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Modulation of pulse travel and blood flow during cuff inflation- An experimental case study

Laura I. Bogatu, Simona Turco, Massimo Mischi, Lars Schmitt, Pierre Woerlee, Erik Bresch, Gerrit J. Noordergraaf, Igor Paulussen, Arthur Bouwman, Erik H.H.M. Korsten, and Jens Muehlsteff

***Abstract* - The blood pressure (BP) cuff can be used to modulate blood flow and propagation of pressure pulse along the artery. In our previous work, we researched methods to adapt cuff modulation techniques for pulse transit time vs. BP calibration and for measurement of other hemodynamic indices of potential interest to critical care, such as arterial compliance. A model characterized the response of the vasculature located directly under the cuff, but assumed that no significant changes occur in the distal vasculature.**

This study has been tailored to gain insights into the response of distal BP and pulse transit time to cuff inflation. Invasive BP data collected downstream from the cuff demonstrates that highly dynamic processes occur in the distal arm during cuff inflation. Mean arterial pressure increases in the distal artery by up to 20 mmHg, leading to a decrease in pulse transit time of up to 20 ms. *Clinical Relevance*: Such significant changes need to be taken into account in order to improve non-invasive BP estimations and to enable inference of other hemodynamic parameters from vasculature response to cuff inflation. A simple model is developed in order to reproduce the observed behaviors. The lumped-parameter model demonstrates opportunities for cuff modulation measurements which can reveal information on parameters such as systemic resistance, distal arterial, venous compliances and artery-vein interaction.

I. Introduction

The BP cuff is a standard device used in critical care and it offers ample possibilities of modulating the blood flow and travel of pulse along the artery. It is interesting to explore potential opportunities for improved non-invasive BP estimation and inference of other hemodynamic parameters from response of the circulation to occlusion-based perturbations. In our previous work [1, 2], we characterized cuff-induced perturbation effects to determine how factors such as BP [2] or arterial compliance [1] are expressed in vasculature responses to cuff inflations. We focused on a measurement setup involving ECG, a cuff placed over the brachial artery and PPG placed at finger site downstream from the cuff. The ECG-PPG combination was used to measure delays in pulse arrival time (PAT) caused by the cuff inflation. This setup enabled study and modelling of the localized change in transmural pressure over the brachial arterial wall and its impact on pressure pulse travel. As cuff inflates, the arterial transmural pressure over the length of the cuff is decreased, thus increasing the time it takes for the pulse to propagate down the brachial artery. Our earlier work consisted primarily of measurements performed non-invasively, complemented by computer simulations and a model which characterized brachial vasculature, but excluded effects occurring in the distal limb vasculature (under the assumption that distal BP and distal pulse transit time do not change significantly throughout cuff inflation).

In this study we present clinical data acquired during OR procedures which shows that highly dynamic processes are occurring in the distal arm during cuff inflation. It appears that distal arterial blood pressure increases significantly, which impacts pulse transit time. This process needs to be taken into account in the methods presented in [1, 2] in order to correctly interpret the arterial BP and PAT response to cuff inflation.

Therefore, our initial brachial artery model [1, 2] is complemented by a distal arm circulation model based on acquired experimental data. The observed dynamic processes reveal opportunities for measuring non-standard but highly relevant clinical parameters related to systemic resistance, arterial compliance and arterial-venous interaction. Experimental data obtained during a case study and the model for the distal arm are presented below.

II. Materials and Methods

A. Measurement procedure

A sensor arrangement consisting of ECG, brachial BP cuff, intra-arterial line (radial ABP) and finger PPG (Fig. 1) is used to collect data from an anesthetized and mechanically ventilated patient undergoing a non-cardiac surgery. All sensors are standard devices common in clinical practice [3]: Philips Comfort Care Adult Cuff [4], Edwards Lifesciences TruWave disposable pressure transducer (Edwards Lifesciences, Irvine, CA) and PPG model Philips M1191B

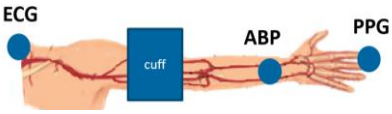

Figure 1. Sensor arrangement.

L. I. Bogatu is with Philips Research, Eindhoven, Netherlands, 5656AE and Eindhoven University of Technology, Netherlands, 5612AZ (laura.bogatu@philips.com).

