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Analog simulation of aortic and of mitral regurgitation

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ABSTRACT

By using an equivalent electronic circuit either mitral or aortic regurgitation was simulated. Simulation allowed not only a measurement of various pressures within the cardiovascular system and cardiac output, but also mitral and aortic flow.

In normal conditions mitral and aortic flows were monophasic, anterograde. In valve regurgitation mitral and aortic flows were, as expected, biphasic.

In mitral regurgitation, during systole and diastole the valve flow was retrograde and anterograde, respectively.

In aortic regurgitation, during systole and diastole the valve flow was anterograde and retrograde, respectively.

The magnitude of the regurgitant valve flow was measured by time-integration and compared to the net flow, i.e. cardiac output. Valve flow was determined not only by the magnitude of valve dysfunction, but also by the resistive/capacitive characteristics of the "falsely" attached regurgitant circuit. If the regurgitant valve flow was large enough, it in turn affected the function of the left ventricle.

The present investigation suggests that many features observed in patients with mitral or aortic regurgitation can be qualitatively satisfactorily simulated. In some respects even quantitative simulation is possible. However, for simulation of chronic mitral or aortic regurgitation, in the analog electronic circuit additional adjustments—in capacitance of the left ventricle and pulmonary system—would be required.

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1. Introduction

It is well known that various physiological processes can be simulated by using digital or analog approaches [1,2]. Recently it has been shown that in simulations also electric analog circuits can be used [3–5]. However, the majority of these simulations have been devoted to studying the cardiovasular system [6–11]. In the most recent simulation clinical cardiological conditions were simulated and compared to data obtained in patients [12].

The aim of the present investigation is to study—by using the same simulation approach—the mitral and aortic regurgitation. This seems important because in this type of valve dysfunction the extra haemodynamic load is determined not only by the resistance, but also by the capacitance (i.e. "elastic" resistance) of the "falsely" attached vascular system. Thus, in aortic regurgitation the arterial system may be important. Contrary to that, in mitral regurgitation the pulmonary vascular system may play a role. This further means that the regurgitant blood flow through the defective valve, if large enough, could significantly affect the time course of ventricular contraction [13]. Therefore, it may be speculated that the *same degree of valve dysfunction* (i.e. regurgitation through the defective valve) may have quite different consequences if dysfunction occurs either at the aortic or at the mitral valve.

A study of the mechanisms described above may improve our understanding of the haemodynamic changes in mitral and aortic regurgitation.

In the present circuitry only acute changes can be studied well. But attempts should be made to compare these results with data observed in man, both in acute and in chronic conditions [14,15].

2. Methods

In the present paper the same simulation circuit is used as recently described [12]. Homeostasis is achieved by negative feedback, by clamping the mean arterial pressure cf. [16]. This approach is similar to that described by voltage-clamping the membrane potential in muscle fiber [17,18].

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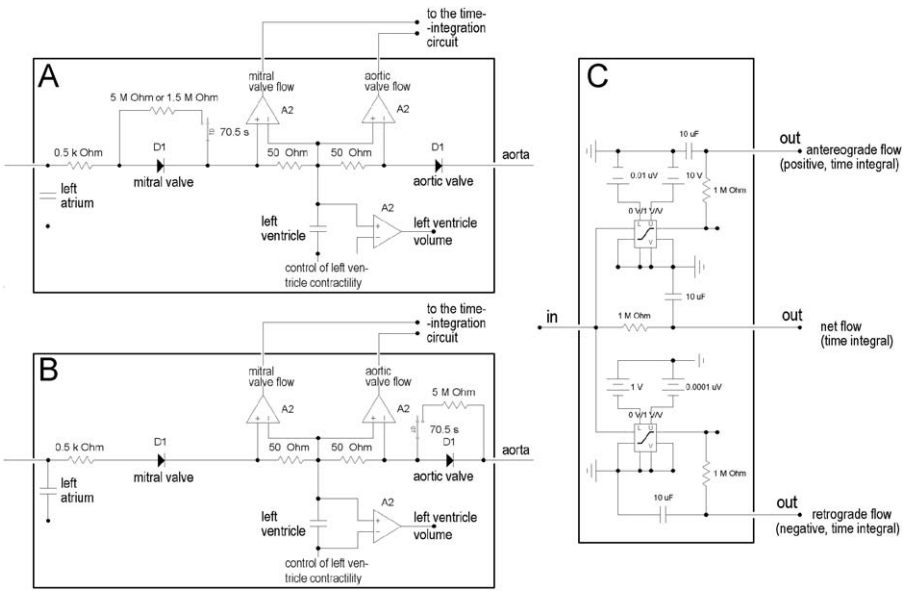

