

Study of Left Ventricle Pressure-Volume Loops in Renal Stenosis Case Using Equivalent Electrical Circuit

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Abstract

In this study, we have obtained the cardiac pressure-volume graphs of Renal Artery Stenosis disease with different stenotic values, moreover, the graphs are compared with normal condition. The cardiovascular Electronic circuit includes 42 elements including main arterial vessels. We have extracted the vessels geometrical data from the medical texts. The calculated pressure drops and the compliance variations of artery stenosis are applied to the electronic model. Then, the graphs have been obtained for different stenotic values (20%, 50%, 70%, and 90%) using ORCAD/MATLAB software. Finally, the results of modeling have been discussed and compared with the clinical data. The results of stenosis show that hypertension could be a symptom of the disease. When the rate of stenosis increases the blood pressure rises. This study is a tool for students in order to see the cardiovascular system operation in normal condition or abnormal.

Keywords: Renal Artery Stenosis (RAS); Pressure-volume (P-V) loop; Cardiovascular; Modelling

Introduction

Renal Artery Stenosis (RAS) occurs when blood flow leading to the kidneys is constricted by a plaque and becoming increasingly common because of atherosclerosis in an ageing population (McLaughlin et al., 2000; Vashist et al., 2002). It affects the middle and distal renal artery in the patients and may lead to the renal failure. Previous studies (Hassani et al., 2007; Kamran et al., 2008) found a positive correlation between blood pulse pressure and the rate of renal stenosis and noted that the hypertension due to the stenosis depends on many genetic factors such as fibrillin. The investigations (Hassani et al., 2007; NIH, 2003; Kamran et al., 2008) indicate that increasing pulse pressure is associated with the stenosis expansion rate which leads to hypertension. Patients usually present with hypertension and varying degrees of renal impairment, although silent RAS may be present in many patients with vascular diseases (Agroyannis et al., 2002). Several studies have evaluated the prevalence of RAS in patients undergoing angiography for a variety of extra renal vascular problems including abdominal aortic aneurysms, coronary artery disease, peripheral vascular disease and aorta-occlusive disease (Scoble, 1995; Greco and Breyer, 1997; Hirschberg et al., 1998). Drs. Chaudhry and Schainfeld emphasized the important point that hypertensive pulmonary edema could occur in patients with bilateral RAS (Rajachandran, 1997). RAS is a narrowing or blockage of the artery that supplies blood to the kidney. It is caused by atherosclerosis, fibromuscular dysplasia of the renal artery wall, or scar formation in the artery (Colm and Magee, 2005; Vagaonescu and Dangas, 2007; Bude et al., 2003; Khan, 2009; Coen et al., 2003) have shown that the RAS may lead to the hypertension, fluid retention, progressive renal failure and flash pulmonary edema using the combination of the hemodynamic and electronic parameters.

We have presented the pressure-volume graphs of renal stenosis with different rates using an electronic cardiovascular system. The study includes three parts:

- A review of the electronic cardiovascular system (Kamran et al., 2007)

- Obtaining the left ventricle pressure-volume loops using the mathematical method.
- Comparing the loops with the relevant clinical observations.

Material and Methods

The review of the cardiovascular system model (Kamran et al., 2007; Hassani et al., 2007; Kamran et al., 2008)

The equivalent model of the system including the pulsatile heart and the arterial tree are illustrated in Figure 1. The electronic parameters are correlated to their mechanical parameters as follows: voltage (1volt) is analogous to pressure (1mmHg), capacitance (1000 μ F) to compliance (1ml/Pa), resistance (1k Ω) to resistance (1Pa.s/ml), and inductance (1 μ H) to inertance (1Pa.s²/ml) (Hassani et al., 2007; Kamran et al., 2008).

The electronic model consists of forty two elements represented the left and right ventricles, systemic arteries and veins, and pulmonary arteries and veins. Each element consists of a conduit for viscous blood flow characterized by a linear resistance and a volume storage element specified by a linear capacitor (Hassani et al., 2007; Kamran et al., 2008). The inertance of each element is featured by a linear inductor. The reference pressure is atmosphere pressure (or ground in electronic model). The energy of systolic contraction is modeled by superposing of three voltage suppliers and two ideal diodes (Hassani et al., 2007; Kamran et al., 2008). The voltage suppliers

