A model to simulate the haemodynamic effects of right heart pulsatile flow after modified Fontan procedure

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Abstract

The effect of pulsatile pulmonary flow after the modified Fontan procedure was examined in a model that simulated the right heart. An inlet overflow tank (preload), axial pulsatile pump, Wind-Kessel model (afterload), and an outlet overflow tank were connected in series. The standard conditions were flow 2·00 l/min with 12 mm Hg preload pressure, 3·0 Wood units resistance, and an outlet overflow tank pressure at 6 mm Hg. The pump rate was set at 80 beats/min. The simulated pulmonary arterial pressure and pulmonary flow waves produced by this model closely resembled those obtained from patients who had undergone the modified Fontan procedure. All variables except the preload were fixed and changes in pulmonary flow were examined at preload pressures of 8, 12, 15, and 17 mm Hg. As the peak pulmonary arterial pressure increased so did pulmonary flow, until it was greater than during the non-pulsatile state. Because the afterload of this model was fixed, this result suggests that there was a concomitant decrease in resistance.

This model indicates that pulsatile pulmonary blood flow is likely to have a beneficial effect on the pulmonary circulation after the modified Fontan procedure.

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After the modified Fontan procedure the right atrial and pulmonary arterial pressure waves, which are almost entirely a function of right atrial contraction and relaxation, have a characteristic configuration.[1–3] It has not known however, whether the pulsatile blood flow from the right heart has any effect on the pulmonary circulation. We studied this question in a model we constructed to simulate the right heart after the modified Fontan procedure.

Method

Figure 1 shows the model simulating the right heart after the modified Fontan procedure. The inlet overflow tank (preload), axial pulsatile pump, Wind-Kessel model (afterload), and the outlet overflow tank (left atrium) were connected in series and filled with water at 37°C. A rise in the level of the water in the inlet overflow tank was prevented by an open cylinder. The height of this cylinder determined the preload. In this model the axial pulsatile pump had no prosthetic valves in either the inlet or outlet parts to better reproduce conditions present in the right atrium after the modified Fontan procedure. The Wind-Kessel model, which provided the afterload, was constructed of a compliance chamber and a servocontrolled valve. The compliance chamber was filled with air to one third of its total volume.[4] The standard conditions were established in a non-pulsatile state: flow was 2·00 l/min with a 12 mm Hg preload, the afterload was 3·0 Wood units, and the outlet overflow tank pressure was 6 mm Hg. Afterload and outlet overflow tank pressures were fixed throughout the experiment. The pump was set at a rate of 80 beats/min. Under standard conditions this model produced pulmonary arterial pressures and pulmonary flow waves that resembled those recorded in a patient who had undergone the modified Fontan procedure (fig 2). The driving pressure, systolic fraction, and the vacuum were adjusted (while the standard conditions were maintained) to create a pulsatile flow that produced graduated increases peak pulmonary arterial pressures to values 1 mm Hg greater than the preload. This was done to examine the changes generated in the pulmonary flow waves. The same manipulations were performed at the other preload values of 8, 15, and 17 mm Hg.

We used a Sharp control air driving pump (H-4000). All fluid filled catheters were connected to Gould P-50 pressure transducers. The flow rate was measured electromagnetically with a Nihonkouden MFV-2100 flowmeter and a FF-150T cannulating flow

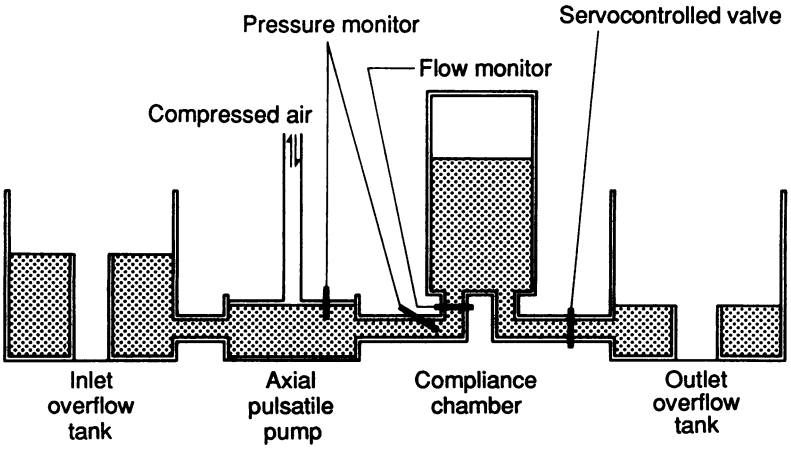

Figure 1 Simulation of the right heart after a modified Fontan procedure. The inlet overflow tank (preload), axial pulsatile pump, Wind-Kessel model (afterload), and the outlet overflow tank (left atrium) were connected in series and filled with water at 37°C.

