Estimating Relative Change in Ventricular Stroke Work from Aortic Pressure Alone: Proof of Concept Study

S.Kamoi[1], C.G.Pretty[1], Y.S.Chiew[1], A.Pironet[2], T.Desaive[2], G.M.Shaw[3], J.G.Chase[1]
[1]Department of Mechanical Engineering, University of Canterbury, Christchurch, New Zealand,
(e-mail: shun.kamoi@pg.canterbury.ac.nz)
[2]GIGA Cardiovascular Science, University of Liege, Liege, Belgium
[3]Intensive Care Unit, Christchurch Hospital, Christchurch, New Zealand

Abstract

Continuous Ventricular Stroke Work (VSW) estimation requires accurate estimate of both stroke volume and aortic pressure. However, accurate beat-to-beat stroke volume measurement is highly invasive and thus typically unavailable in clinical practice. This study analyses the accuracy of a model-based method estimating relative change in VSW using only aortic pressure measurements. Using data from porcine experiment, the correlation coefficient was determined between the relative change of VSW from directly measured data and the model-based estimate of VSW. The result showed good agreement with, R=0.71. The model accurately captured the trend of VSW using only aortic pressure measurements and thus offers significant clinical value in early diagnosis and improving care for cardiovascular dysfunction.

1 INTRODUCTION

Ventricular Stroke Work (VSW) is an important physiological parameter for assessing cardiovascular function [1]. Decreased VSW is related to many cardiovascular dysfunctions and initial signs of heart failure [2]. Providing real-time continuous estimation of VSW could enable diagnosis of shock at an early stage improving patient outcomes [3].

VSW can be calculated using measured Stroke Volume (SV) and Mean Aortic Pressure (MAP) [4]. However, accurate, continuous SV measurement requires highly invasive instrumentation, such as admittance or conductance catheters directly inserted into the ventricles, and thus are not clinically feasible. Currently, intermittent estimates of SV are made by thermodilution or continuous estimates using continuous cardiac ouput monitors [5]. However, derived SV in the latter case are simply functions of MAP and the accuracy is low during haemodynamic instability due to fixing certain physiological parameters [6].

This paper presents an aortic model for estimating relative change in VSW from aortic pressure measurement. Pressure-velocity (PU) gradient in the aortic compartment is identified from aortic pressure contour and the relationship between VSW and the gradient is analyzed. The presented method updates physiological parameters every heart beat and thus, eliminates inaccuracy from constraining key metrics. To validate the model, the correlation was determined between measured VSW and estimated aortic PU gradient.

2 METHODS

The aortic model in this study incorporates two existing theories on flow in an elastic tube: 1) reservoir-excess pressure [7]; and 2) Pulse wave propagation [8]. The schematic of the process for estimating relative change in VSW from aortic pressure measurement is shown in Figure 1.

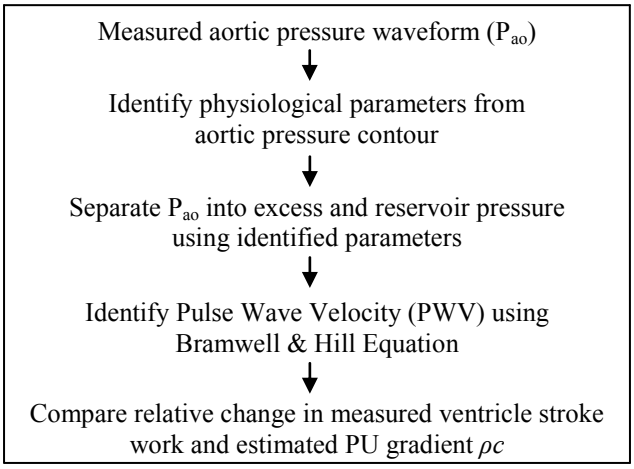

Figure 1 - Schematic of estimation procedure

2.1 Porcine Experiments

Experiments were performed on pure pietrain pig. During the experiment, several step-wise positive end expiratory pressure (PEEP) recruitment manoeuvres (RM) were performed, causing changes to SV and VSW [9]. This experiment were conducted to investigate respiratory failure, but extensive CVS measurements were recorded. Details of the experimental procedure are published elsewhere [10].

