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Clinical paper

The influence of nonlinear intra-thoracic vascular behaviour and compression characteristics on cardiac output during CPR☆

Yvette Koeken[a,b], Paul Aelen[a,b,*], Gerrit J. Noordergraaf[b], Igor Paulussen[a,b], Pierre Woerlee[a], Abraham Noordergraaf[c]

[a] Biomedical Sensor Systems, Philips Research, Eindhoven, The Netherland
[b] Dept of Anaesthesiology & Resuscitation, St. Elisabeth Hospital, Tilburg, Hilvarenbeekseweg 60, 5022 GC Tilburg (NL), The Netherland
[c] University of Pennsylvania, Philadelphia, PA 19104 USA

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ABSTRACT

Clinical observations suggest that the assumption of a linear relationship between chest compression pressure and cardiac output may be oversimplified. More complex behaviour may occur when the transmural pressure is large, changing the compliances and resistances in the intra-thoracic vasculature. A fundamental understanding of these compression induced phenomena is required for improving CPR.

An extensively used, lumped element computer model (model I) of the circulation was upgraded and refined to include the intrathoracic vasculature (model II). After validation, model II was extended by adding variable compliances and resistances (model III) to the vascular structures. Successively, ranges of compression pressures, frequencies, duty cycles and compression pulse shapes were applied while controlling all other parameters. Cardiac output was then compared.

The nonlinearities in compliance and resistance become important, limiting factors in cardiac output, starting in our experimental series at 70 mmHg peak compression pressure, and increasing with higher pressures. This effect is reproducible for sinusoidal and trapezoidal compression forms, resulting in lower cardiac output in all experiments at high compression pressures. Duty cycle and wait time are key parameters for cardiac output.

Our data strongly indicate that vascular compliance, especially the ability of vessels to collapse (and potentially the cardiac chambers), can be a central factor in the limited output generated by chest compressions. Just pushing 'harder' or 'faster' is not always better, as an 'optimal' force and frequency may exist. Overly forceful compression can limit blood flow by restricting filling or depleting volume in the cardiac chambers and central great vessels.

1. Introduction

Current recommendations for cardiopulmonary resuscitation (CPR) are to compress "hard and fast" and then to release fully.[1,2] Focus on the quality of manual compressions[3] and the introduction of automated compression devices have rekindled interest in compression effects on the thoracic vasculature.[4,5] Compressions distort the thoracic wall and the shapes of underlying structures.[6] This can alter the compliance and resistance of vessels,[7] as a function of their transmural pressure (TMP), defined as $TMP = P_{internal} - P_{external}$. High intrathoracic pressures may even cause vascular closure, undermining chances for effective, life supporting, flow.[8]

Chest compressions pump blood forward. However, this pump amplifies traditional vascular parameters into explicitly time-varying parameters. As a consequence, the conduct of vessels becomes nonlinear, which can disturb intra-vascular blood volume distribution.[5] While animal models[9] and human observational studies[10] have been used to evaluate CPR induced flow, little has been done to study the occurrence and consequences of variable vascular compliances outside of physiology.[11] Computer modeling tools are well suited to study these complex effects in both physiology[12–14] and CPR situations.[15] Anecdotal clinical observations of an apparently optimal, case dependent, compression force suggest that gains in flow might be achieved by further understanding these effects.[5]

Babbs et al.[16,17] developed an electrical analog, lumped element circulatory model to investigate compression methods including the duty cycle. This simple electrical model was adapted for computer modeling[18] and modified further to investigate optimal

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* Corresponding author at: Biomedical Sensor Systems, Philips Research, Eindhoven, The Netherland.
E-mail address: paul.aelen@philips.com (P. Aelen).