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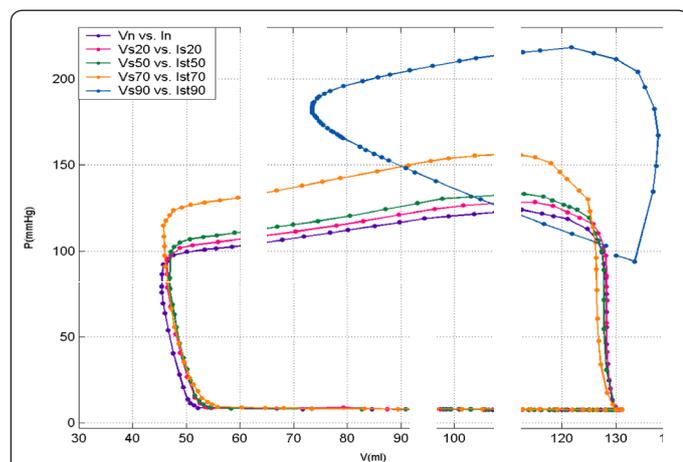


Figure 2: left Ventricle Pressure, Ventricle Volume, and the aorta pressure of 70% Renal Artery Stenosis. Cardiac Pressure-Volume loops of Renal Stenosis.

Renal (Diameter = 2..6mm, L=32mm)	ΔP (Pa) Stenosis	C (ml/Kpa) Stenosis	Diastol/Systole Pressure(mmHg)
20% Stenosis	7.46	0.01512	82-121
50% Stenosis	17.1	0.0236	85-127
70% Stenosis	75	0.116	97-148
90% Stenosis	3790	0.136	145-210

Table 1: Renal artery stenosis parameters (Klabunde, 2007b).

Cardiac pressure-volume loops

These graphs are obtained for Heart Rate of 60 beat per minute and Stroke Volume of 80 ml/min in normal condition. As we can clearly observed, stroke volume has not changed whereas the significant increase in pressure could be understood. Comparing the stenotic blood pressure and stroke volume to normal condition, we observed the following results: In condition of 20% RAS, both Systolic and diastolic pressure increase about 4 mmHg while the blood pressure of 50% RAS is 134/87 mmHg. In 70% RAS status, the systolic blood pressure is 156 mmHg and the diastolic one is 102 mmHg. The 90% RAS is the dangerous condition as the graph shows that the hypertension is 218/142 mmHg. According to the Seventh Report of Joint National Committee, we can define categories of the hypertension caused by RAS as follows: RAS of 20% and 50% hypertension are defined prehypertension. The 70% RAS hypertension is defined stage 1, meanwhile, the 90% RAS hypertension is stage 2, a severe hypertension that is usually prone to stroke and heart failure (NIH, 2003).

Comparison the obtained graphic result to clinical observation

Renal artery disease can cause of narrowing of the vessel lumen (stenosis). The reduced lumen diameter increases the pressure drop along the length of the diseased artery, which decreases the pressure at the afferent arteriole in the kidney. This hypertension is called secondary hypertension in known causes such as renal artery. Reduced arteriolar blood pressure and reduced renal perfusion stimulate renin release by the kidney. It causes to increase the circulating of angiotensin II (All) and aldosterone. These hormones raise blood volume by enhancing renal reabsorption of sodium and water. Increased All causes systemic vasoconstriction and enhances sympathetic activity. Chronic elevation of All promotes cardiac and vascular hypertrophy. The net effect of these renal mechanisms is an increase in blood volume that augments cardiac output by the Frank-Starling mechanism (Klabunde, 2003a; Spinowitz, 2007).

Changes in blood volume affect arterial blood pressure by changing cardiac output. An increase in blood volume increases central venous blood pressure. This increases right arterial blood pressure, right ventricular enddiastolic blood pressure and volume. The increase in ventricular preload raises ventricular stroke volume by the Frank-Starling mechanism. An increase in right ventricular stroke volume increases pulmonary venous blood flow to the left ventricular, thereby increasing left ventricular preload and stroke volume. An increase in stroke volume then increases cardiac output and arterial blood pressure (Klabunde, 2003b; Klabunde, 2007a).

Therefore, hypertension caused by RAS results from both an increase in systemic vascular resistance and an increase in cardiac output (Klabunde, 2007b). We could not have modeled the effect of hormones and nerve in our model, so the hypertension is just caused by the increase in arterial resistance and the stroke volume would not change in this modeling. We would imply that the modeling results are in a good agreement with the clinical data (Klabunde, 2007b).

Besides, the Mean Arterial Pressure (MAP) which is the principal hemodynamic factor could be understood from the diagrams easily (Klabunde, 2003c).

Conclusion

This study is a tool for students in order to see the cardiovascular system operation in normal condition or abnormal.

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