Left ventricular volumes and pressures were measured using 7F admittance catheters (Transonic Scisense Inc., Ontario, Canada) inserted directly into the ventricles through the cardiac wall. Proximal aortic pressure was measured with a 7F pressure catheter (Transonic Scisense Inc., Ontario, Canada) inserted into the aortic arch through the carotid artery. All data were sampled at 200

Hz and were subsequently analysed using Matlab (version 2013a, The Mathworks, Natick, Massachusetts, USA).

2.2 Aortic Model

Continuous aortic pressure waveforms were split into individual heart beat for analysis of beat-to-beat changes in the arterial energy. For each beat, model-based analysis of the pressure contour was performed to estimate aortic PU gradient.

2.2.1 Reservoir-Excess Separation

The pressure separation method used in the aortic model is based on the theory proposed by Wang *et al* 2003 [11]. The theory states that aortic pressure (P_{ao}) can be separated into two components, reservoir and excess pressure. Reservoir pressure (P_{res}), represents energy stored and released by the volumetric change in the aortic compartment. Excess pressure (P_{ex}), defined as the difference between the measured aortic pressure and the reservoir pressure, which accounts for the propagation of waves through the aorta.

$$P_{ao}(t) = P_{res}(t) + P_{ex}(t) \tag{1}$$

The paper also describe the linear proportionality between each pressure component and flow dynamics in the aortic compartment

$$P_{res}(t) - P_{msf} \; \alpha \; Q_{out}(t) \;\; , \;\; P_{ex}(t) \; \alpha \; Q_{in}(t) \tag{2}$$

Where Q_{in}, Q_{out}, and P_{msf} are flow entering/leaving the aortic compartment and mean systemic filling pressure [12], respectively. In this case, proportionality constants are peripheral resistance (R), and characteristic impedance (R_p) for Q_{in} and Q_{out}, [13] respectively. By assuming that pressure decay in the diastolic region results from only volumetric change of the arterial compartment [14] and identifying the exponential decay time constant (τ) in diastole [15]. The diastolic reservoir pressure can be expressed:

$$P_{res}(t) = \left(P_{ao}(t_d) - P_{msf}\right)e^{-\frac{(t-t_d)}{\tau}} + P_{msf} \tag{4}$$

Where t_d is the time of closure of the aortic valve. In this model, the identified exponential decay time constant τ represents product of peripheral resistance and compliance of the aortic wall ($\tau = RC$) [16], and the start of diastole was defined by the time of the minimum rate of change of P_{ao} [17].

To identify reservoir pressure during the whole cycle, conservation of mass and proportionality described in Equation (2) was applied:

$$\frac{dV(t)}{dt} = Q_{in}(t) - Q_{out}(t) \tag{5}$$

$$\frac{dP_{res}(t)}{dt} = a(P_{ao}(t) - P_{res}(t)) - b(P_{res}(t) - P_{msf}) \tag{6}$$

Where a and b are $1/R_pC$ and $1/\tau$ respectively. The analytical solution to Equation (6) for P_{res} is defined:

$$P_{res}(t) = e^{-\beta t}\left(\int_0^t e^{\beta t'}\left(aP_{ao}(t') + bP_{msf}\right)dt' + P_{res,0}\right) \tag{7}$$

Where $\beta = a + b$. The identification of b involves additional assumption that Q_{in} equals Q_{out} in the region between the point of maximum P_{ao} and t_d due to aortic valve closure. Using this additional information, Equations (5) and (6) can be rearranged:

$$\frac{dV(T)}{dt} = 0 \tag{8}$$

$$P_{res}(T) = \frac{aP_{ao}(T) + bP_{msf}}{a + b} \tag{9}$$

Where T is the time when Q_{in} equals Q_{out}. The estimate of b is identified by nonlinear regression using Equations (7) and (9) with maximum value of aortic pressure as first guess of P_{ao} at time T. To identify the correct value of b, the value of P_{ao} is iterated until Equation (8) is satisfied.