S. Turco, M. Mischi and P. Woerlee are with Eindhoven University of Technology, Netherlands, 5612AZ. (s.turco@tue.nl, m.mischi@tue.nl).

J. Muehlsteff, L. Schmitt, E. Bresch are with Philips Research, Eindhoven, Netherlands, 5656AE (jens.muehlsteff@philips.com, lars.schmitt@philips.com, erik.bresch@philips.com).

G. J. Noordergraaf is with Department of Anesthesiology, Resuscitation & Pain Management, Elisabeth-TweeSteden Hospital, Tilburg, 5022GC, Netherlands (g.noordergraaf@etz.nl).

I. Paulussen is with Philips Research, Eindhoven, Netherlands, 5656AE and Elisabeth-TweeSteden Hospital, Tilburg, 5022GC, Netherlands. (igor.paulussen@philips.com).

A. Bouwman, E.H.H.M. Korsten are with Catharina Ziekenhuis, Eindhoven, Netherlands,5623EJ (arthur.bouwman@catharinaziekenhuis.nl, erik.korsten@catharinaziekenhuis.nl).

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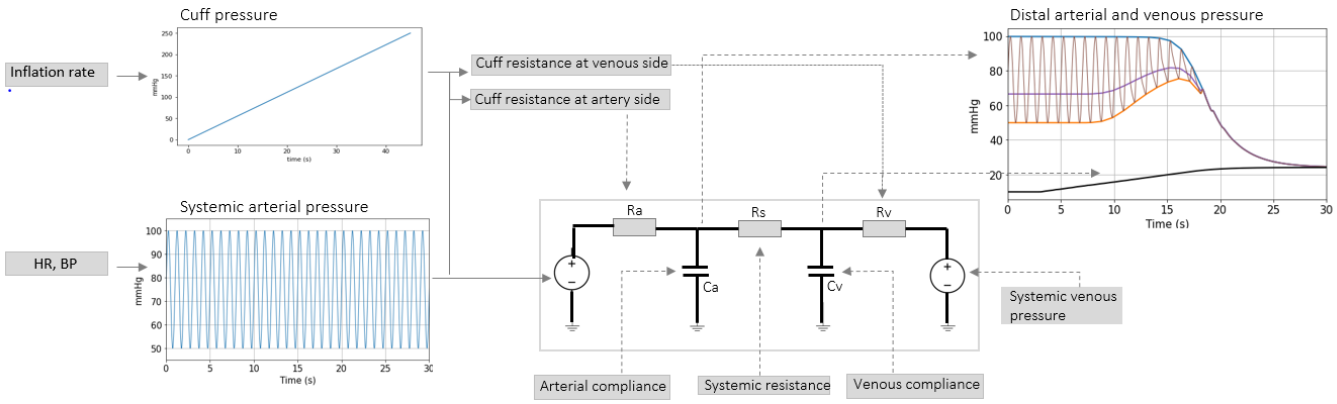

Figure 2. Overview of arm circulation lumped parameter model.

[4]. The data were recorded using a Philips MP50 patient monitor [4] and custom data logging software. The ECG was sampled at 500Hz, and the ABP, PPG, and cuff pressure were sampled at 125Hz. The data collection was approved by the MEC-U ethical committee (St. Antonius Ziekenhuis, Koekoekslaan 1, 3430 EM Nieuwegein, NL. Approval W19.046) and the data was collected at the Elisabeth-TweeSteden Ziekenhuis, Tilburg, NL. Written informed consent was obtained from the patient. As part of this example case study, forty-eight cuff inflations were performed at arbitrary intervals across 7 hours on the anesthetized patient. Cuff pressure, ECG, ABP, and PPG signals are recorded simultaneously and analyzed to characterize cuff inflation effects on the distal vasculature. Thirteen inflations are removed from analysis due to artifacts caused for example by flushing of the arterial line. Simultaneously recorded ECG and PPG are used to compute change in PTT due to cuff inflation over two vascular segments:

1. **Heart to finger site**: $\Delta PAT(CuffPressure)$; Computed as the change in delay between the ECG R-peak signal and the PPG waveform foot of the as cuff pressure increases.
2. **Radial to finger site**: $\Delta PTT_{ABP-PPG}(CuffPressure)$ Computed as the change in delay between the ABP waveform foot of the and the PPG waveform foot as cuff pressure increases.

