The finger volume pulse and assessment of arterial properties

Alberto Avolio

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Graduate School of Biomedical Engineering, University of New South Wales, Sydney, Australia.

Correspondence and requests for reprints to A. Avolio, Graduate School of Biomedical Engineering, University of New South Wales, Sydney 2052, Australia.
Fax: +61 296 632108; e-mail: a.avolio@unsw.edu.au

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Introduction

The non-invasive assessment of cardiovascular function by means of the peripheral arterial pulse has gained substantial interest in recent years due to the integration of sensor technology and the ubiquitous application of microprocessors. In its most basic use, the peripheral pulse provides a signal that establishes the presence of a beating heart and quantifies the cardiac rhythm and its variability with time. However, when transduction methods are employed that relate signal output to system biophysical properties, measures of vascular haemodynamics can be derived from the pulse waveform features. In this issue of the journal, Hashimoto *et al.* [1] use parameters derived from the photoplethysmographic signal obtained in the finger to establish relations between the peripheral pulse waveform features and stiffness parameters of central large arteries.

The measurement of alteration of intensity of absorbed light due to changes in haemoglobin content with each heart beat has been applied since the late 1930s to quantify changes in skin blood flow [2,3]. Because the peripheral volume blood flow pulse is essentially due to a propagating pressure pulse, the time course of the signal indicating flow changes bears a relationship to pressure changes. While a photoplethysmographic signal can be obtained at any location on the skin surface [3,4], the finger is a convenient site at which to apply a sensor and record a continuous volume signal. This signal is used in pulse oxymetry devices for monitoring blood oxygen content [5] and to the quantify level of anaesthesia [6]. However, a substantial amount of work has emanated from Japan where the concept of analysing the finger volume signal waveshape has been applied extensively, relating pulse waveform features not only to peripheral flow, but also to arterial elastic properties and haemodynamic parameters that relate both pressure and flow. [7–10]. The assumption is that factors affecting pressure/flow relations and wave propagation will modify the peripheral volume pulse. The study by Hashimoto *et al.* [1] extends previous work by other investigators [10,11]. While these previous studies assessed changes in specific features of the second derivative of finger volume pulse, Hashimoto *et al.* [1] compare changes in these features with those in surrogate measures of arterial stiffness measured by pulse wave velocity in a group of hypertensive subjects in whom the blood pressure was normalized by antihypertenive treatment over a period of 15 years.

Photoplethysmography and the finger volume pulse

The photoplethysmogram is measured by a device comprising an infrared light source (typically a photodiode emitting light at a wavelength of around 900 nm) and a photodetector (phototransistor). The precise origin of the photoplethysmographic (PPG) signal has not been firmly established [12], but the light intensity is modulated in a complex fashion by the increase in haemoglobin content and expansion of the vascular and tissue volume between the light source and the detector, such that there is a reduction of the signal with each heart beat [12,13].

Although the PPG signal is related in time and morphology to the arterial pressure pulse, the wave contour is not the same. Millasseau *et al.* [14] have quantified the relationship between the PPG signal in the index finger and the pressure pulse obtained non-invasively by the volume clamping technique in the middle finger of the same hand. They found that the modulus of the

transfer function between the pressure signal and the PPG volume pulse showed a significant peak at approximately 6 Hz, with almost double the amplitude relative to that at heart rate frequency. This implies that the volume pulse is more damped than the pressure pulse and there is relative loss of frequency content in the region 1–6 Hz. These factors become relevant when attempting to relate volume signals to pressure-related parameters.

The vasculature in the fingers contains an abundance of alpha adrenergic receptors, so the peripheral blood flow is markedly influenced by sympathetic activity as well as temperature variations [3,15,16]. These effects can produce significant errors, such as to accentuate local effects when relating volume pulse waveform parameters to central large artery properties. These, together with problems related to cuff occlusions, are some of the potential sources of error relevant to the application of the PPG signal in the Penaz volume clamping technique [17], which is used for calibrated and continuous non-invasive measurement of arterial pressure (e.g. Finapres device [18]).