Once the parameters a and b are identified from the aortic pressure contour, P_{ao} is separated into reservoir and excess pressure using Equations (7) and (1). Example of the separated pressures and flow dynamics in the aortic compartment for the whole heart beat is shown in Figure 2. In addition, volume stored and released by the aortic system (ΔV_{ao}) is also shown as the difference between area under the curve of Q_{in} and Q_{out}.

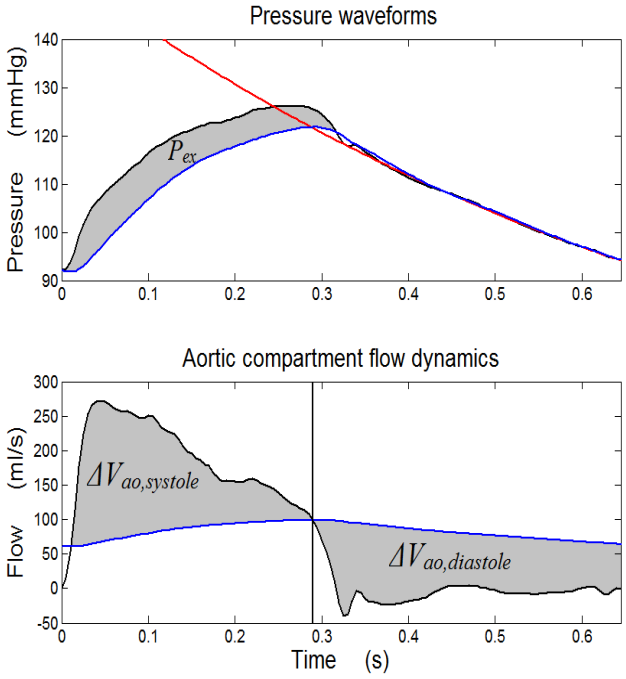

Figure 2 - Top panel: example of aortic pressure separation showing estimated diastolic curve (red line), reservoir pressure P_{res} (blue line), excess pressure P_{ex} (shaded grey area), and measured aortic pressure P_{ao} (black line). Bottom panel: Estimated aortic inflow Q_{in} (black line), outflow Q_{out} (blue line), and zero net flow time T (vertical black line), and volume changes in aortic comparment (shaded grey area).

2.2.2 Pulse Wave Velocity

To determine the velocity of the pulse wave through aortic compartment, the Bramwell-Hill equation was applied [18]:

$$c = (\rho D)^{-1/2} \tag{10}$$

$$D = \frac{\Delta V_{ao}}{V_{ao}\Delta P_{ex}} \tag{11}$$

Where ρ and D are blood density and aortic distensibility, respectively, and c is PWV. The advantage of applying this theory is that knowledge on thickness, elasticity or radius of the arterial wall is not required to determine distensibility. In addition, only the value of aortic volume proportionality is necessary and the exact absolute value of volumetric change in the aortic compartment is not required.

To determine aortic distensibility, the integral of the seprated pressure waveforms P_{res} was used with the assumption that aortic compartment volume at the start of systole equals the volume at the end of diastole [19].

$$\Delta V_{ao}(systole) = \Delta V_{ao}(diastole) \tag{12}$$

$$\frac{\Delta V_{ao}}{V_{ao}} = \frac{\int_{t_d}^{t_f} P_{res}(t) - P_{msf}\, dt}{\int_{t_0}^{t_d} P_{res}(t) - P_{msf}\, dt} \tag{13}$$

Where t_0 and t_f are time at start and end of the heart beat, respectively. Once the volume proportionality is identified, Equations (10) and (11) are used to calculate PWV in the aortic compartment. Finally, the pressure and velocity relationship ρc defined by the Joukowsky equation [20] can be determined.

$$\rho c = \frac{\Delta P}{\Delta V} \tag{14}$$

2.2.3 Data Analysis

The original aortic waveform data was pre-processed by removing regions where obvious measurement error occurred due to equipment, catheter disturbance or failure. In this study, VSW were calculated from the area enclosed by the measured left ventricular pressure-volume ($P_{lv} - V_{lv}$) loop .

$$VSW = \int^{v_{lv}} P_{lv}(v_{lv})dv_{lv} \tag{15}$$

2.2.4 Limitations

Data from only one pig were analyzed in this study. However, more than 1500 heart beats across a range of SV values induced by changes in PEEP were analyzed. Thus, despite the analysis being subject specific, this study demostrates the feasibility of accurately and continuously identifying the relative change in VSW using only aortic pressure measurements.