B. Model

A model is used to obtain an understanding of the factors that influence the observed dynamic behavior occurring in the distal arm during cuff inflation. The model also gives insights on hemodynamic parameters that can be derived from analyzing the cuff-induced changes in artery pressure.

The model presented by Seagar et al in [5] was adapted for our experimental situation. In [5], the response of limb circulation to a constant vein-occluding cuff pressure was analyzed via a parametric model of the limb circulation; the model parameters were representative of clinically relevant indices related to thrombosis. The model however included parameters such as systemic resistance and blood vessel compliance, which are also relevant to hemodynamic monitoring, as stated by the author:

"... a particularly appropriate application for the model is to use changes in the model parameters to monitor circulatory changes of the limb, such as those, for instance, that may occur during clinical anaesthesia." - Seagar et al

The venous thrombosis model in [5] is therefore adjusted to characterize our invasive BP data. Figure 2 shows an overview of the distal arm model. Systemic arterial pressure, cuff pressure and several parameter values are used as inputs to the model. The model generates distal arterial pressure and distal venous pressure. R_a represents resistance to blood flow over the brachial artery. Its value is estimated via the Poiseuille equation (Eq. 1),

$$R_a(P_{tm}) = \frac{8\eta L}{\pi\, r(P_{tm})^4}, \quad (1)$$

where η is viscosity of blood, L is length of cuff and r is brachial artery radius. P_{tm} is transmural pressure across the arterial wall (Eq. 2):

$$P_{tm} = P_{art} - P_{cuff}, \quad (2)$$

where P_{art} is arterial pressure under the cuff. Absolute value of radius r is obtained from ultrasound measurement of brachial artery performed by Bank et al in [6]. As cuff pressure P_{cuff} increases, the artery radius r decreases as function of transmural pressure. Eq. 3 describes the process of arterial collapse mechanics [7]:

$$A(P_{tm}) = d\,\frac{\ln(a\,Ptm + b)}{1 + e^{-c\,Ptm}}, \quad (3)$$

where A is artery area, a, b, c, d are parameters describing arterial collapse. R_{v} represents resistance to blood flow over the brachial vein and it can also be defined via Eq. 1. Evidence on vein collapse behavior ([8, 9], including MRI images collected as part of our previous studies [10, 11]) confirm that the vein collapses at approximately -10 mmHg transmural pressure. The physiologic venous pressure range is between 5 mmHg and 15 mmHg [8], from which it can be concluded that the vein collapses in the very first part of the inflation (before cuff reaches 30 mmHg). R_{s} represents peripheral resistance of the arm. Physiological ranges for R_{s} in the arm were obtained from [12], where values of 106 $mmHg\ s\ ml^{-1}$ are reported. C_{a} and C_{v} represent arterial and venous compliance respectively. In [12] arterial compliance C_a in the arm is approximated in the order of 0.03 $ml\ mmHg^{-1}$. C_{v} is approximated as about 30 times larger than C_{a} [13].

III. Results

A. Measurements

The cuff inflation effects on distal arm arterial pressure are shown in Fig. 3. In Fig. 3.A the cuff pressure during inflation is shown. Fig. 3.B shows the radial arterial pressure. Effects

of cuff inflation on the distal limb lead to MAP change of up to 20 mmHg. These changes were neglected in our previous study of pulse wave travel along the arm [1, 2]. In addition to this, different phases of the inflation process identified in the BP signal carry physiological meaning. Venous collapse, partial arterial collapse and subsequent buildup of blood in the limb are reflected in the signal segment where gradual increase in diastolic pressure (P_{dia}) and slight decrease in systolic pressure (P_{sys}) occur. After the moment of complete arterial collapse, arterial pressure decreases exponentially towards an equilibrium value. This exponential decay signal segment is not affected by heart activity, breathing artifacts, or blood flow in/out of the limb – it is only influenced by arterial-venous interaction characterized by C_{a}, C_{v} and R_{s} (Fig. 2). The dynamic effects are also reflected in the propagation of pressure pulse in the distal arm. Previous models characterizing travel of pressure pulse [1, 2] assumed that no significant changes in PTT occur in the distal arm during cuff inflation. Our data however gives insights on the correctness of this assumption. Fig. 3.E shows a decrease in $\Delta PTT_{ABP-PPG}$ of about 10 ms over the radial site to finger length, therefore the drop in ΔPTT over the entire length of the distal arm is calculated to be approximately 20ms.

