

Modeling arterial pulse pressure from heart rate during sympathetic activation

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INTRODUCTION

Arterial pulse pressure (PP) is associated with adverse cardiovascular outcomes and provides prognostic utility beyond that of mean arterial pressure [1]. In addition, PP is sensitive for the detection of central hypovolemia and PP variation has been introduced as a biomarker of fluid responsiveness to guide fluid treatment in clinical medicine. Continuous PP is not routinely available since PP tracking requires arterial cannulation. The duration between the systolic peak and post-dicrotic notch peak (SP-PDP) within a blood pressure wave (Figure 1) as measured from a plethysmographic arterial wave was shown to be related to PP [2]. We tested the hypothesis that not only SP-PDP duration is related to PP but heart rate (HR) as well.

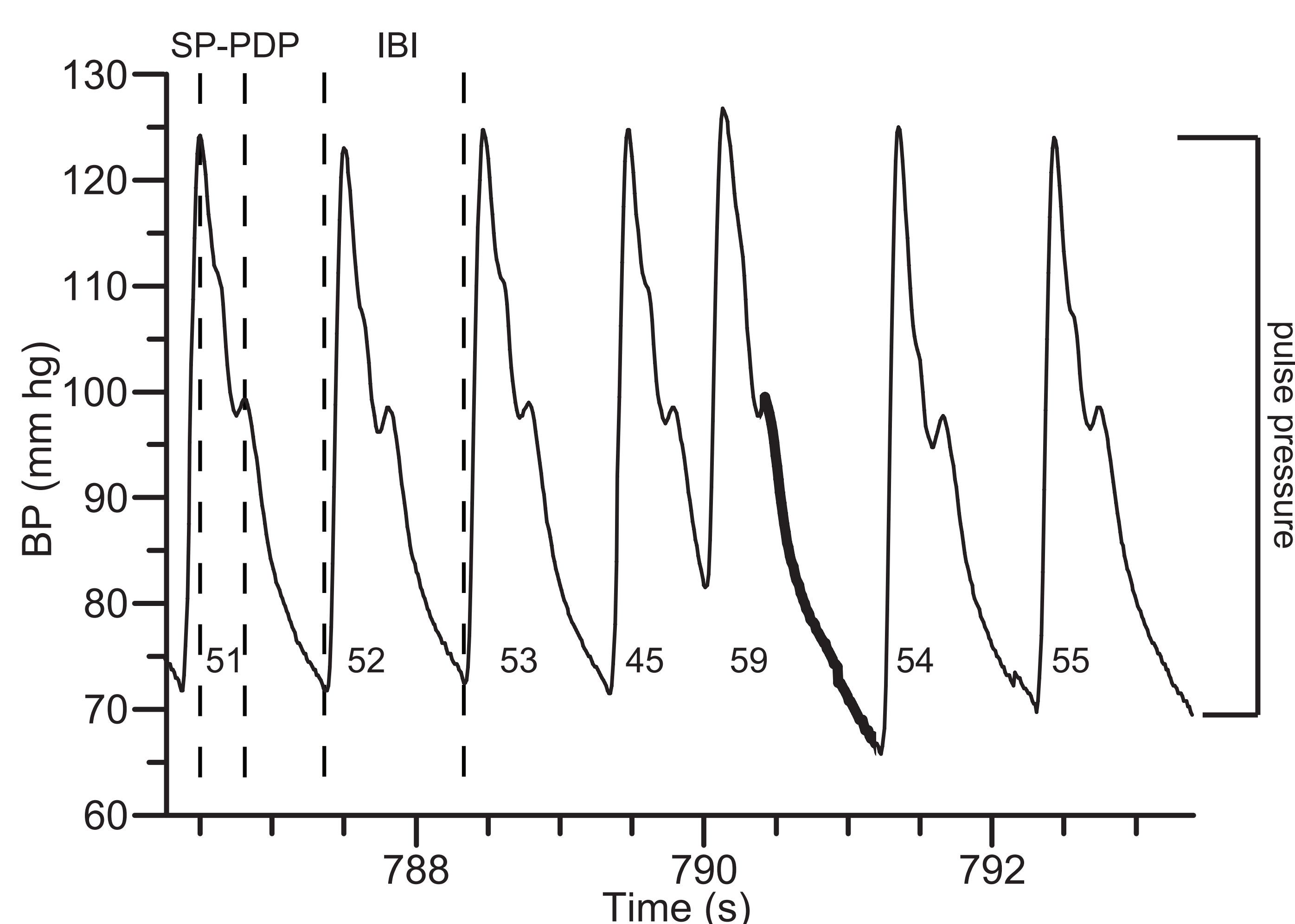


Figure 1. Blood pressure wavelines. With increased interbeat-interval (IBI) the pulse pressure increases during diastole. Pulse pressure is annotated for each beat at its root. SP-PDP: Systolic peak to post-dicrotic notch peak duration.

OBJECTIVES

- To model the relation between PP, SP-PDP duration and HR during hypovolemia
- Calculate the error of the created models
- Compare the predictive power of HR to that of the SP-PDP duration for estimating PP

MATERIALS & METHODS

Forty-four healthy volunteers (21 female; mean age 24 (SD 4) years; height 177 (10) cm and weight 73 (11) kg) were exposed to -50 mm Hg lower body negative pressure (LBNP) until the development of pre-syncope.

