

A LUMPED PARAMETER MODEL FOR THE STUDY OF THE VENOUS RETURN IN THE TOTAL CAVO-PULMONARY CONNECTION

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INTRODUCTION

The Total Cavo-Pulmonary Connection (TCPC) is an important and complex surgical intervention that aims to heal a broad spectrum of congenital diseases by radically modifying the original setting of the circulatory tree. These changes have side-effects on various cardiovascular variables throughout the circuit and on the post-surgical physiology overall.

BACKGROUND

The use of mathematical models of the blood circulation is of help in the prediction of variables, which are difficult to evaluate clinically. Moreover, it provides the means to study the way in which changes in one or more parameters affect the overall circulatory physiology. A mathematical model of the short/mid term post-operative paediatric circulation may help analyse and make decisions about the use of this therapeutic strategy.

METHODS

A lumped parameter model (LPM) of the healthy paediatric blood circulation has been developed based on a full range of characteristic constants that describe the behaviour of heart, pulmonary and systemic circulations, in particular the venous return.

The parameter identification was carried out by modifying literature values for a healthy adult [1] through an innovative *differential scaling* technique based on different scaling coefficients for the head, trunk and legs. The scaling equations are consistent with the literature [2]. The additional parameters were adjusted to fit the measured physiological tracings. Blocks describing the major veins include terms for the resistance, compliance, inertance, for the venous valves, and take into account vein collapsibility. The model also considers the effects of respiration and of the moving diaphragm (Figure 2 - P_{IT}).

The LMP for the healthy child was modified in order to describe the post-operative status. A model of the univentricular heart mimics the chronic alterations in the performance of the human heart with a single functional ventricle. The TCPC model also describes the long-term adaptation following the intervention by a non-active

representation of the baroflex control [3]. This appears to be essential, as the predictions are not satisfactory in its absence. The model obtained was tested against clinical data from the literature [4, 5].

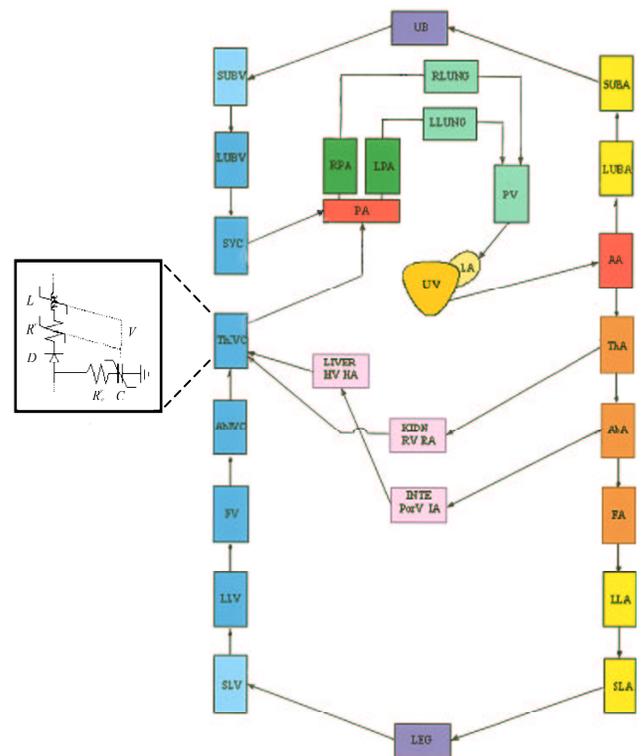


Figure 1. LPM net showing the blocks that represent the various compartments of the TCPC circulation (in particular the typical venous block)