To summarize the effects across all 35 inflations, the BP change per inflation is condensed as shown in Fig.4: for each inflation the maximum increase in diastolic pressure, the maximum decrease in systolic pressure and the maximum increase in mean pressure (MAP) are calculated. The results are shown in Fig. 5. On average across all inflations, the diastolic pressure increases by 25 mmHg, the systolic pressure decreases 29 mmHg and the mean arterial pressure increases by 16 mmHg.

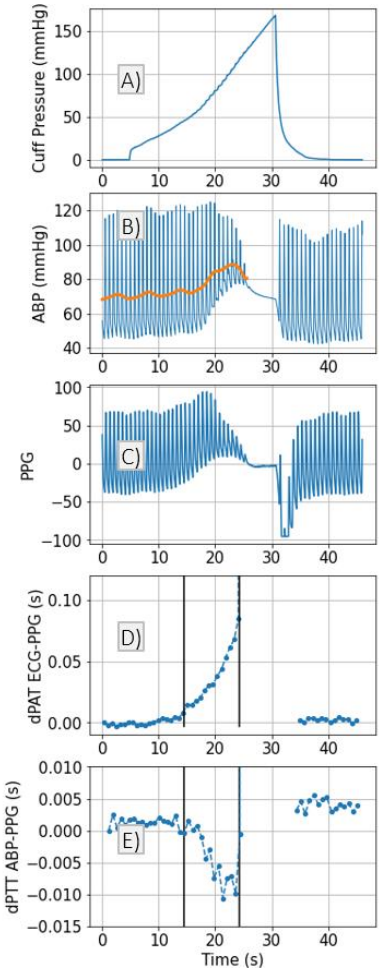

Figure 3. Example measurement.

We quantify the decrease in radial-finger ΔPTT (Fig. 6A) for all 35 cuff inflations. On average across all inflations, the decrease in ΔPTT is 10 ms. As shown in Fig. 1, the ΔPTT is measured between radial to finger site. Therefore, it can be concluded that the ΔPTT change over the entire distal arm (from distal edge of the cuff to the finger) is approximately 20 ms (Fig. 1). Figure 6B shows the cuff pressure at which the minimum value of ΔPTT occurs for all 35 cuff inflations. It is found that the minimum value of ΔPTT occurs when cuff approaches the systemic systolic pressure value. From this, it can be deduced that the cuff inflation (and resulting distal BP increase) systematically lead to a drop in ΔPTT. The effect cannot be attributed to other factors.

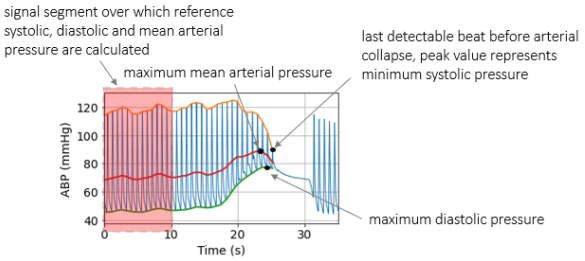

Figure 4. Illustration showing reference P_{sys}, P_{dia}, MAP values and maximum MAP, maximum Pdia, minimum Psys.

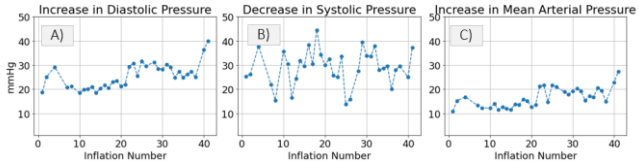

Figure 5. Absolute difference between reference BP values and BP values altered by cuff inflation. A) Maximum increase in P_{dia} per inflation. B) Maximum decrease in P_{sys} per inflation. C) Maximum increase in MAP per inflation.

