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Robust computation of pulse pressure variations

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ABSTRACT

Evidence of arterial pulse pressure variations caused by cardio-pulmonary interactions, and their connection to volume status via the Frank–Starling relationship, are well documented in the literature. Computation of pulse pressure variations from arterial pressure measurements is complicated by the fact that systolic and diastolic peaks are not evenly spaced in time. A robust, structurally uncomplicated, and computationally cheap algorithm, specifically addressing this fact, is presented. The algorithm is based on the Lomb–Scargle spectral density estimator, and ordinary least squares fitting. It is introduced using illustrative examples, and successfully demonstrated on a challenging porcine data set.

1. Introduction

1.1. Cardio-pulmonary variations

One of the earliest descriptions of cardio-pulmonary interaction dates back to 1854, and is due to Greisinger. His discovery lay the foundation for Kussmaul's 1873 description of the pulsus paradox in his paper *Über schwielge Madiastino-pericarditis und den Paradoxen Puls*[1] [1].

Cardio-pulmonary interactions are a consequence of changes in intrathoracic pressure over the respiratory cycle. The schematic illustration of Fig. 1, together with elementary physics, is sufficient to explain these interactions.

A decrease[2] in intrathoracic pressure (during spontaneous inspiration, or the expiratory phase of positive pressure ventilation), results in increased venous return. This is particularly true for the right heart, which receives systemic venous return. The returned blood originates from outside the thorax, and the flow is consequently aided by the decreased intrathoracic pressure. Since the lungs reside within the thorax, the pulmonary venous return, to the left heart, is not affected by this pressure gradient. However, the increased systemic venous return associated with decreased intrathoracic pressure, results in increased pulmonary blood flow, and consequently an increased filling (preload) of the left ventricle. The decreased intrathoracic pressure, increases the load on the left heart, as it is ejecting blood out of the thorax via the aorta. As long as the left heart can cope with this relative increase in afterload, there is an increase in left ventricular stroke volume.

To conclude, the intrathoracic pressure variation over the respiratory cycle results in a left ventricular stroke volume variation, via the cardio-pulmonary interactions described above. While present to some extent during spontaneous breathing, the variation increases notably in patients under positive pressure ventilation. A comprehensive summary of the subject is provided in [2].

The left ventricular stroke volume (SV) is related to arterial pulse pressure (PP), being the difference between systolic and diastolic pressure, through Laplace law. While SV and PP cannot be assumed to be linearly proportional [3], they exhibit a strong correlation. Consequently, stroke volume variations (ΔSV) cause pulse pressure variations (ΔPP[3]) over the respiratory cycle [4]. This is of clinical interest, as measurement of pulse pressure variations, invasively by arterial catetherization, or noninvasively by pulse plethysmograph (PPG) [5], is less complicated than that of stroke volume.

1.2. Pulse pressure variations and hydration status

Evidence linking cardio-pulmonary variations to hydration status was first presented in 1983 [6]. Since then, numerous reports have contributed to strengthen this evidence. It has also been shown that cardio-pulmonary variations constitute a better predictor of hydration status than static parameters, such as central venous pressure. See [7] for a comprehensive review.

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[1] English translation: Concerning calous mediastinopericarditis and the paradoxical pulse.

[2] The use of 'decrease' and 'increase', is with respect to other phases of the respiratory cycle. I.e., we are only considering variations induced by respiration.

[3] Sometimes PPV is used to denote pulse pressure variations. However, we prefer ΔPP, as PPV can be confused with positive pressure ventilation.

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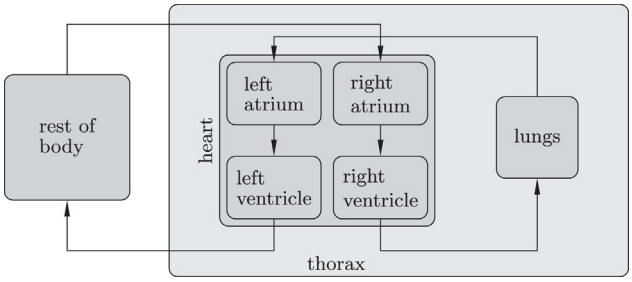

Fig. 1. Schematic drawing of the heart and lungs enclosed in the thorax, which can be viewed as a pressure chamber, with pressure variations over the respiratory cycle. The right heart receives systemic venous return from outside the thorax, and ejects the returned blood into the pulmonary circulatory system, residing inside the thorax. The left heart receives pulmonary venous return, from inside the thorax, and ejects the returned blood into the systemic circulatory system, outside the thorax.

The link between cardio-pulmonary variations and hydration status can be explained using the Frank–Starling relationship, conceptually illustrated in Fig. 2, and originally due to Maestrini (1886–1975), rather than Frank and Starling, after whom it is named. The curve relates end-diastolic volume to stroke volume of the left heart. As described above, end-diastolic volume varies with intrathoracic pressure over the respiratory cycle, illustrated by vertical grey line pairs. As seen in Fig. 2, the magnitude of the corresponding ΔSV is larger at the steep section of the curve, than at the plateau.

