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Control of a Mock Circulatory System to Simulate the Short-Term Baroreflex[§]

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Abstract – **A mock circulatory system (MCS) integrated with the baroreceptor reflex, a neurological function that regulates the mean systemic arterial pressure (P_{sa}) by adjusting heart rate, ventricular contractility, and systemic resistance through negative feedback, was developed to simulate the key hemodynamic variables in response to various physiological load changes. The MCS consists of two compliance chambers representing the left atrium and systemic artery, a proportional valve as a variable resistor mimicking the systemic vascular resistance (SVR), and a centrifugal pump as a current source simulating the pumping mechanism of the heart. The model of the baroreceptor reflex was implemented in software to generate the reference signals of the cardiac output (CO) and SVR. These two reference signals along with the models of the centrifugal pump and the proportional valve were used to control the rotational speed of the pump and the gap of the valve such that the desired CO and SVR can be reached. Performance of the MCS was tested under different cardiovascular demand levels from resting to heavy exercise. The test results show that this simple MCS was able to simulate the response of key hemodynamic variables comparable to the same variables produced by a complex model from a computer simulation. The MCS performed well in simulating the hemodynamic variables under resting and mild exercise conditions. This novel MCS implementation provides a much more physiological meaningful tool comparing with existing MCS. It is a valuable asset for studying the physiology of the circulation, for heart assist devices testing, and for bioengineering education**.

I. INTRODUCTION

A mock circulatory system (MCS) that can model major parts of the human circulatory system as closely as possible is an important tool for engineers wishing to test prototype cardiovascular devices *in vitro* [1]. Such tests on an MCS have a much shorter turn-around time and cost significantly less than a full-scale animal test. There is always, however, a trade-off between the degree to which an accurate reproduction of hemodynamic variables under different conditions is sought and the physical complexity of the MCS constructed by several groups [1]-[3]. While it is possible to use modeling techniques based on Navier-Stokes equations for a multi-dimensional description of flow in pipelines, such a model makes it difficult to extract measurable signals in order identify parameters [1]. On the other hand, the realization of the MCS as an RLC circuit will allow us to define electrical impedance, voltages and currents that correspond to measurable fluidic resistances, pressures and flow rates.

Ventricular assist devices (VAD), mechanical pumps used in patients with cardiovascular diseases who are awaiting cardiac transplantation, are one type of the devices that use MCS for bench testing. Evaluation of a prototype VAD and its control strategy is usually performed either by animal experiments or by mock circulation tests on the bench top. Although animal experiments can provide a close approximation to the human circulation, there are several associated disadvantages that make a bench-top experiment more desirable. The existing mock circulatory loops have very limited ability to simulate the intrinsic control of the heart and circulation to varying load. Since the load of the heart is strongly affected by the VAD, it is desirable that a mock circulation system could properly simulate the hemodynamic response of the native heart to the VAD support by including the intrinsic control mechanism of the cardiovascular system in the MCS.

The short-term carotid baroreflex is important in performing the intrinsic control by maintaining arterial blood pressure at a level to ensure optimum organ perfusion of nutrients and waste removal [5],[6]. The baroreflex, like many physiological control systems, takes the form of a negative feedback control loop. Pressure deviations are detected by cells in the carotid sinus which send impulses to the central nervous system (CNS) via the afferent neurons. The CNS responds with efferent sympathetic and parasympathetic stimuli which affect the heart rate, maximum elastance of the heart muscle, and vascular resistance as shown in Fig. 1 [7].

