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Non-invasive model-based estimation of aortic pulse pressure using suprasystolic brachial pressure waveforms

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ABSTRACT

Elevated central arterial (aortic) blood pressure is related to increased risk of cardiovascular disease. Methods of non-invasively estimating this pressure would therefore be helpful in clinical practice. To achieve this goal, a physics-based model is derived to correlate the arterial pressure under a suprasystolic upper-arm cuff to the aortic pressure. The model assumptions are particularly applicable to the measurement method and result in a time–domain relation with two parameters, namely, the wave propagation transit time and the reflection coefficient at the cuff. Central pressures estimated by the model were derived from completely automatic, non-invasive measurement of brachial blood pressure and suprasystolic waveform and were compared to simultaneous invasive catheter measurements in 16 subjects. Systolic blood pressure agreement, mean (standard deviation) of difference was −1 (7) mm Hg. Diastolic blood pressure agreement was 4 (4) mm Hg. Correlation between estimated and actual central waveforms was greater than 90%. Individualization of model parameters did not significantly improve systolic and diastolic pressure agreement, but increased waveform correlation. Further research is necessary to confirm that more accurate brachial pressure measurement improves central pressure estimation.

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1. Introduction

Commentary on the recent literature has identified "large artery stiffness" as being a biomarker for cardiovascular disease in humans (Franklin, 2008). However, the concept of large artery stiffness is not precisely defined and has been further subdivided into regional, local and systemic stiffness. Each of these concepts may be measured using a variety of techniques (Laurent et al., 2006) producing a plethora of candidate biomarkers for cardiovascular disease.

A framework for the evaluation of the clinical value of biomarkers has been suggested (Vasan, 2006). In consideration of these, the European Network for Non-invasive Investigation of Large Arteries has released a position statement (Laurent et al., 2006) highlighting three methods for measuring arterial stiffness, of which one is central pulse-waveform analysis (including central pressure and augmentation index). Pulse-waveform analysis via applanation tonometry has received much attention following studies which have shown that central blood pressure (but not brachial pressure) predicts cardiovascular events in aged populations (Pini et al., 2008) and is more strongly related to vascular hypertrophy, atherosclerosis and cardiovascular events (Roman et al., 2007). It has been suggested that measurement of central pressure may improve the identification and management of patients at increased risk of cardiovascular diseases (McEniery et al., 2008).

A number of methods for measurement of central (aortic) blood pressure exist. Invasive measurement by aortic catheterization is considered the gold standard. Less invasive estimation of central pressure can be performed by the application of a generalized transfer function to a calibrated waveform obtained from radial artery tonometry (Chen et al., 1997). Calibration (and validation of the technique) is performed against accurate intra-arterial radial manometer systems (Cockcroft and Wilkinson, 2002; Pauca et al., 2001), although in practice a non-invasive upper-arm blood pressure monitor is used for calibration, potentially introducing further error into the estimate of central blood pressure. There remains concern in some quarters about the accuracy of generalized transfer functions in particular patient groups (Hope et al., 2004).

In contrast to the generalized transfer function approach, various researchers have attempted to individualize the transfer function to specific patients based on a model of the arterial system. Segers et al. (2000) attempted to apply a transmission line model of the radial–aortic/carotid segment with parameters

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related to readily measured indices or patient characteristics, such as age, blood pressure; however, they were unable to find any relation. Stergiopulos et al. (1998) developed a model of forward- and backward-propagating wave components to relate aortic pressure to peripheral pressure and velocity with model's parameters having explicit physical meaning. Validation of the technique against a computational model of an arterial tree showed that the central pressure waveform could be accurately reconstructed.

The technique of Stergiopulos requires the peripheral measurement of both pressure and velocity waveforms. These may be obtained by radial applanation tonometry and Doppler flow velocity measurement, respectively. However, these techniques both require significant skill to obtain reproducible results, and cannot easily be obtained both simultaneously and ipsilaterally. Clinical validation of the method is, therefore, difficult.

2. Method

In this study, a model based on the theory of pressure wave reflections is coupled with particular measurement techniques to result in a clinically applicable method for determining aortic pressures. The method does not require measurement of peripheral velocity waveforms and some results are presented from a clinical trial.

2.1. Arterial model

The system under consideration is the pressure wave propagation through a volume of blood enclosed by the left subclavian and brachial arteries, see Fig. 1. At the brachial end, a suprasystolic cuff is applied to almost completely occlude the artery (Payne et al., 2007). In this work, the external pulse is transduced directly from the suprasystolic cuff pressure.

Previously published work by our group describes the derivation of the general form of the pressure wave propagation model based on a simplified water-hammer acoustic model (El-Aklouk et al., 2008a). In this model, the wave travels along the artery unaffected in shape, only delayed as suggested by Stergiopulos et al. (1998).

In this work, we additionally assume that artery has parallel sides (Westerhof et al., 1969), but that the end conditions of the artery cause an abrupt change in impedance. Finite element models show that the radius transition under the suprasystolic cuff happens within a few millimeters (Lan et al., 2008). The presented model is most correct for the left arm as the right subclavian artery is one branching generation removed from the aorta. There is no reflection from the distal end of the cuff and in practice, no pulse can be heard or palpitated at the distal edge of a suprasystolic cuff.