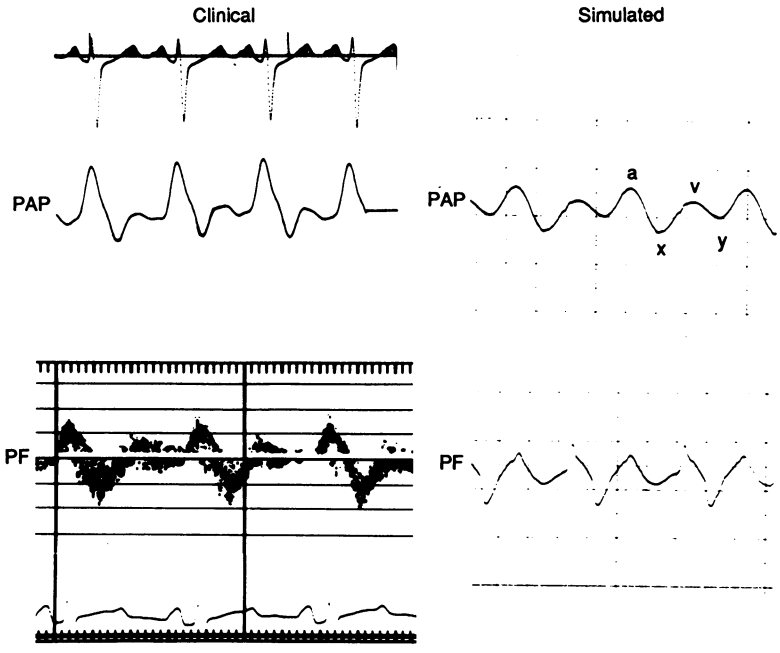

Figure 2 Clinical and simulated pulmonary arterial pressure (PAP) and pulmonary flow (PF) waves. Clinical PF waves were recorded by Doppler echocardiography. Simulated PAP and PF waves closely resemble the clinical examples.

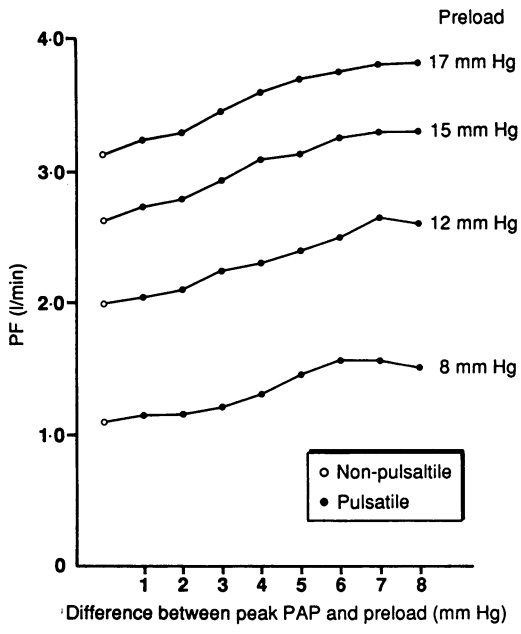

Figure 4 Changes in pulmonary flow (PF) examined at preloads of 8, 12, 15, and 17 mm Hg when all another variables remained constant. Each pulsatile flow pattern was produced by an increase in the peak pulmonary arterial pressure (PAP) in 1 mm Hg steps above the preload.

transducer. Pressure and flow signals were recorded on a San-ei 361 eight channel polygraph.

Results

Under standard conditions, with a 12 mm Hg preload, we simulated eight pulsatile flow patterns with peak pulmonary arterial pressures 1 mm Hg greater than the preload—that is 13, 14, 15, 16, 17, 18, 19, and 20 mm Hg when the preload was 12 mm Hg. We recorded pump pressure, pulmonary flow, pulmonary arterial pressure, and waves at each pressure (fig 3). These waves closely resembled clinical tracings. As peak pulmonary arterial pressure became increasingly greater than the preload, pulmonary flow increased from 2·00 l/min in the non-pulsatile state to a maximum of 2·65 l/min at a peak pulmonary arterial pressure of 19 mm Hg (fig 4). Changes in pulmonary flow were also seen when the preload was set at 8, 15, and 17 mm Hg (fig 4) and all other standard conditions were held constant. At any given preload there was a corresponding pulmonary arterial pressure that produced the greatest pulmonary flow. For an 8 mm Hg preload the largest pulmonary flow seen was 1·55 l/min at a peak pulmonary arterial pressure of 14 mm Hg. At a 15 mm Hg preload and 22 mm Hg peak pulmonary arterial pressure, pulmonary flow was 3·30 l/min. With a preload of 17 mm Hg and a peak pulmonary arterial pressure of 25 mm Hg, pulmonary flow was 3·80 l/min. Peak flow rates under non-pulsatile conditions for preloads of 8 mm Hg, 15 mm Hg, and 17 mm Hg were 1·10 l/min, 2·65 l/min, and 3·15 l/min respectively. Pulmonary flow when peak pulmonary arterial pressures were optimal was much greater than under non-pulsatile conditions.

