bioengineering
Imperial College London
London, United Kingdom

Figure. Traces show (A) blood pressure, (B) flow velocity, and (C) calculated forward and backward wave intensity in the radial artery over a single cardiac cycle (t=0 is the peak of the R wave on ECG). Pressure and flow were measured using applanation tonometry and pulsed wave ultrasound, respectively (further details of methodology are given in Reference 3) and are derived from an ensemble average of 6 individual cycles. The data shown were recorded in a healthy 49-year-old man with a brachial blood pressure of 113/80 mm Hg. SBP₂ is the late systolic shoulder as described by Munir et al, S is a forward compression wave generated by ventricular ejection, R₁ is a backward reflected compression wave, and D is a forward expansion wave generated by ventricular deceleration. The period in late systole when there is no significant wave intensity is indicated by the arrow and the gray shading.


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Alun D. Hughes, Justin E. Davies, Darrel Francis, Jamil Mayet and Kim H. Parker

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