Minimal model simulation of diseases affecting the cardiovascular system

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Introduction

Medical staff diagnosing cardiovascular system (CVS) dysfunction are often faced with a confusing array of measured data, where the cause of the original dysfunction is often clouded by the effects of the autonomous nervous system. A robust mathematical model of the CVS would help medical staff and researchers delineate the relative effects of a variety of dysfunctions and reflexes on CVS haemodynamics. The result would be improved understanding of the pathophysiology of the CVS and insight into the most beneficial therapies that could be applied in each case. This research investigates the ability of a minimal cardiovascular system model to simulate the haemodynamic effects of dysfunctions that affect the heart and circulatory system.

Aim to assist medical staff, students and researchers in understanding, diagnosis and treatment selection for CVS dysfunctions.

Method

A minimal approach has been taken to modeling the cardiovascular system using a minimal number of governing equations and parameters. Model parameters are adjusted to simulate a variety of CVS disease states found in critically ill patients. Simulation results are compared with known physiological responses.

Cases simulated include:

- Aortic valve dysfunctions
  - Stenosis
  - Regurgitation
- Shock
  - Heart failure
  - Hypovolemic shock
  - Septic shock
  - Obstructive Shock:
    - Pericardial tamponade
    - Pulmonary embolism

The autonomous nervous system

A basic model of the autonomous nervous system is assumed where a drop in mean arterial blood pressure (MAP) will activate the following reflexes responses:

- Increase the contractility of both ventricles.
- Increase heart rate.
- Increase systemic vascular resistance through vasoconstriction.
- Reduce venous dead space through venous vasoconstriction, thus increasing right atrial filling pressure.

NOTE: Autonomous reflexes

In all cases were mean arterial blood pressure drops to 80mmHg, the autonomous reflexes are simulated by increasing the contractility of both ventricles by 35%, the heart rate is increased to 120 beats per minute, the systemic vascular resistance is increased by 35% and venous dead space is reduced by 1.9 litres to increase venous return.

Results

Model simulations of the disease states listed were performed, including the four examples shown. Simulations of each disease state compared well with commonly accepted changes in arterial and venous pressures in the pulmonary and systemic circulation systems, end-diastolic ventricle volumes and cardiac output. Once a dysfunction is simulated, the sensitivity of the cardiovascular system to changes in parameters such as contractility, heart rate, systemic vascular resistance and blood volume can be easily investigated. Compensatory mechanisms were found to have different influences on CVS dynamics depending on the operating conditions of the CVS.

Conclusion

The minimal cardiovascular system model is found to simulate a diverse range of dynamics that are characteristic of different types of CVS dysfunction, while other models we studied were only shown to simulate dysfunctions in specific areas of the CVS. This research shows the potential of a simple model for use as a tool for investigating CVS function and determining suitable treatment strategies for the entire cardiovascular system.

Heart Failure Simulation

Scenario:

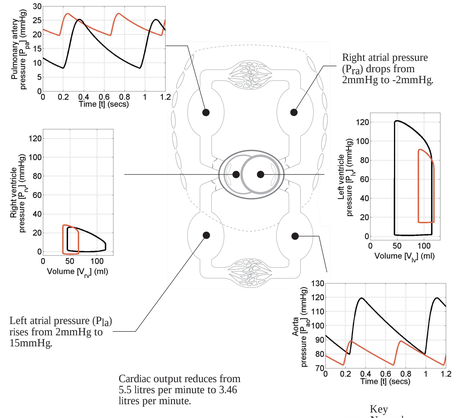
- The left heart receives an inadequate supply of oxygen due to a blockage in the coronary artery. Contractility is reduced by a factor of 4 and diastolic elastance is increased by a factor of 4.

Pathophysiology:

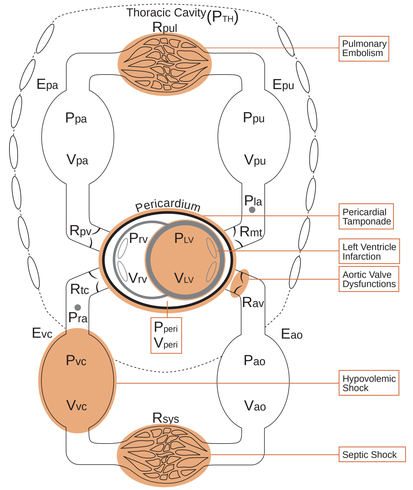
- Mean arterial pressure decreases and the autonomous nervous system is activated, stabilizing the mean arterial blood pressure at 80mmHg.
- Increased pulmonary pressures are caused by reduced left ventricle function relative to the normal right ventricle function, and autonomous nervous system induced increases in stressed blood volume. These increased pulmonary pressures can cause fluid buildup in the lungs.
- Increased pulmonary venous pressure causes increased left ventricle filling pressure and over distention of the left ventricle, which is unable to pump the additional blood supply.