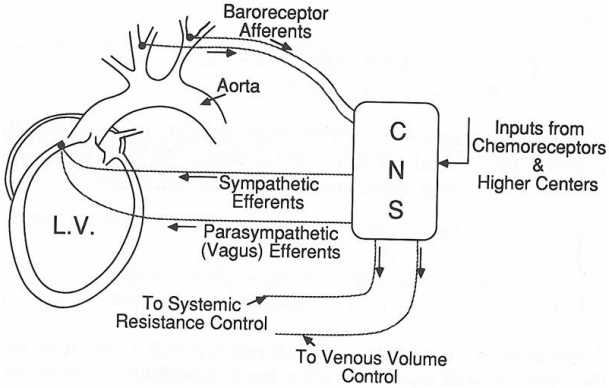

Fig. 1, CNS control of the heart activities

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In this paper, a control scheme that mimics the baroreflex as shown in Fig. 2 was designed to control MCS. The goal is to regulate P_{sa} under different hemodynamic load conditions. A modified three-element windkessel model was chosen as the MCS together with a programmable proportional valve and a centrifugal pump as actuators. The model of the baroreflex was implemented in a microprocessor to generate the desired P_{sa} based on the activity level of the human body and determine the required heart rate (*HR*), the maximum elastance of the ventricle (E_{max}), and systemic vascular resistance (*SVR*) based on the difference between the desired P_{sa} from the model and the actual P_{sa} measurement from the MCS. These three variables, *HR*, E_{max}, and *SVR*, were then used to control the MCS by changing pump speed (ω) to achieve desired cardiac output and varying the gap of the proportional valve (g) to adjust the systemic resistance.

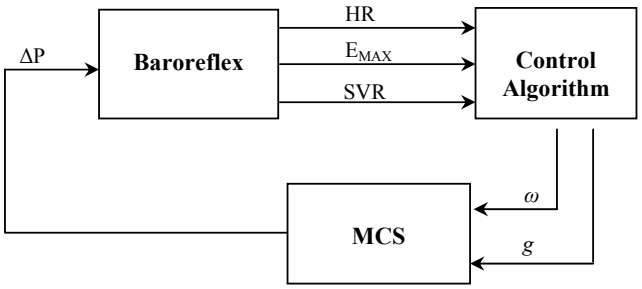

Fig. 2. Block diagram of the baroreflex control of the MCS

Performance of the system was tested by varying the activity level from resting to heavy exercise in the baroreflex model and comparing the resulting hemodynamic variables from the MCS with that from computer simulation [8] and from literature [5] under the same test conditions. The results from the MCS experiment are similar to that from literature [5], which implies that this new MCS is capable to simulate the cardiovascular system at different activity levels.

II. Mock Circulatory System

The MCS used in this study, as shown in Fig. 3, is a modified three element windkessel model. It consists of two chambers as arterial compliance (C_{sa}) and venous compliance (C_{la}), one proportional valve as the total peripheral resistance (*SVR*), and one centrifugal pump as the pumping mechanism of the native heart. Since the model is aimed at studying steady-state behavior of the circulatory system, the effect of fluid inertia that is usually found in more complex dynamic models is neglected [9].

The compliance chambers were constructed from Perspex cylinders with diameters sufficient to produce the required capacitive effect. A centrifugal pump head (BP-80, Medtronic), driven by the BVP-Z electrically controlled motor (Ismatec SA, Switzerland), was used to circulate the blood analogue (35% glycerol and 65% water by volume at room temperature) in the MCS. The low-friction design of the pump eliminated physical contact between the pump drive and the pump head. Torque is transmitted through a magnetic coupling between a ring shaped rotating magnet on the drive and a similar magnet within the pump head impeller. The pump speed can be varied from 500 to 2750 rpm by changing the voltage input to the motor controller through a data acquisition system. The PV14 proportional valve (Omega Engineering, Stamford, CT) represents *SVR* in the MCS. The valve orifice is controlled by a 4-20mA current signal that specified any valve position from fully open to fully shut. A voltage-to-current signal conditioner (CCT-01, Omega Engineering) was used to control the valve using a voltage signal through a data acquisition system. All these fluid elements were connected by ⅜ inch Tygon S-50-HL tubing.