Continuous non-invasive finger BP (Nexfin) was acquired from which the duration of SP-PDP and HR were extracted. The data was averaged for each minute. We computed linear mixed effect (LME) models to describe the relations of fixed effects: HR, SP-PDP or both with PP. We included random effects to correct for each subject and either HR or SP-PDP, depending on whether HR or SP-PDP was being used as a fixed effect. The available data was split into two groups. Modeling PP was done in two steps: 1. using half the data to develop the models and the remaining data to compute the errors, 2. then repeating the process with reversed groups. This resulted in two sets of equations for PP.

RESULTS

Three subjects were excluded due to failure to detect the peaks in the BP wave. Group A consisted of data from subjects 1 through 21 and group B contained data from subjects 22 through 41. 37 Subjects experienced pre-syncope during LBNP. Mean time until pre-syncope was 837s; SD 368s and ranged from 2.5 to 26 min; 7 subjects completed 30 minutes without experiencing pre-syncope.

Equations for the LME models for fixed effects: HR, SP-PDP, or both for group A are:

$$PP = 67.3 - 0.35 \cdot HR \quad (1)$$

$$PP = -23.4 + 0.23 \cdot SP-PDP \quad (2)$$

$$PP = 8.4 + (0.17 \cdot SP-PDP) - (0.18 \cdot HR) \quad (3)$$

and for group B:

$$PP = 70.0 - 0.37 \cdot HR \quad (4)$$

$$PP = -13.9 + 0.20 \cdot SP-PDP \quad (5)$$

$$PP = 44.0 + (0.07 \cdot SP-PDP) - (0.29 \cdot HR) \quad (6)$$

Model residuals were 9.0; 11.6 and 6.4 for equations 1 through 3, and 10.3; 11.5 and 9.7 for equations 4 through 6. An example of a subject on which models were tested is shown in Figure 2. Median errors for the entire group computed as the difference between measured PP and modeled PP are shown in Table 1.