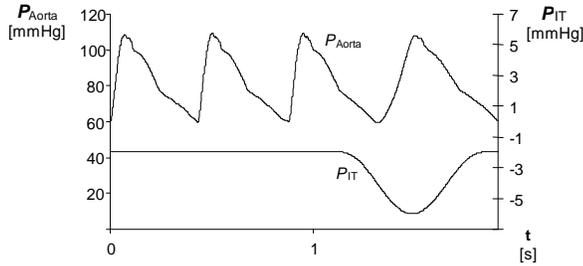


Figure 2. Aortic (P_{Aorta}) and intrathoracic (P_{IT}) pressures

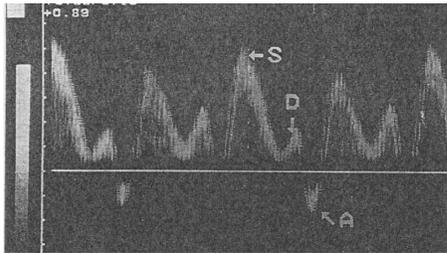
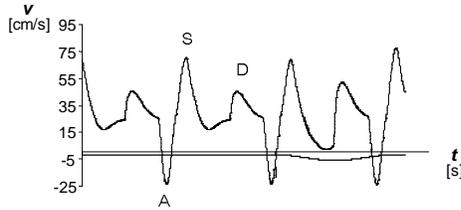


Figure 3. Predicted velocity in the SVC of a healthy child (top) and Echo-Doppler recording of the velocity in the SVC of a healthy child (bottom).

RESULTS

The models give accurate predictions of the tracings and absolute values of the time variables in both the healthy and the post-operative state. A quantitative example is given in Figures 3 and 4 (P_{IT} shown).

Simulations of post-operative scenarios were attempted. The effect of a paralysed diaphragm is a slight decrease in CO of about 1%, and a significant reduction of the respiratory peaks in IVC and SVC flow velocity tracings (Figure 5). This may be a clue in Doppler-based diagnosis of diaphragm disorders. An increase in pulmonary resistances (PAR) produces an increase in venous pressure and a decrease in lung perfusion, CO and systemic pressure. If the increase is due to the occlusion of only one pulmonary artery, the mean

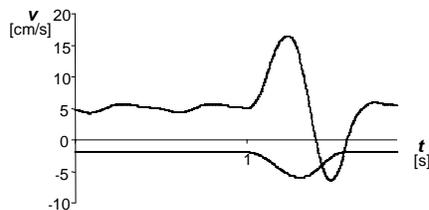


Figure 4. Hepatic vein blood velocity in a TCPC patient

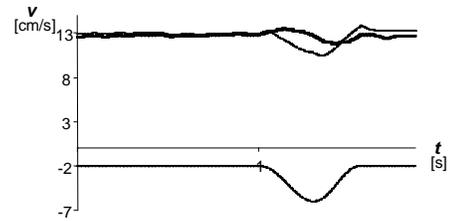


Figure 5. Predicted velocity in the SVC of a TCPC patient with normal (thin) and paralysed (bold) diaphragm

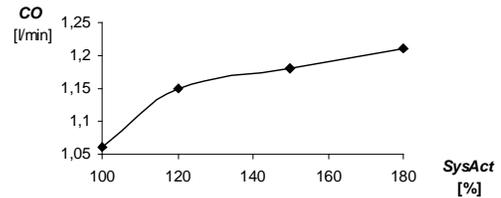


Figure 6. Increase in CO due to exercise in a TCPC patient. SystAct is a percent measure of exercise severity with respect to pre-exercise status (100%)

pulmonary pressure remains practically constant, as well as the mean aortic pressure, total lung perfusion and CO .

The effect of exercise (modelled by increasing cardiac inotropy and decreasing limb resistances) results in a milder rise in CO for healthy subjects (Figure 6). The increase in arterial mean pressure is not great, as it also happens in the healthy. The increase in venous pressure is slight.

CONCLUSIONS AND FURTHER DEVELOPMENTS

From reliable LMP models, by introducing minor parameter changes suggestions can be made on different medical or surgical procedures in the repair of congenital cardiac malformations.

A model of the oxygen consumption and active short-term baroflex regulation could be added in the future.

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