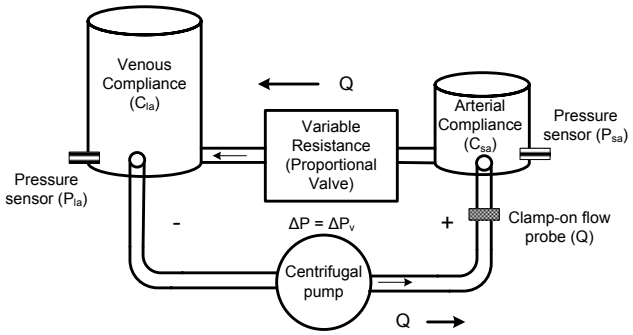

Fig.3. Schematic of MCS, showing sensors and actuators, where Q represents the cardiac output *CO*.

Pressures in both compliance chambers, P_{sa} and P_{la} as the mean arterial pressure and the left atrial pressure, were measured at the bottom of the chambers by using two pressure transducers (BLPR, World Precision Instruments, Sarasota, FL) with a digital bridge amplifier (DBA-8000, World Precision Instruments). The pressure drop across the centrifugal pump (ΔP) and the valve (ΔP_v) is represented by the difference between P_{sa} and P_{la}. The rate of the flow circulating in the MCS, mimicking cardiac output, was measured by a clamp-on ultrasonic probe with a tubing flow meter (ME9PXL703 and TS410, Transonic Systems Inc., Ithaca, NY).

Data acquisition and control were implemented using a workstation running LabView™ (National Instruments, Austin, TX) and downloaded to a PXI chassis (NI PXI-1042) containing an embedded Pentium 4-based controller (NI PXI-8186) and a FPGA-based data acquisition card (NI PXI-7831R). A graphical user interface was operated from the workstation to control the pump speed and the gap of the proportional valve and acquire pressure and flow data from the embedded control via an Ethernet interface at a sampling rate of 1 kHz.

III. Control Scheme

The block diagram representing the control scheme of the entire system is shown in Fig. 4. The baroreflex model [5] receives the suggested activity level from the operator, the pump flow (cardiac output, *CO*) and mean arterial pressure P_{sa} from the MCS, to provide the effect on the *HR*, E_{max}, and *SVR*. Combination of these regulated variables allows changes in the baroreflex in response to cardiovascular demand variations, such as the onset of

exercise, to be studied. In this model, pressure perturbations are detected by carotid sinus baroreceptors which send impulses to the CNS via the afferent neurons. Increases in the blood pressure within the carotid sinus (P_{sinus}) cause the walls of the blood vessels to stretch which leads to an increase in the discharge frequency of the afferent neurons f_{as} described by

$$f_{as}(t) = \frac{f_{\min} + f_{\max} e^{\frac{P_{sinus}(t) - P_0}{K_a}}}{1 + e^{\frac{P_{sinus}(t) - P_0}{K_a}}}, \quad (1)$$

The baroreflex is also sensitive to the rate of change in arterial pressure, which is supposed to produce an offset in f_{as}. However, because this study focuses on a non-pulsatile cardiac model, this offset is ignored. The values and brief definitions of the constants used in this baroreflex model are as in [5] The discharge frequency of the afferent neurons f_{as} in (1) changes the sympathetic efferent firing rate f_{es} and the parasympathetic efferent firing rate f_{ev} [10] as

$$f_{es}(t) = f_{es\infty} + (f_{es0} - f_{es\infty}) e^{-K_{es} f_{as}(t)} \quad (2)$$

and

$$f_{ev}(t) = \frac{f_{ev0} + f_{ev\infty} e^{\frac{f_{as}(t) - f_{as0}}{K_{ev}}}}{1 + e^{\frac{f_{as}(t) - f_{as0}}{K_{ev}}}}, \quad (3)$$