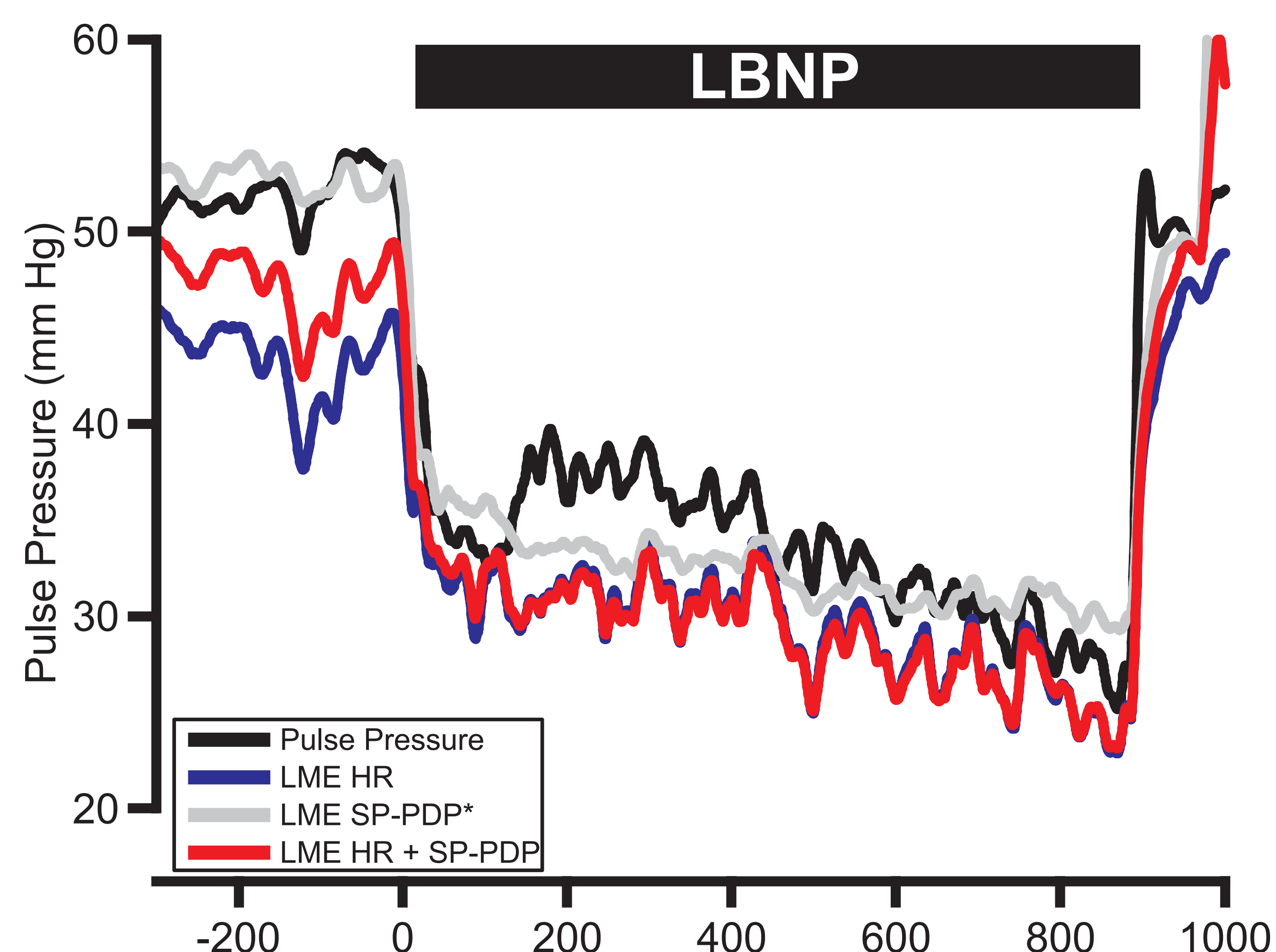


Figure 2. Model results. three linear mixed effect (LME) model estimations of pulse pressure during LBNP in a representative subject. Best fitted model for this subject is annotated with an asterisk.

Table 1. Median [25% 75%] errors of the estimated pulse pressure per modeled parameter in mm Hg.

Error during	SP-PDP model	HR model	Combined model
Baseline	8.6 [4.3; 14.3]	6.0 [3.9; 9.8]	7.7 [5.4; 12.2]
LBNP	5.2 [3.3; 8.9]	4.9 [3.8; 7.6]	4.9 [3.7; 8.6]

CONCLUSION

LME models based on heart rate provide information about arterial pulse pressure under conditions of sympathetic activation by LBNP, and under those conditions heart rate predicts pulse pressure.

References

1. Selvaraj, S. (2016). *J Am Coll Cardiol*, 67(4), doi: 10.1016/j.jacc.2015.10.084
2. Baruch, M. C. (2011). *Nonlinear Biomed Phys*, 5(1), 1. doi: 10.1186/1753-4631-5-1