To fully examine the robustness in capturing the fluctuations in VSW using this aortic model, further study needs to be carried out with aortic pressure contours influenced by a range of cardiovascular dysfunction.

3 Results

Investigated ranges of SV, VSW, and MAP in this study are presented in Table 1. The estimated ρc compared to the calculated VSW are presented in Figure 3. Correlation plots between estimated ρc and measured VSW are presented in Figure 4.

Table 1: Investigated range of physiological parameters SV, VSW, and MAP. Data are presented as the median [5-95th percentiles]

Investigated Physiological Range		
SV (ml)	***VSW*** (Joule)	***MAP*** (mmHg)
24.1[14.9-28.8]	0.27[0.15-0.33]	80.7 [70.8-96.2]

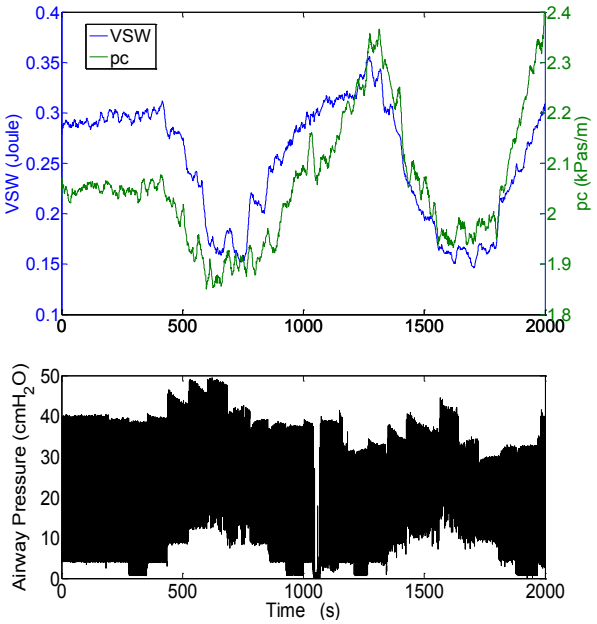

Figure 3 – Top Panel: showing VSW variation induced by recruitment maneuvers (blue line) and estimated PU gradient value pc from model based analysis of aortic pressure contour (green line). Bottom Panel: simultaneously measured airway pressure to show the PEEP changes.

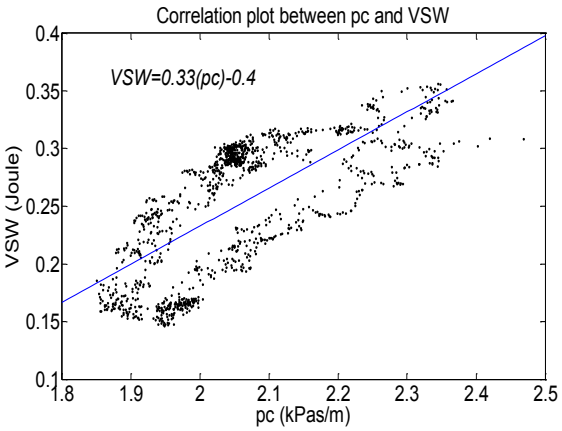

Figure 4 – Estimated pc versus calculated VSW showing relationship between the two parameters. The plot indicates that there are high degree of agreement with correlation coefficient of R=0.71.

4 Conclusion

Physiological models are simplified representations of reality that can provide clinicians with information for decision making, without the need for additional invasive direct measurements. The model presented in this study show the potential for continuous, accurate VSW trend to be captured by estimating PU relationship in the aortic compartment and using the identified value as an index to evaluate relative change in VSW. Incorporating this model in clinical settings can lead to not only early diagnosis of deteriorating patients, it enables the response of clinical interventions such as inotropes, vasoactive, and fluid therapy to be evaluated. The aortic model shows the ability for improving cardiac and circulatory treatment in the critical care environment.