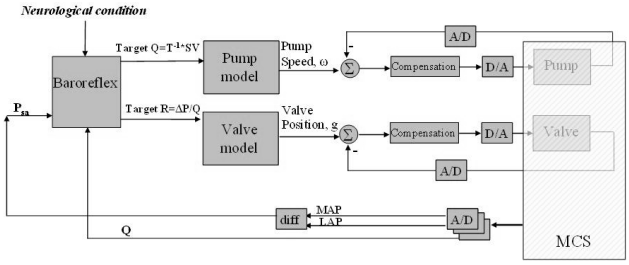

Fig. 4, Block diagram of the control scheme

The systemic resistance (*SVR*) consists of three parallel resistances, active muscle resistance (R_{am}), extra-splanchnic resistance (R_{ep}), and splanchnic resistance (R_{sp}). The effects of the sympathetic efferent firing rate, $f_{es,}$ on these resistances are exhibit identical model structure having only different parameter values:

$$e_{R_i}(t) = \begin{cases} G_{R_i}(\ln[f_{es}(t - D_{R_i}) - f_{es\min} + 1], & f_{es} \geq f_{es\min} \\ 0, & f_{es} < f_{es\min} \end{cases}, \quad (4)$$

$$\frac{d\Delta R_i(t)}{dt} = \frac{-\Delta R_i(t) + e_{Ri}(t)}{\tau_R}, \quad (5)$$

$$R_i(t) = \Delta R_i(t) + R_{0i}, \quad (6)$$

where i represents *ep*, *sp*, and *am*, respectively. The total systemic resistance *SVR*(t) is then determined by

$$SVR(t) = \frac{1}{\sum_{i=ep,sp,am} \frac{1}{R_i(t)}} \quad (7)$$

The effect of f_{es} to the contractility (represented by E_{max}) can be expressed as:

$$e_{E_{\max}}(t) = \begin{cases} G_{E_{\max}}(\ln[f_{es}(t - D_{E_{\max}}) - f_{es\min} + 1], & f_{es} \geq f_{es\min} \\ 0, & f_{es} < f_{es\min} \end{cases} \quad (8)$$

$$\frac{d\Delta E_{\max}(t)}{dt} = \frac{-\Delta E_{\max}(t) + e_{E_{\max}}(t)}{\tau_{E_{\max}}} \quad (9)$$

$$E_{\max}(t) = \Delta E_{\max}(t) + E_{\max_0} \quad (10)$$

Since E_{max}(t) occurs near the end of left ventricular systole, which can be approximated as

$$E_{\max}(t) \approx \frac{P_{es}(t)}{V_{es}(t) - V_0}, \quad (11)$$

where P_{es}(t) and V_{es}(t) are end-systolic pressure and volume of the left ventricle. V_0 is the unstressed volume of the ventricle. *SV* (in ml/beat) represents the blood volume ejected from the left ventricle in one hear beat, which is the difference between the end-diastolic volume and the end-systolic volume, $SV = V_{ed} - V_{es}$. By approximating P_{es} as mean arterial pressure P_{sa}, obtained from the pressure measurement at the arterial compliance in the MCS, and rearranging (11) leads to,

$$SV(t) = V_{ed} - V_0 - \frac{P_{sa}(t)}{E_{\max}(t)}. \quad (12)$$

The overall effect of cardiac CNS stimulation is a balance between the opposing parasympathetic (delivered via the vagus nerve) and the sympathetic responses. Increases in the f_{es} as the sympathetic response is represented by (13) and (14):

$$e_{T_s}(t) = \begin{cases} G_{T_s}(\ln[f_{es}(t - D_{T_s}) - f_{es\min} + 1], & f_{es} \geq f_{es\min} \\ 0, & f_{es} < f_{es\min} \end{cases} \quad (13)$$

$$\frac{d\Delta T_s(t)}{dt} = \frac{-\Delta T_s(t) + e_{Ts}(t)}{\tau_{Ts}}. \quad (14)$$