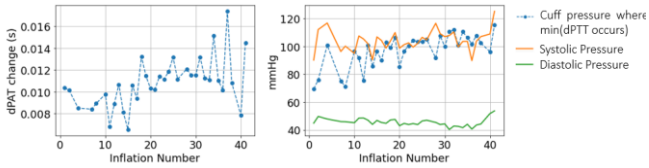

Figure 6. A) Maximum drop in ΔPTT per inflation. B) P_{cuff} at which maximum drop in ΔPTT occurs, P_{sys} and P_{dia} systemic values per inflation.

B. Model

The model shown in Fig. 2 is used to simulate distal arterial BP change with respect to cuff inflation. Systemic arterial diastolic pressure is set to 50 mmHg and systemic arterial systolic pressure is set to 100 mmHg. The chosen values are similar to those observed in the patient data (Fig. 3). Heart rate is set to 1 Hz and total cuff inflation duration is set to 25 s. The model parameters are chosen based on values reported

in literature: systemic venous pressure is set to 10 mmHg, R_s is 106 mmHgs/ml, C_a is 0.03 ml/mmHg, C_v is 0.9 ml/mmHg. Figure 2 (upper right plot) shows distal arterial and venous pressures outputted by the model. The simulation results show the same features and trend in the diastolic, mean and systolic radial artery pressure. Therefore, the model can be used to represent the effects observed in the patient data (Fig. 3.B).

IV. Discussion

The data shown in Fig. 3 reveal interesting responses of the arterial blood pressure to cuff induced blood flow perturbation. In our previous characterization of this phenomenon [1, 2] we assumed that the distal vasculature is not significantly affected by cuff inflations; our model characterized transmural pressure changes occurring directly under the cuff, while not accounting for transmural pressure changes occurring in the distal arm.

Figure 3.B shows the extent to which distal BP is altered due to cuff inflation: P_{dia} (Fig 3.B) increases by up to 30 mmHg, MAP increases by up to 20 mmHg. This effect is also evident in the 20 ms drop in distal PTT (Fig. 3.E). The cuff-induced effects on distal vasculature are systematic and are seen across all 35 cuff inflations (Fig 5, Fig. 6): on average, P_{dia} increases by 25 mmHg and MAP increases by 16 mmHg in the example patient.

Simulations conducted via the model (Fig. 2) with parameters within ranges close to values reported in literature mimic effects observed in the patient data. Five cuff inflation stages which are identified based on the patient data (Fig. 3.B) are also present in the model output (Fig. 2 upper right plot):

1. Cuff pressure value is below systemic venous pressure: arm circulation remains unaltered.
2. Cuff pressure increases beyond systemic venous pressure: vein collapses, blood flow out of the limb is stopped and buildup of blood begins to occur in the limb via the artery; artery pressure is not visibly altered at this stage.
3. Cuff pressure increases beyond systemic diastolic pressure leading to increase in distal diastolic pressure.
4. Cuff pressure approaches systemic systolic pressure: minimal amount of blood flows into the limb with each beat, decrease in the distal systolic pressure is observed.
5. Eventually blood flow is stopped - arterial and venous pressures tend towards an equilibrium value.

The model closely mimics all 5 stages of the cuff inflation process, meaning that the model can be used to represent changes in distal arterial pressure during cuff inflation and that physiological meaning can be attributed to the model parameters. Future work will tackle inference modalities aimed at estimating such parameters from patient data. The presented measurement strategy, data and model-based analysis are aimed at demonstrating that the response of the circulation to cuff-induced perturbations can contain extensive information regarding hemodynamic status, including: artery-vein interaction, arterial compliance, systemic resistance. Changes in distal blood pressure may affect the pressure envelope obtained in the oscillometric method. Furthermore, the changes in distal blood pressure we observed are important for the calibration procedure of PAT based continuous non-invasive BP measurements [2, 14]. Further development of measurement strategies for assessing circulation response to occlusion-based perturbations can potentially benefit critical care practice [15] and provide a potential tool for study of vascular pathological conditions.

V. Conclusion

This study improved our understanding of vasculature response to occlusion-based perturbations. Initial characterizations of cuff-induced perturbations assumed that changes of the distal vasculature were insignificant. However, our new experimental evidence shows that highly dynamic processes occur in the distal vasculature during cuff inflation. Such processes need to be modelled and taken into account for correct interpretation of the vasculature response to occlusion-based perturbations in hemodynamic monitoring.

VI. References

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