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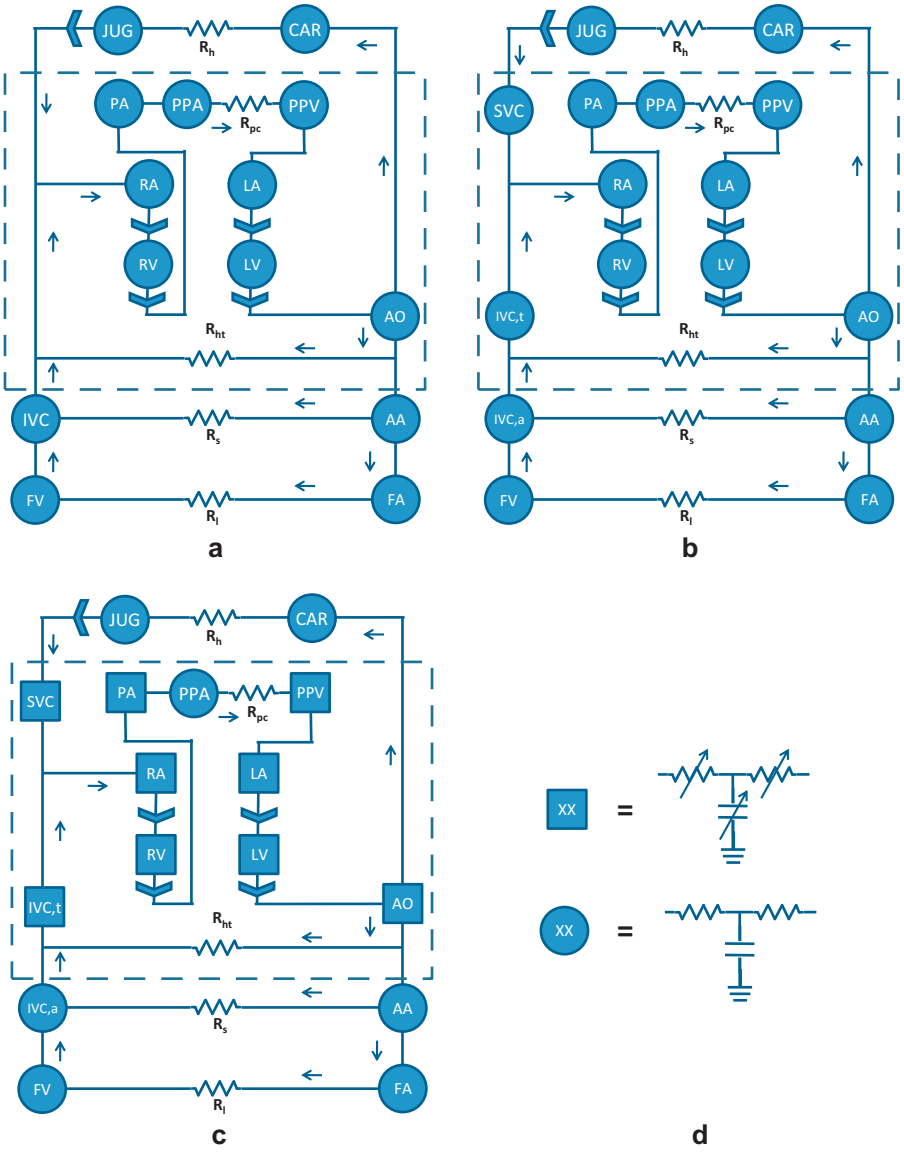

Fig. 1. Models I–III. Schematic overview of models I (a), II (b) and III (c). Circles represent constant segments, squares represent collapsible segments. Each element consists of 2 resistances, each of half the value in Appendix A, and a capacitor (d). Major vascular resistances, the cardiac, peripheral and Niemanns' valves are included. The arrows represent the direction of positive flow. See Section 2 for further explanation. The abbreviation of each element is explained in Table 1.

compression waveforms.[19] However, these models described cardiac output as a first-order function (i.e. increasing linearly, as a straight line, with compression force). This seems to be an appropriate approximation, if limited to lower compression pressures. For higher compression pressures, nonlinear effects have to be incorporated. In early models, volume conservation, avoidance of negative volumes, and variable compliances or resistances were either not present or limited in their scope.[16,18,19] These aspects in circulatory models have been touched on previously. [20] The impact of vascular collapse on flows due to high transmural pressures induced by CPR, have only recently been considered.[5,22]

To investigate the effects of variable compliances, resistances and volume redistribution caused by compressions, we reintroduce an extensively reported description of the circulation.[17–19] Our aim is to provide insight into the occurrence and impact of vascular changes using a small, lumped element, model with nonlinear TMP–volume relationships and clinically relevant compression force, frequency, and duty cycles ranges.

2. Materials and methods

Meador adapted Babbs et al.'s[17,18] electrical model for computer use, using SPICE 2G.6 (Simulation Program with Integrated Circuit Emphasis), a general purpose analog circuit simulator. This program uses building blocks with resistors and capacitors, inductors, etc., called lumped elements, to fill equations which can then be solved.[18] The format used by Meador,[18] and the elements described by Babbs[19] were used as reference values (Fig. 1a). This combination was programmed in LT-SPICE IV and became our model I.[21]

The venae cavae in Babbs' model were then redefined to explicit intra- and extra thoracic positions, generating model II (Fig. 1b). In models I and II, the volume in an element is modeled as a charge on a normal (i.e. constant) electric capacitor. This, as with Meador and Babbs, allows elements (vessels) to contain negative "blood volumes" since capacitors can contain negative charges and thereby "empty beyond zero".[18,19] To confirm that these changes had not altered their basic conduct, models I and II were compared under specific experimental conditions and with data from the reference