The effect of vagal response to the heart rate change is expressed as

$$e_{Tv}(t) = G_{Tv} f_{ev}(t - D_{T_v}), \quad (15)$$

and

$$\frac{d\Delta T_{Tv}(t)}{dt} = \frac{-\Delta T_{Tv}(t) + e_{Tv}(t)}{\tau_{Tv}}. \quad (16)$$

The period of one cardiac cycle, T_c, in seconds is the sum of the basal period (T_0) and the changes induced by the vagal and sympathetic responses as

$$T_c(t) = T_0 + \Delta T_{Tv}(t) + \Delta T_{Ts}(t). \quad (17)$$

The cardiac output (*CO* in L/min) can be determined by

$$CO(t) = SV(t) / T_c(t) \cdot 0.06, \quad (18)$$

where *SV*(t) and T_c(t) are determined by (12) and (17).

The time-varying *SVR* from (7) and *CO* from (18) were used to determine the gap of the proportional valve and the rotational speed of the centrifugal pump based on the models of these two actuators. The model of the proportional valve, based on Ohm's law, represents the pressure drop across the valve, $\Delta P_{v,}$ as

$$\Delta P_V(g, CO) = R_V(g, CO) \cdot CO, \quad (19)$$

where $R_V(g, CO)$ is the fluid resistance of the valve. The variable g represents the fractional opening of the valve, a substitute variable of the valve orifice. Therefore, R_V

mimics *SVR* in the MCS, which is a function of the flow through the valve and the orifice size. The function $R_V(g,CO)$ and its parameters were determined by least-squares fit to the experimental data [12]. $R_V(g,CO)$ is an affine function,

$$\Delta P_V(g,CO) = R_V(g,CO)\cdot CO = R_{v_1}CO + R_{v_2}CO^2, \quad (20)$$

where R_{v1} and R_{v2} are functions of g,

$$R_{v_1}(g) = b_0 g^{b_1} + b_2, \quad R_{v_2}(g) = c_0 g^{c_1} + c_2 \quad (21)$$

with constant values b_0 = 1.6059 x 10^9, b_1 = -5.7960, b_2 = 1.8576, c_0 = 1.6252 x 10^{10}, c_1 = -6.6853, and c_2 = 0.34620. Given *SVR* and *CO,* a unique solution of g is difficult to obtain using the valve model in (20), therefore, a two-dimensional look-up table within the physiological range was implemented in Matlab™ to search for an optimal g for a given *CO* to minimize the difference between the predicted and actual ΔP_v. A physiological pressure and flow range of 35 to 150 mmHg and 2 to 9 *L*/min was selected to improve the look-up speed and accuracy of the table.

The model of the centrifugal pump can be expressed as

$$\Delta P(\omega,CO) = (k_0 + k_1\omega)CO + k_2\omega^2, \quad (22)$$

where ΔP is the pressure drop across the pump head, ω is the pump rotational speed in revolutions per minute (rpm), and *CO* is the flow rate through the pump in liters per minute (L/min). ΔP consists of two key variables: hydrostatic pressure produced due to pump rotation (denoted as $k_2\omega^2$) [12] and internal fluid resistance in the pump (represented by an affine function of ω as $k_0 + k_1\omega$). The model parameters in (22) were identified as k_0 = 0.2371, k_1 = 1.272x10^{-2}, and k_2 = 6.341x10^{-5} through a linear regression analysis.

In the MCS, ΔP can be determined by the pressure difference between the systemic arterial pressure P_{sa} and the left atrial pressure P_{la}. The pump flow *CO* is the cardiac output. For a given ΔP from the pressure measurements and *CO,* from the baroreflex model in (18) the reference pump speed can be determined by solving (22) for ω,

$$\omega = \frac{-k_1CO + \sqrt{(k_1CO)^2 - 4k_2(k_0CO - \Delta P)}}{2k_2}. \quad (23)$$

LabView™ was used to design a host-client virtual instrument (VI) to serve as an interface between the MCS hardware and the baroreflex algorithm. This interface acquired data from the various pressure and flow sensors and relayed control signals to the pump and valve. The Real-Time Workshop (RTW) of Matlab™ was used to generate an optimized C dynamically linked library (DLL) of the baroreflex implemented in Simulink™. The DLL and client VI were downloaded to the embedded PXI controller over Ethernet prior to starting the experiment from the host VI located on the monitoring workstation.