Treatment:

- Beta-blockers. To reduce right ventricle activity causing lower pulmonary pressures and slow the sick left ventricle.
- Vasodilators. To reduce the systemic vascular resistance and reduce left ventricle afterload.
- Diuretics. To reduce stressed blood volume, lowering pulmonary pressures and reducing fluid buildup in the lungs.



Cardiovascular System Model



Valvular Dysfunction Simulation

AORTIC STENOSIS

Scenario:

- Calcium deposition on the surface of the aortic valve limiting its ability to open properly. Resistance through the aortic valve is increased by a factor of 7.

Pathophysiology:

- Blood pressure and cardiac output change minimally. However, systolic left ventricle pressures increase significantly due to increased afterload.

Treatment:

- Valve replacement.

AORTIC REGURGITATION

Scenario:

- The aortic valve becomes damaged and will not close properly. Blood is able to flow back into the ventricle during diastole, but with a much higher resistance of 7 times that of forward flow.

Pathophysiology:

- Mean arterial pressure decreases and the autonomous nervous system is activated, stabilising the mean arterial pressure at 80mmHg.
- The stroke volume of the left ventricle increases from 70ml to 140ml, however, due to excessive backwards flow through the aortic valve cardiac output reduces significantly.
- Left ventricle filling pressure increases due to both a reduction in pumping effectiveness of the left ventricle and autonomically activated increased stressed blood volume.

Treatment:

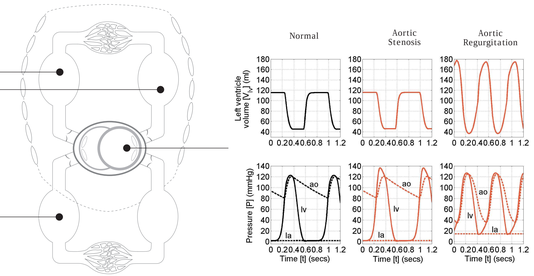
- Valve replacement.

Pulmonary artery pressure (P_{pa})
Normal: P_{pa} = 25/8 mmHg
Stenosis: P_{pa} = 20/7 mmHg
Regurgitation: P_{pa} = 28/19 mmHg

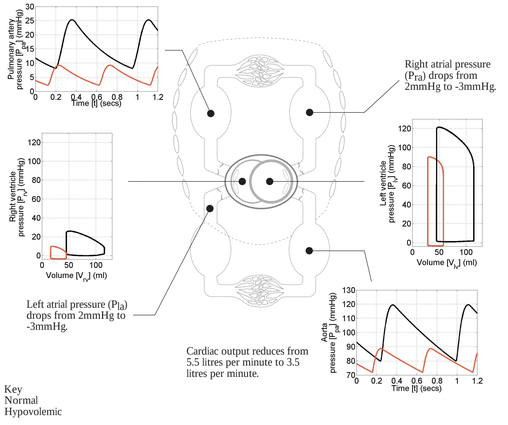
Left atrial pressure (P_{la})
Normal: P_{la} = 2 mmHg
Stenosis: P_{la} = -3 mmHg
Regurgitation: P_{la} = 24 mmHg

Right atrial pressure (P_{ra})
Normal: P_{ra} = 2 mmHg
Stenosis: P_{ra} = -3 mmHg
Regurgitation: P_{ra} = -1 mmHg

Cardiac output (CO)
Normal: CO = 5.5 l/min.
Stenosis: CO = 5.5 l/min
Regurgitation: CO = 3.4 l/min



Hypovolemic Shock Simulation



Scenario:

- Significant blood loss due to hemorrhage. Total blood volume is reduced by 2.5 litres.

Pathophysiology

- All pressures in the circulation system decrease along with the cardiac output. Blood pressure drops and the autonomous nervous system is activated stabilizing the mean arterial blood pressure at 80mmHg.

Treatment

- Increase blood volume.

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