Fig. 1. Modifications to the basic analog electronic circuit (cf. [12]) required to induce valve failure and to measure the time course of valve flows and their time integrals. (A) Modifications of the left ventricle circuit to induce mitral regurgitation. Note the switch which closes at 70.5 s of simulation time, inducing a shunt across mitral valve (diode D1). Mitral and aortic valve flows are measured via operational amplifiers A2 as voltage drop across 50 Ω resistors. If required the output of A2 amplifiers is connected to the integration circuit (C). (B) Modifications of the left ventricle circuit to induce aortic regurgitation. Note the switch which closes at 70.5 s of simulation time, inducing a shunt across the aortic valve (diode D1). For further description refer to (A). (C) Time-integration circuit (time constant: 10 s) to measure valve flows. Anterograde and net flow (both positive) recorded as upward deflection, respectively. Retrograde flow (negative) recorded as downward deflection.

Minor modifications in the circuit (cf. [12]) to meet additional needs:

Normal mitral and aortic valves are simulated by diode D1. Input to the left ventricle is slightly modified (Fig. 1A and B).

In physiology, resistance to flow is expressed in arbitrary units U, i.e. (100 mmHg)/(100 ml/s). In man, total systemic and pulmonary resistance is about 1 and 0.15 U, respectively. This means that the valve shunt should be chosen accordingly. However, it seems convenient to express the shunt magnitude not only in resistance units U, but also in conductance units, U^{-1}, i.e. (100 ml/s)/(100 mmHg).

Mitral regurgitation is simulated for two conditions of regurgitation: valve shunt 2 U^{-1} (0.5 U, by placing a 5 MΩ resistor parallel to diode D1, i.e. the same shunt level as in aortic regurgitation) or valve shunt 6.7 U^{-1} (0.15 U, by placing a 1.5 MΩ resistor parallel to diode D1, i.e. approximately equal to the total pulmonary resistance; Fig. 1A).

Aortic regurgitation is simulated for one condition only: valve shunt 2 U^{-1} (0.5 U, by placing a 5 MΩ resistor parallel to diode D1, i.e. about 50% of total peripheral resistance; Fig. 1B).

In order to measure valve flows, two 50 Ω resistors are inserted into the ventricle; one, in series, at the output side of (mitral) diode D1; the other one, in series, at the input side of (aortic) diode D1. By measuring the voltage drop across these resistors the aortic and mitral flow, both anterograde (normal flow) or retrograde (reverse flow), can be measured (Fig. 1A and B). By attaching a suitable circuit the time integral of flows can be obtained (integration time constant is 10 s; Fig. 1C). In this way the anterograde, the retrograde and total valve flow can be compared with the net systemic flow, i.e. cardiac output measured at the level of capillaries and venules.

Note that in the present model atria *are not contracting*.

Analysis of the circuit is performed by using *Electronics Workbench* (EWB) Personal version 5.12 [19].

Results are expressed graphically as already described [12], as the time course of equivalent variables. Thus electrical variables: voltage, current, resistance, capacitance and charge correspond to physiological variables: pressure, blood flow, resistance, capacitance and volume (for details refer to [9,11,12]). Acronyms of variables studied are listed in Table 1.

Table 1
Recorded variables (with corresponding units) and acronyms used in text and illustrations.

Variable	Acronym
Aortic pressure (mmHg)	AoP
Cardiac output (ml/min)	CO
"Contractible" volume of veins (ml)	CVV
End-diastolic volume of left ventricle (ml)	EDVLV
End-systolic volume of left ventricle (ml)	ESVLV
End-diastolic volume of right ventricle (ml)	EDVRV
Isovolumetric contraction time (ms)	ICT
Isovolumetric relaxation time (ms)	IRT
Intrathoracic pressure (mmHg)	ITP
Left atrial pressure (mmHg)	LAtP
Left ventricular pressure (mmHg)	LVP
Left ventricular volume (ml)	LVV
Mean arterial pressure (mmHg)	MAoP
Stroke volume of the left ventricle (ml)	SVLV
Unit of resistance to flow (100 mm Hg/100 ml/s)	U
Unit of conductance to flow (100 ml/s/100 mmHg)	U^{-1}

In some simulations it seems desirable to show not only the time course of LAtP, but also its mean value (i.e. its time integral). To meet this end a time-integration subcircuit is designed. It is the same as that used for AoP integration, except that its time constant is 20 s.

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