V. Performance Evaluation

The purpose of this evaluation is to determine the overall performance of our baroreflex model and the MCS design under a series of tests defined in literature [5]. This evaluation is crucial to ascertain the correct interaction of the sub-models that make up the MCS, and establish the accuracy of this design. The model of the MCS along with the control scheme was implemented in Simulink™ to compare the hemodynamic variables simulated by the model of the MCS model [8] with that from literature [5],[6]. The same simulation was repeated with the control scheme implemented in real-time with an embedded controller to control the MCS. The basal values of key hemodynamic variables obtained from our simulated MCS and experimental MCS are listed in Table 1.

TABLE 1
COMPARING BASAL VALUES IN EXPERIMENTAL AND SIMULATED MCS.

	Experimental MCS	**Simulated MCS**
Pump Speed (RPM)	1831	n/a
Target *CO* (L/min)	5.45	5.24
Valve Gap (%)	40.1	n/a
Target *SVR* (mmHgs/ml)	0.922	1.016
ΔP (mmHg)	89.4	88.8
***SV* (ml)**	88	78
E_{max}	2.76	2.86
***HR* (bpm)**	62.3	66.8

Three tests were conducted to evaluate the performance of this new MCS: artificial heart pacing, open loop evaluation of the baroreflex response, and the onset of moderate and heavy exercise. In the artificial heart pacing experiment, the baroreflex regulation of the heart rate was disabled. The heart rate was manually set from 30 to 170 beats/min with an increment of 10 beats/minute. The remaining regulatory pathways affecting *SVR* and E_{max} were left intact. The resulting steady state values of *SV* and *CO* are shown in Fig. 5a and Fig. 5b, respectively. The stroke volume from our experiment was 25% lower than the reference data while the cardiac output was 41% higher than the reference.

The open-loop performance of the carotid baroreflex response was evaluated by disabling the feedback path that connects ΔP as an input to the baroreflex. This pressure was manually set from 40 to 150 mmHg with an increment of 10 mmHg. The corresponding effects on P_{sa}, *CO*, *HR*, *SV* and *SVR* after a steady state was reached were recorded. Normalized error indices were found to be 0%, 20.7%, 5.6%, 8.8% and 14.2% for *HR*, P_{sa}, *CO*, *SV* and *SVR* respectively. Fig. 5c-5e show the response of P_{sa}, *CO*, and *SVR* as the carotid sinus pressure was varied.

Voluntary moderate and heavy exercise levels were simulated with vasodilation of peripheral vasculature and offsets to the efferent sympathetic (f_{ev} in (2)) and vagal neural pathways (f_{es} in (3)) feeding into the baroreflex [6],[10]. The offsets, in the form of ramp inputs lasting 5 seconds before reaching their target values listed in Table 2, mimic the commands from the central nervous system at the

onset of different exercise levels and trigger the baroreflex intro preparing the circulatory system to accommodate the physiological demand. The steady state values of *HR*, *SVR*, P_{sa}, *CO*, *SV* and E_{max} were then recorded. Fig. 5f shows the percentage change in these hemodynamic parameters for exercise of medium intensity and compares the reference values to the simulated and hardware implementation of our MCS.