5 References

[1] S. Tibby and I. Murdoch, "Monitoring cardiac function in intensive care," Archives of disease in childhood, vol. 88, pp. 46-52, 2003.

[2] J. Hunter and M. Doddi, "Sepsis and the heart," British journal of anaesthesia, vol. 104, pp. 3-11, 2010.

[3] E. Rivers, B. Nguyen, S. Havstad, J. Ressler, A. Muzzin, B. Knoblich*, et al.*, "Early goal-directed therapy in the treatment of severe sepsis and septic shock," New England Journal of Medicine, vol. 345, pp. 1368-1377, 2001.

[4] S. J. Sarnoff and E. Berglund, "Ventricular function I. Starling's law of the heart studied by means of simultaneous right and left ventricular function curves in the dog," Circulation, vol. 9, pp. 706-718, 1954.

[5] J. A. Alhashemi, M. Cecconi, and C. K. Hofer, "Cardiac output monitoring: an integrative perspective," Crit Care, vol. 15, p. 214, 2011.

[6] L. Camporota and R. Beale, "Pitfalls in haemodynamic monitoring based on the arterial pressure waveform," Crit Care, vol. 14, p. 124, 2010.

[7] J. Lighthill, Waves in fluids: Cambridge university press, 2001.

[8] F. N. van de Vosse and N. Stergiopulos, "Pulse wave propagation in the arterial tree," Annual Review of Fluid Mechanics, vol. 43, pp. 467-499, 2011.

[9] T. Luecke and P. Pelosi, "Clinical review: positive end-expiratory pressure and cardiac output," Critical Care, vol. 9, p. 607, 2005.

[10] V. E. Drunen, Y. Chiew, Z. Zhao, B. Lambermont, N. Janssen, C. Pretty*, et al.*, "Visualisation of Time-Variant Respiratory System Elastance in ARDS Models," Biomedical Engineering/Biomedizinische Technik, 2013.

[11] J.-J. Wang, A. B. O'Brien, N. G. Shrive, K. H. Parker, and J. V. Tyberg, "Time-domain representation of ventricular-arterial coupling as a windkessel and wave system," American Journal of Physiology-Heart and Circulatory Physiology, vol. 284, pp. H1358-H1368, 2003.

[12] J. J. Maas, B. F. Geerts, P. C. van den Berg, M. R. Pinsky, and J. R. Jansen, "Assessment of venous return curve and mean systemic filling pressure in postoperative cardiac surgery patients*," Critical care medicine, vol. 37, pp. 912-918, 2009.

[13] K. H. Parker, J. Alastruey, and G.-B. Stan, "Arterial reservoir-excess pressure and ventricular work," Medical & biological engineering & computing, vol. 50, pp. 419-424, 2012.

[14] G. G. Belz, "Elastic properties and Windkessel function of the human aorta," Cardiovascular Drugs and Therapy, vol. 9, pp. 73-83, 1995.

[15] W. Guier, G. Friesinger, and R. Ross, "Beat-by-beat stroke volume from aortic-pulse-pressure analysis," Biomedical Engineering, IEEE Transactions on, pp. 285-292, 1974.

[16] N. Westerhof, J.-W. Lankhaar, and B. E. Westerhof, "The arterial windkessel," Medical & biological engineering & computing, vol. 47, pp. 131-141, 2009.

[17] F. L. Abel, "Maximal negative dP/dt as an indicator of end of systole," American Journal of Physiology-Heart and Circulatory Physiology, vol. 240, pp. H676-H679, 1981.

[18] J. C. Bramwell and A. V. Hill, "The velocity of the pulse wave in man," Proceedings of the Royal Society of London. Series B, Containing Papers of a Biological Character, pp. 298-306, 1922.

[19] R. H. Thiele and M. E. Durieux, "Arterial waveform analysis for the anesthesiologist: past, present, and future concepts," Anesthesia & Analgesia, vol. 113, pp. 766-776, 2011.

[20] A. S. Tijsseling and A. Anderson, "The Joukowsky equation for fluids and solids," in Proceedings of the 9th International Conference on Pressure Surges, 2004, pp. 739-751.