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Object-oriented Modeling of Renal Autoregulation for a Cardiovascular System

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Abstract: Renal autoregulation maintains blood flow over a wide range of perfusion pressures despite pronounced nonlinear vessel mechanics and neurogenic influences. In lumped-parameter cardiovascular models, vascular beds are often represented by linear resistances, which fail to capture characteristic pressure-flow relations and myogenic behavior. In this contribution, a nonlinear representation of the renal vascular resistance is proposed using a MOSFET-based circuit analogy. The model reproduces pressure-dependent flow limitation, a critical closing pressure, and shifts in the pressure-flow characteristic under varying sympathetic activation. Additionally, a simple control structure is introduced combining a fast pressure-driven myogenic response with a slow adaptive mechanism restoring renal blood flow.

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I. Introduction

Autoregulation of renal blood flow is a key physiological mechanism ensuring stable organ perfusion despite fluctuations in arterial pressure [1]. Experimental pressure-flow measurements reveal a pronounced nonlinearity characterized by flow saturation, pressure-dependent conductance, and a critical closing pressure that increases with sympathetic activation [2]. In lumped-parameter cardiovascular models, such behavior is often approximated by linear resistive elements, limiting the ability to reproduce autoregulatory dynamics and static transfer characteristics [3]. The objective of this work is 1) to introduce a nonlinear flow resistance suitable for lumped-parameter models that reproduces experimentally observed pressure-flow relations, and 2) to demonstrate how a simple control concept can emulate renal autoregulation without explicitly modeling all underlying physiological mechanisms.

II. Material and methods

Fig. 1 illustrates the qualitative pressure-flow relationship of arterioles under varying sympathetic activation. Compared to a rigid tube, real vessels exhibit a nonlinear characteristic with a finite pressure threshold below which flow ceases. Increasing sympathetic tone shifts the curve toward higher pressures while reducing effective conductance.

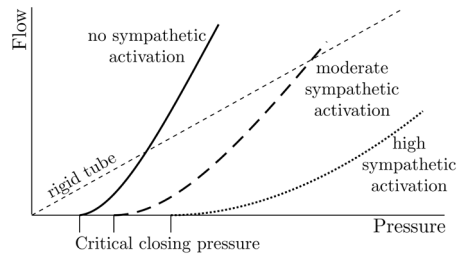


Figure 1: Qualitative pressure-flow characteristics of arterioles.

This behavior cannot be represented by a single linear resistance. Instead, a nonlinear element is required that allows pressure-dependent conductance, a critical closing pressure, and modulation by sympathetic activation.

II.I. MOSFET-Based Nonlinear Resistance

To reproduce this behavior, the renal vascular bed is modeled using an extended MOSFET-based circuit analogy implemented as an active load (cf. Fig. 2). In this representation pressure corresponds to voltage, flow corresponds to current, and vascular tone modulates the effective channel conductance. The total systemic resistance was assumed to be r = 1.0 mmHg s cm^{-3}, with a cardiac output of 5.5 L/min under physiological conditions. The renal vascular bed was assigned a baseline fraction of systemic conductance $r_{rel,kidney}$ = 1.2/5.5 reflecting an average renal perfusion of approximately 1.2 L/min that is maintained by autoregulatory mechanisms.

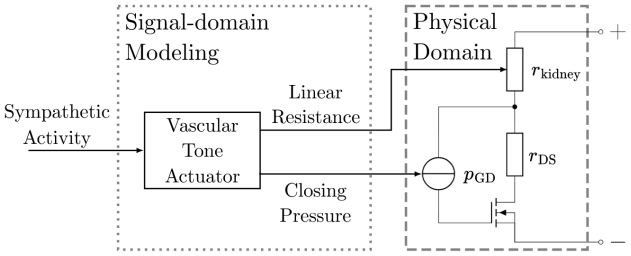


Figure 2: Object-oriented Simscape implementation of the nonlinear renal vascular resistance. The signal-domain block Vascular Tone Actuator maps sympathetic activity to two physiological control actions to the renal vascular bed.

Fig. 3 shows the simulated pressure-flow characteristics of the MOSFET-based plant for different sympathetic activation levels. The resulting curves closely resemble the qualitative behavior depicted in Fig. 1, including a progressive shift of the critical closing pressure and a reduction in slope at higher activation levels.

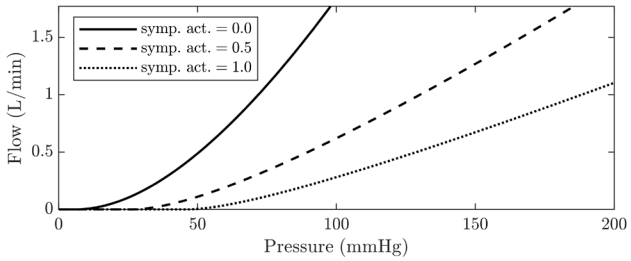


Figure 3: Simulated pressure-flow characteristics of the MOSFET plant for varying sympathetic activation.