TABLE 2
OFFSET VALUES FOR EXERCISE TEST

Offset Value at Steady-state	Moderate Exercise	Heavy Exercise
f_{ev} offset	2.99	2
f_{es} offset	11	19

VI. DISCUSSION AND CONCLUSION

The hardware implementation produced a qualitative trend with the artificial heart pacing test in good agreement with the reference results [5]. However, the quantitative results were less promising and deemed not significant, since there is no intention to run the model in open-loop with *HR* as an input, as the test suggests. The target and actual cardiac output of the hardware implementation of the MCS deviated more at higher heart rates (Fig 5b). This is likely due to a higher than expected fluid resistance introduced by the pump head and the tubing in the MCS.

For the carotid baroreflex response test, good qualitative performance was observed. With the exception of the P_{sa} and *SVR* parameters, the remaining had error indices with respect to the reference of less than 10%. The most important observation was the higher than expected target and actual values of peripheral resistance.

The exercise tests are considered the most important in all three tests, as they provide a measure of the robustness of the MCS and baroreflex algorithm. One observation made during this series of tests was the inability of the centrifugal pump to deliver the maximum cardiac output demanded by the heavy exercise test. At 2950 RPM (maximum pump speed) and with the proportional valve fully open, the maximum attainable cardiac output was 9.36L/min while the target for heavy exercise was 12.9L/min. In addition, the lowest attainable peripheral resistance (accompanying maximum vasodilation) in the hardware was 1.087mmHgs/ml, while the target resistance was 0.45mmHgs/ml. Reference [10] explained qualitatively how an operating point shift in the mean arterial pressure should accompany conditions such as voluntary exercise. The hardware test was able to illustrate this increase in the regulated set-point for the P_{sa} better than that from the simulation.

The overriding design paradigm was to select simple MCS and baroreflex models that would lend themselves well to a practical implementation without sacrificing agreement with reference results. We were able to successfully model the behavior of the baroreflex under different situations and identify accurate fluid element models of a pump and valve as actuators in the physical MCS. Limitations were encountered mainly with the available hardware, i.e. insufficient maximum flow and minimum resistance. To improve resemblance of our MCS to the human circulatory system, a pulsatile heart function can be explored.

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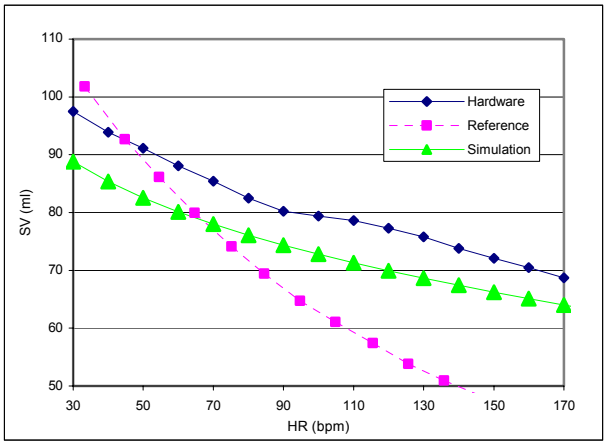

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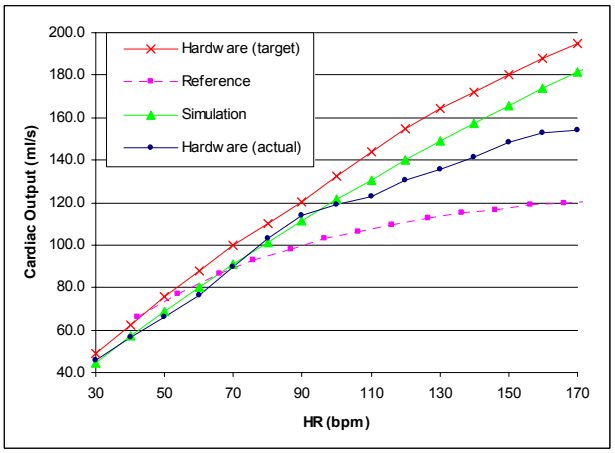