Due to the relation between venous return and end-diastolic pressure, the latter is increased if a blood volume expansion is performed through intravenous fluid administration. I.e., volume expansion corresponds to moving to the right in Fig. 2, resulting in smaller ΔSV.

One of the main objectives of intravenous fluid management, in intensive care and a majority of surgeries, is to maximize cardiac output (CO) [2], defined as the product of left ventricular stroke volume and heart rate. Consequently, it is motivated to perform volume expansion until the plateau of the Frank–Starling curve of Fig. 2 is reached. Once the plateau is reached, further volume expansion only marginally increases stroke volume, and consequently cardiac output. Relatedly, further volume expansion may lead to hyperhydration, with adverse effects, including increased mortality in some patient groups, as described in, e.g., [8]. Exploiting the previously mentioned correlation between ΔSV and ΔPP, the rationale is therefore to perform volume expansion, as long as it results in decreased ΔPP.

1.3. Pulse pressure variation algorithms

There exist several commercial monitors, which compute cardio-pulmonary variation indices, such as ΔPP, with the intention to guide clinicians in the titration of intravenous fluids. Most of these monitors, including the PiCCO (Pulsion Medical Systems, Feldkirchen, Germany) and FlowTrac (Edwards Life Science, Irvine, CA), utilize propriety algorithm. As pointed out in [7], the availability of open algorithms is essential for the research community. This is particularly true for research on closed-loop controlled fluid management systems [9,10], where dynamics of the monitor influence the closed-loop system; its performance, robustness, and ultimately even its stability.

While there exist a multitude of algorithms for estimation of respiratory rate from PPG in spontaneously breathing individuals [11], there exist few published algorithms for the computation of pulmonary variation indices in mechanically ventilated patients. A notable exception is the algorithm published in [12], and implemented in the Philips Intelivue MP70 (Philips Medical System, Suresnes, France). That algorithm relies on peak detection, with subsequent estimation of enveloping functions. Unlike the algorithm to be proposed herein, it relies on uniform resampling of data, does not impose structure (e.g., sinusoidal) on the envelopes, and does not define pulse pressure between beats.

1.4. Robust computation of pulse pressure variations

A simple algorithm for fast online computation of arterial pulse pressure variations (ΔPP) will be introduced, demonstrated, and discussed. The algorithm has few parameters, which all have intuitive interpretations, enabling the recommendation of sound default values. It is robust to outliers, and provides a measure of its output confidence. Minimal example implementations in the Matlab programming language can be downloaded from [13].

Demonstration of the algorithm is performed using porcine arterial pressure data, sampled at 100 Hz by catetherization of the ascending aorta. The data was acquired as part of a closed-loop hemodynamic stabilization project. Experimental conditions and compliance with ethical standards were reported in [14]. Details concerning study ethics are additionally found at the end of this paper, under “Compliance with ethical standards”. Cardiovascular similarities between human physiology and porcine models [15], support the thesis that successful demonstration of the proposed algorithm in a porcine model indicates applicability to human arterial pressure data.

2. Algorithm

This section presents a robust, simple, and computationally cheap algorithm for computation of ΔPP (%), defined through:

$$\Delta\mathrm{PP} = 100 \cdot \frac{\mathrm{PP}_{\max} - \mathrm{PP}_{\min}}{\mathrm{PP}_{\mathrm{mean}}}, \tag{1}$$

where the max, min, and mean of the pulse pressure signal, PP, are taken over a historic window, with a duration T, exceeding the respiratory period.

Computation of ΔPP is complicated by the fact that PP is traditionally defined as the difference between consecutive systolic and

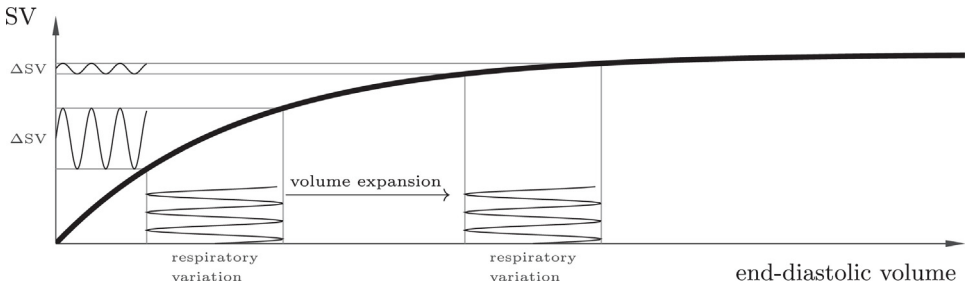

Fig. 2. Conceptual illustration of the Frank–Starling relationship between left end-diastolic volume and stroke volume. Volume expansion shifts the interval, across which intrathoracic pressure varies over each respiratory cycle, to the right, thereby decreasing ΔSV and consequently ΔPP.