Note that many of these assumptions do not hold as well for other measurement techniques. In particular, the use of an arterial line or tonometry method does not allow the assumption of a constant, abrupt change in impedance nor an assumption of zero blood flow as the impedance is provided by the peripheral circulation, including arterioles and capillaries. Measurement at a radial site also introduces a significant bifurcation into the arterial system being studied, as well as further compromising the thin-walled tube assumption.

Using the above assumptions, we may derive the time–domain relationship between the total oscillatory pressure in the aorta, p_{t0}, and total oscillatory pressure under the essentially occluding cuff, p_{t3}, to be

$$p_{t0}(t) = \frac{b}{b+1} p_{t3}(t-dt) + \frac{1}{b+1} p_{t3}(t+dt) \quad (1)$$

where t is the time variable, b the reflection coefficient for a pressure wave travelling distally at the cuff, and dt the time taken for a pressure wave to travel from the subclavian root to the cuff occlusion.

It can be seen that under the conditions and assumptions described above, the aortic pressure waveform can be easily reconstructed in the time–domain from the pressure at the occlusion, using only two parameters, b and dt.

The waveform p_{t3} is assumed to be related to the cuff pressure oscillation in accordance with form described by El-Aklouk et al. (2008b).

2.2. Clinical testing

Clinical evaluation of the estimation method was performed at the Auckland City Hospital. Twenty-two patients undergoing elective diagnostic cardiac catheterization were enrolled. Pre-procedure sedation, vascular access and aortic catheterization followed our standard laboratory protocols. The tip of the cardiac catheter was placed in the aortic root and connected to a calibrated fluid-filled pressure transducer. Brachial blood pressure and suprasystolic pressure waveforms were recorded using an R6.5 monitor (Pulsecor Limited, Auckland, New Zealand). The R6.5 monitor incorporates a POEM2 oscillometric blood pressure module (Welch Allyn, Skaneateles Falls, NY, USA) which has been shown to meet the Association for the Advancement of Medical Instrumentation (AAMI) SP10 specification for blood pressure measurement, and achieve an A/A grade under the British Hypertension Society (BHS) evaluation protocol. The device first measures blood pressure, then automatically inflates to 25 mm Hg above the measured systolic pressure. Simultaneous recordings of aortic pressure and suprasystolic brachial cuff waveforms were made.

2.3. Data analysis

Digitized, scaled, suprasystolic pressure waves were processed using Mathematica version 6.0.3 (Wolfram Research Inc., Champaign, IL) to obtain the mean beat over the 10 s of collected data by fitting a Fourier series with a fundamental frequency equal to the heart rate. Eq. (1) was then applied to reconstruct the average central (subclavian root) pulsatile pressure waveform, which was assumed to oscillate about the measured central mean pressure. Systolic and diastolic pressures were taken to be the maximum and minimum of the estimated central pressure waveform, respectively.

Actual systolic and diastolic pressures were obtained from the 10 s of recorded, invasive pressures by taking the mean of systolic and diastolic pressures for each pulse. As described in Westerhof et al. (2008), patients with cardiovascular instability resulting in aortic and brachial mean pressure differences of >9 mm Hg were excluded. Agreement between estimated and actual pressures was performed using the method of Bland and Altman (1986). Results for agreement between paired measures are reported as mean difference $\pm$ twice standard deviation of the difference, where actual central blood pressure is the subtrahend

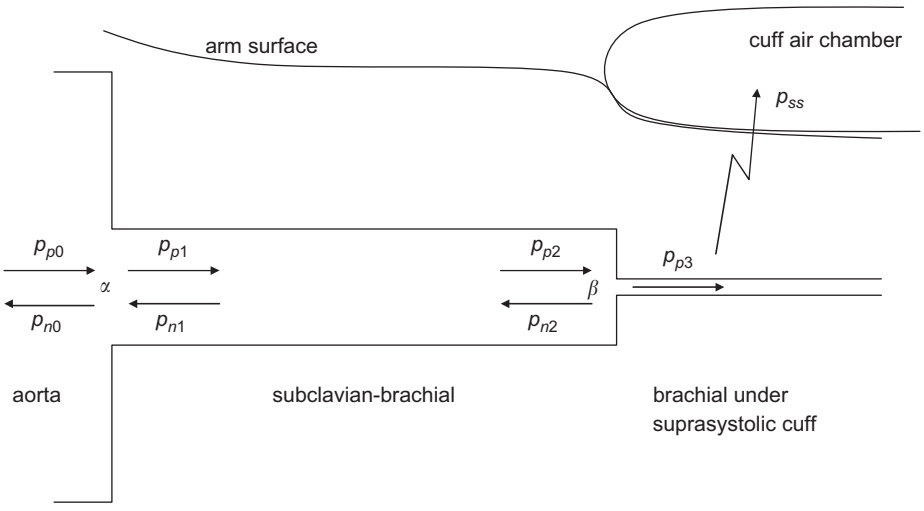

Fig. 1. Diagram of arterial model pressure wave propagation.

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