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Identification of sources of low frequency variability of arterial blood pressure: cardiac output acts as a buffer and not as a source

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Abstract

Arterial blood pressure (ABP) short term variability is due to beat-by-beat fluctuations in cardiac output (CO) and total peripheral resistance (TPR), which have distinct effects at low and high frequencies. In particular, it was shown that CO is able to buffer TPR slow oscillations in the LF band, but it has not been addressed if CO can contribute to oscillations of ABP in this band. In this paper, we propose a model for the identification of ABP variability sources, in order to show evidence that CO fluctuations are not a source of ABP LF oscillations, but they only buffer ABP variability of vasomotor origin.

I. Introduction

Short term variability of arterial blood pressure (ABP) [1,2] is caused by oscillations of total peripheral resistance (TPR) and of cardiac output (CO); the latter combine the effects due to heart rate (HR) variability (HRV) and to stroke volume (SV) variability. The experimental work by O'Leary and Woodbury [2] showed that TPR is the main drive of ABP slow

oscillations (low frequency, LF ~ 0.1Hz), while CO is responsible for faster rhythms (high frequency, HF ~ 0.25Hz). They also discussed the role of CO in mediating LF oscillations of ABP observing an increase in ABP power at LF after CO beat-by-beat fluctuations were experimentally removed. This result led them to conclude that CO buffers LF oscillations of TPR, thus explaining the lower amplitude of the spectral power density of ABP at LF when CO spontaneously oscillated than in absence of CO variability.

Other studies have tried to investigate the problem of modeling the contribution of CO fluctuations [3] or more simply RR fluctuations [4] to ABP fluctuations with similar findings as to the buffering effects of RR and CO on the LF oscillations of ABP. Still, to our knowledge the issue of the potential contribution of CO to the genesis of ABP variability in the LF band has not been fully addressed and no conclusion has been drawn in this regard, although the aforementioned studies have clearly shown that CO and RR can buffer LF fluctuations of ABP.

In this paper, a model to identify the contribution of TPR and CO to ABP variability is proposed, with the aim of shedding light on their role in mediating both LF and HF oscillations of ABP. The analysis presented here shows the application of this system identification approach to spontaneous variability in awake, conscious dogs, at rest.

II. Methods

A. Experimental protocol

Six adult mongrel dogs (20–25 kg) of either gender were chronically instrumented with a fluid-filled catheter (Abbott Laboratories) in the abdominal aorta for beat-to-beat ABP recording and an ultrasonic flow probe (Transonic Systems) around the ascending aorta for beat-to-beat CO recording. After full recovery from surgeries necessary to implant the instrumentation, continuous recordings of ABP and CO took place with the dogs at rest and fully awake, for ~ 15 minutes. Data were recorded continuously from Gould 4600 series signal conditioners connected to a Dataq Instruments DI-710 a/d conversion device with Windaq software. Sampling frequency of the recordings was 300 Hz.

All procedures were reviewed and approved by the Wayne State University Animal Investigation Committee.

B. Pre-processing of the signals

Raw signals were preprocessed first by averaging their values over every cardiac cycle, in order to obtain a stepwise continuous signal which was then resampled to 2 Hz, with anti-aliasing filter at 1 Hz. Zero-mean time series of beat-to-beat ABP and CO fluctuations, normalized with respect to their mean values, were then derived (1):

$$ABP(t) = \frac{ABP_r(t) - \overline{ABP_r}}{\overline{ABP_r}} \quad CO(t) = \frac{CO_r(t) - \overline{CO_r}}{\overline{CO_r}} \quad (1)$$

In Eq. 1 ABP(t) and CO(t) are the zero-mean variability time series of ABP and CO resulting from preprocessing, $ABP_r(t)$ and $CO_r(t)$ are the time series obtained after downsampling to 2 Hz and $\overline{ABP_r}$ and $\overline{CO_r}$ and their respective mean values.

Segments approximately seven minutes long were selected from ABP and CO recordings. The ABP time series used in eq. (1) was the time series of diastolic arterial pressure (DAP).

C. Model of ABP variability

The structure chosen for system identification (eq. 2) was a linear moving average (MA) model with CO as the input, ABP as the output and n as the residual noise:

$$ABP(t) = \sum_{i=0}^n b_i \cdot CO(t-i) + n(t) \quad (2)$$

The identification was performed by means of WPCR analysis [5]. According to this modeling solution, and consistently with the physiologic identification and interpretation of the sources of ABP variability [2], the latter is separated into the contributions due to CO and those due to the fluctuations of TPR uncorrelated from CO, represented by noise n .

D. Spectra computation and statistical analysis

Auto-regressive (AR) spectral analysis of DAP time series and CO time series from the measurements and of noise time series resulting from system identification was carried out. Consistently with [2], the LF and HF bands were defined as follows: the LF band was confined to frequencies lower than 0.1 Hz, while the HF band was defined as the interval of frequencies between 0.1 and 0.5 Hz.

The contribution of CO to the overall variability of ABP was discussed computing the following ratios: ratio between LF power of estimated ABP and LF power of ABP, and ratio between HF power of estimated ABP and LF power of ABP. The statistical significance of the difference between these two ratios was tested by means of Student's unpaired t -test. The reason why an unpaired test was performed is that not all six dogs were considered for the analysis in the two bands. In particular, only three of the six dogs were considered for the computation of the ratio between estimated ABP and true ABP in the HF, and five out of six for the same ratio in the LF. The selection of the two subgroups of dogs was based on the visual recognition of clear peaks in each of the two bands: absent a clear peak in one of the bands, the dog would not be considered for the computation of the ratio in that band.

III. Results

Figure 1 shows a typical example of the prediction of ABP from CO and of the spectra of CO, measured ABP, estimated ABP and noise for one of the dogs. The spectra of this specific dog show peaks both in the low and high frequency bands of CO and ABP: for this reason, this animal was therefore included in the computation of the ratios between estimated and true ABP in both bands.

The ratio between the LF power of ABP estimated via the model and the LF power of measured ABP was 0.096 ± 0.027 , and the ratio between the HF power of estimated ABP and the HF of true ABP was 0.731 ± 0.056 . The unpaired t -test revealed that the two ratios significantly differ, with a p -value < 0.1 .

IV. Discussion

The results presented in this paper showed that CO was able to predict HF fluctuations of ABP, while it appeared from the spectral computation that CO can act as a buffer of LF variability of ABP, and as the only source of HF variability of ABP (TPR is characterized by slower dynamics, typical of the LF band). The low value obtained for the ratio between LF power of estimated ABP and LF power of true ABP also brought further evidence that CO does not explain LF ABP fluctuations. The fact that this value was not zero could be due to the estimation of noise in the measurements. On the other hand, less than 100% of HF power

of ABP variability was explained by CO, and this could be interpreted as an indication of the direct role of intrathoracic pressure variations and respiration on the arteries, that could explain what is not explained by CO. Also, a potential source of errors in our study is due to the linearity of the model, that cannot account for non linear effects and only accounts for the oscillations of TPR that are uncorrelated to CO fluctuations.

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Fig. 1.

Upper diagram: results of the identification of ABP (black) from CO (red), with noise (green) for one dog; lower diagram: spectra of CO (red), ABP from measurements (black), noise (green) and of ABP estimated from the model (blue) for the same dog. Series and spectra are both unitless because the series were normalized (eq. 1).