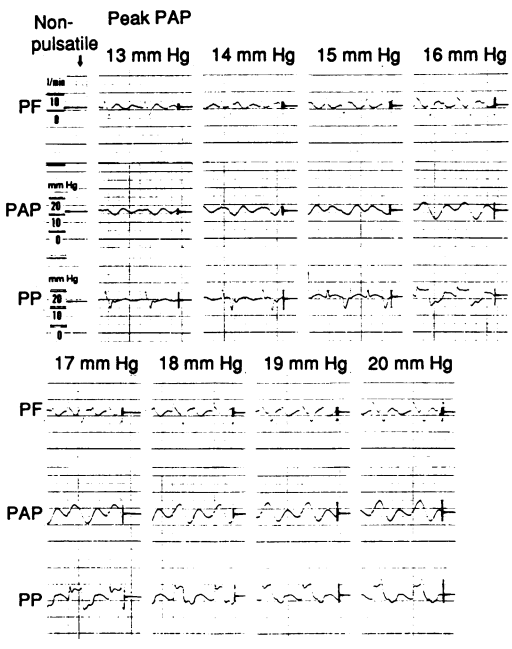

Figure 3 Non-pulsatile and pulsatile pulmonary flow (PF), pulmonary arterial pressure (PAP), and pump pressure (PP) waves under standard conditions: flow rate 2·00 l/min, with 12 mm Hg preload, resistance, 3·0 Wood units and an outlet overflow tank pressure of 6 mm Hg in a non-pulsatile state. As the peak PAP increased above the preload, the PF increased from 2·00 l/min in the non-pulsatile state to a maximum of 2·65 l/min at a peak PAP of 19 mm Hg.

Discussion

The results of clinical and experimental studies that examined the importance of atrial contraction in providing energy to the pulmonary circulation in patients after a modified Fontan procedure are conflicting. Yacoub reported that pulsatile pulmonary blood flow had a beneficial effect on gas exchange and affected the regulation of pulmonary vascular tone after this operation.[5] Using a velocity transducer Nakazawa *et al* showed that atrial contraction enhanced flow into the pulmonary circulation.[6] However, the results of several experimental[7,8] and clinical[9,10] studies suggest that atrial

contraction may not be essential for the maintenance of the pulmonary and systemic circulations. Thus the importance of pulsatile pulmonary blood flow from atrial contraction after the modified Fontan procedure remains controversial.

Changes in the right heart can be difficult to evaluate in vivo because changes in the left heart can affect the right heart. In the present right heart model we directly investigated the haemodynamic effects of pulsatile pulmonary flow. The axial pulsatile pump in this model had a contraction characteristic that was linearly related to the afterload. When Lau *et al* studied the instantaneous pressure-volume relation of the right atrium during isovolumic contraction in the canine heart they found a straight line relation between the end systolic pressure and volume.[11] This suggests that the valveless axial pulsatile pump model may be able to simulate the haemodynamic effects of the right heart after the modified Fontan procedure. In fact, the pulmonary arterial pressure and pulmonary flow waves simulated in this model closely resemble the waveforms obtained from patients after the modified Fontan procedure. On the other hand, de Leval *et al*, using a similar system, reported that the introduction of pulsation predominantly increased the proximal venous pressure.[12] The reasons for the discrepancy between their results and ours is not clear. However, their report did not specify the pulsatile conditions or include the pulmonary artery pressure tracings. We believe that it is important that the simulated pulmonary arterial pressure trace mimics a clinical one before meaningful conclusions can be drawn. We found that the pulmonary arterial pressure trace was affected by the pump driving pressure, fraction of systole, and the vacuum. In fact, in our model, suboptimal pulsatile conditions produced poor pulmonary flow, resulting in pulmonary arterial pressure tracings that did not resemble clinical ones. Such suboptimal conditions might contribute to increased back flow rather than to forward flow. Whether one of these factors or some other factor is responsible for the discrepancy between our results and those of de Leval *et al* is not clear and requires further study.

Nevertheless, in our model when the pulsatile conditions were optimised to produce a pulmonary arterial pressure trace that mimicked a clinical one, we found that as the peak pulmonary arterial pressure increased, pulmonary flow gradually increased until it was greater than during the non-pulsatile state. The resistance in this model is fixed by the non-elastic tube. So an increase in pulmonary flow with pulsation suggests that pulsation decreases the resistance. These results suggest that pulsatile pulmonary blood flow has a beneficial effect on the pulmonary circulation after the modified Fontan procedure.

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