II.II. Autoregulation Control Concept

Renal autoregulation arises from multiple mechanisms acting on different time scales. The myogenic response reacts rapidly to changes in transmural pressure, whereas slower mechanisms, such as metabolic and renal feedback (e.g. Renin-Angiotensin system), adjust vascular tone to maintain an adequate flow level. Rather than explicitly modeling each mechanism, a phenomenological control structure is adopted that captures their net effect.

The overall control input u(t), representing sympathetic vascular tone, is defined as the superposition of two components:

$$u(t) = u_{myo}(t) + u_{ad}(t), \tag{1}$$

where the myogenic component (fast) is represented by

$$u_{myo}(t) = K_P \cdot (p(t) - p_0), \tag{2}$$

where p(t) is the renal perfusion pressure and p_0 is the reference pressure and the adaptive component (slow) can be expressed by

$$\dot{u}_{ad}(t) = K_I \cdot \Big(Q_{ref} - Q(t)\Big), \tag{3}$$

where Q(t) is renal blood flow and Q_{ref} the reference flow. Physiological actuator dynamics are enforced using a first-order lag, rate limiting, and saturation. Anti-windup is applied to the integrator to ensure stable operation within the admissible range of vascular tone.

III. Results and Discussion

Fig. 4 shows the simulated renal blood flow in response to a pressure step from 80 mmHg to 120 mmHg and back with a target renal flow of 1.1 L/min.

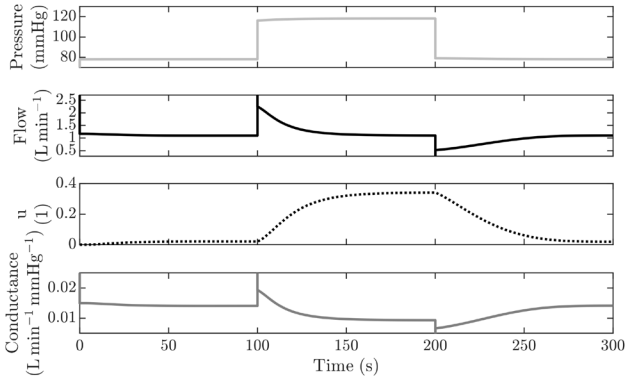


Figure 4: Renal blood flow response to a pressure step from 80 to 120 mmHg under closed-loop autoregulation.

Immediately after the pressure increase, flow rises due to the passive hydraulic effect. Subsequently, the myogenic component induces vasoconstriction, reducing flow toward its reference level. The resulting transient behavior is qualitatively consistent with experimental observations reported in the literature of renal autoregulation, exhibiting an initial overshoot followed by gradual recovery [4].

Fig. 5 depicts the steady-state pressure-flow relationship of the closed-loop system. Over a wide pressure range, renal blood flow remains approximately constant, demonstrating effective autoregulation. At low pressures, flow collapses due to the critical closing pressure, while at higher pressures conductance is actively reduced.

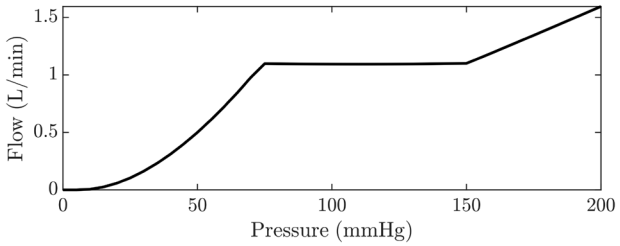


Figure 5: Steady-state pressure-flow relationship demonstrating renal autoregulation.

The presented model demonstrates that essential features of renal autoregulation can be reproduced using a simple nonlinear resistance combined with a phenomenological control structure. While the adaptive component is not intended to represent a specific physiological pathway, it captures the aggregated effect of slow regulatory mechanisms. Compared to direct flow control, the proposed structure preserves a pressure-driven myogenic response, resulting in more realistic transient behavior and pressure-flow characteristics. The model is intended for the physiological operating range of renal autoregulation and does not represent extreme pathological conditions.

IV. Conclusions

A nonlinear MOSFET-based resistance model and a two-path control concept were introduced for modeling renal autoregulation in lumped-parameter cardiovascular models. The approach reproduces key static and dynamic features observed experimentally while remaining computationally efficient and conceptually simple. Future work will focus on parameter identification and extension to other vascular beds.

AUTHOR'S STATEMENT

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