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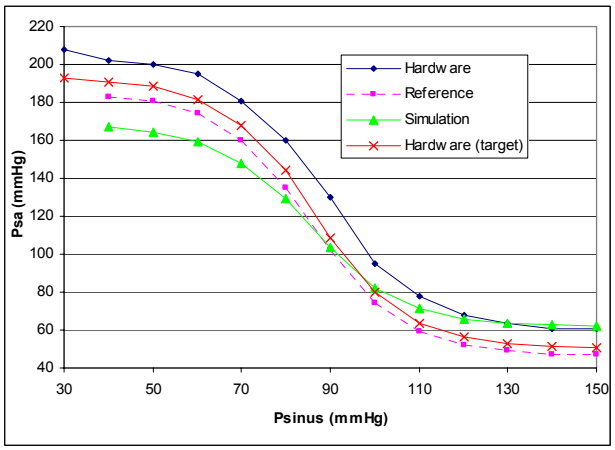

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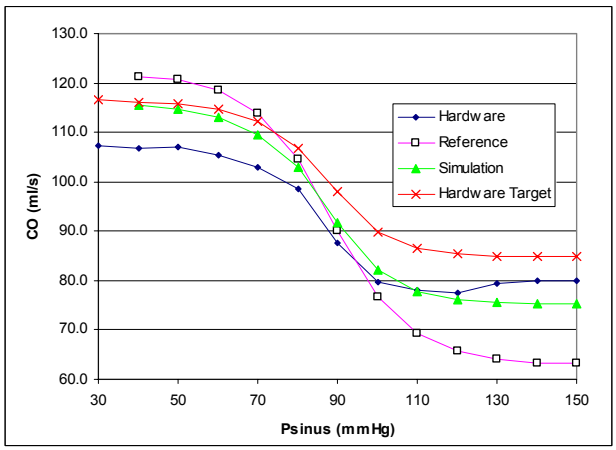

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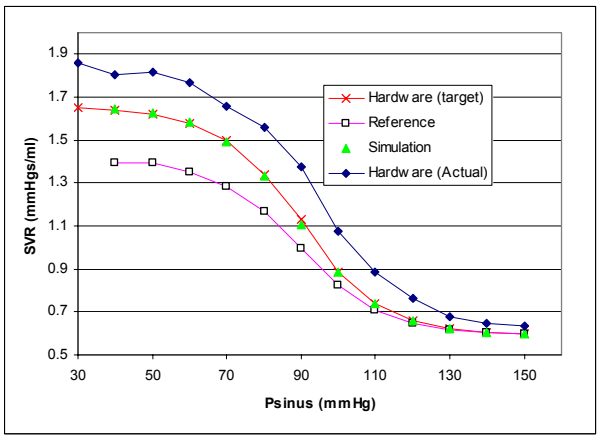

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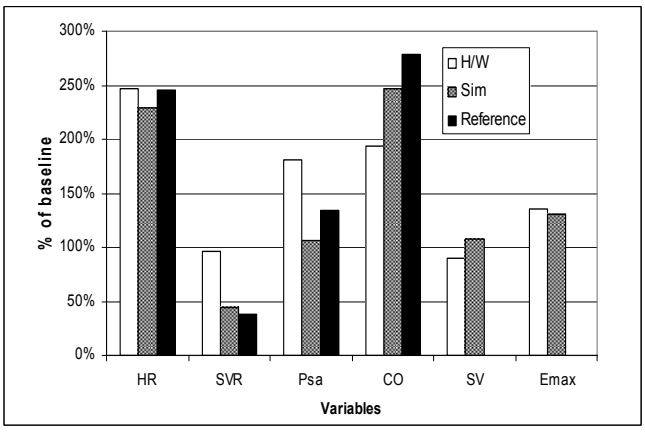

(f)

Fig. 5, Plots of the test results comparing response of simulated, experimental and reference MCS [5][6]. (a), (b): Artificial heart pacing test; (c), (d), (e): Open-loop baroreflex response test; (f): Steady state effect on basal parameters after onset of medium intensity exercise.