Use of the second derivative of the PPG signal

Because the PPG signal is qualitatively related to the pressure pulse, and the shape of the pressure pulse has been shown to be associated with arterial properties such as generalized arterial stiffness that occurs with age or effects of vasoactive agents [19], features of the PPG signal, such as the height of the inflection point relative to pulse height, have been used to quantify haemodynamic parameters such as wave reflection [20]. However, because of the smooth appearance of the PPG signal due to damping, other features, such as early and late systolic inflections, cannot be detected readily. Thus, the second derivative signal (SDPTG) was proposed [10] as a means to accentuate and locate inflection points and a specific nomenclature has been adopted, such that five sequential waves are designated *a*, *b*, *c*, *d* and *e*. The *a*, *b*, *c* and *d* waves occur in systole and *e* in diastole. The height is expressed in relation to the height of the *a* wave, and extensive use has been made of the parameters to quantify changes in arterial parameters [1,9,10,11]. The ratios *b*/*a* and *d*/*a* have been shown to have a strong relationship with age and *d*/*a* with systolic augmentation index derived from the central aortic pressure [10]. Because there is a known association of age and pulse wave velocity, the study by Hashimoto *et al*. [1] examines the possible relationship between absolute values of *d*/*a* and *b*/*a* with pulse wave velocity. An age index (AGI) has also been determined as $(b-c-d-e)/a$. [1,10]. While there is no specific reason given for defining AGI in this way, it was found that this parameter has a strong correlation ($r=0.75$) with age when applied to large population groups [10,11].

Pulse wave velocity and SDPTG

The methodology employed by Hashimoto *et al*. [1] to measure pulse wave velocity (PWV) utilizes an additional signal from the phonocardiogram. The transit time (t) is obtained from the pulse signals measured simultaneously at the carotid and femoral arteries and the distance (D) is obtained as the superficial distance between the second intercostal space and at the sternum and the femoral artery location. D is then corrected by a factor of 1.3 to account for the aortic curvature and t is increased by a factor tc obtained from the time difference between the second heart sound and at the incisura of the carotid pulse. Although this technique differs somewhat from the recommended guidelines developed recently [21], the PWV values calculated in this way show the expected changes with age.

While PWV measured by Hashimoto *et al*. [1] is essentially a property of the distensibility of the aortic wall and depends passively on arterial pressure, the parameters derived from the peripheral pulse contain components that depend on both central and peripheral effects. Furthermore, aortic PWV does not depend on the time course of ventricular ejection (as long as pressure remains unchanged), whereas the shape of the peripheral pulse depends on both the time course of ventricular ejection and central and peripheral wave transmission and reflection phenomena [22]. An increase in acceleration of the early ejection phase would affect the *b*/*a* ratio of the SDPTG but would not be detected as a change in PWV. Hence, when relating parameters derived from the peripheral pulse to aortic stiffness measured by PWV, the inherent assumption is that the aortic flow wave remains unchanged. Although Hashimoto *et al*. [1] do not address this issue explicitly, but mention it as a possible limitation to the study, the assumption would seem to apply to the population studied because it was essentially homogenous in terms of age (61.4 ± 10.5 years) and antihypertensive treatment (87% being on calcium channel blockers).

Overall, the study found a significant relationship between parameters of SDPTG and PWV, although the correlation coefficients were relatively low (–0.164 for *b*/*a* and 0.239 for *d*/*a*). The explanation is that PWV and SDPTG are associated with different mechanisms and reflect different information with respect to central and peripheral arterial properties. This is an important observation. However, while there may be some merit in uncovering specific haemodynamic mechanisms, it is important to acknowledge the substantial effects of the measurement technique itself and the potential sources of errors that could be present. Although there is

substantial individual variability in the SDPTG parameters, such as PWV, it can be a useful tool for studying large populations or groups with specific cardiovascular diseases. This was evident in the difference in results in subjects with uncontrolled hypertension [1,11]. A recent study has also demonstrated the potential use of this technique to study changes in arterial distensibility during development in a large ($n = 1495$) population of children and adolescents aged 9–17 years [23].

Effect of heart rate

One of the findings of the study by Hashimoto *et al*. [1] was a dependence of PWV and *b*/*a* and *d*/*a* ratios on heart rate. If this is independent of blood pressure, the implication is that heart rate has an intrinsic effect on large artery stiffness. This observation has to be taken with caution, especially if PWV is measured in the aortic trunk, where the passive pressure effects on wall stiffness are much greater than active effects due to sympathetic influences on smooth muscle tone. Although a number of studies have shown a heart rate dependence on large artery stiffness [24], the possibility of measurement errors in accurate calculation of transit times or pulse waveform features cannot be discounted, especially in studies showing the dependence of heart rate and arterial stiffness with cardiac pacing [25]. A recent study by Green *et al*. [26] has shown no effect of cardiac pacing on forearm vascular response when measured in terms of changes in diameter for the same pulse pressure.

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