A Mathematical Model Of Transient Adjustments During Renal Autoregulation

Reprinted by special permission of the Symposium on Biomedical Engineering.

P. M. Hutchins, B.S. F. L. Thurstone, Ph.D.
The Bowman Gray School of Medicine
Winston-Salem, North Carolina 27103 Wake Forest College

ABSTRACT

This report describes a model that may be used in the evaluation of the mechanism(s) of action engaged in renal autoregulation of blood flow. This model was derived from both the steady-state and transient behavior of the renal autoregulatory response. The response computed by an analog computer simulation of the model compared favorably to those observed in experimental animals.

I. INTRODUCTION

The ability of the kidney to maintain a constant blood flow and glomerular filtration rate in the face of variations in perfusion pressure has been the subject of much investigation. Many theories have been proposed to explain this autoregulation of blood flow. Of all the hypotheses attempting of elucidate the mode of action, only two have not been discredited to a large extent.

The osmotic feedback theory, as suggested by Guyton (Ref. 1), has been in existance too short a time to have undergone careful scrutiny.

The myogenic theory, as proposed to exist in the muscle bed by Folkow and adapted to the renal vasculature by Waugh, has endured because of: (1) the difficulty of separating the smooth muscle of the renal afferent arteriole from surrounding influences, and thus noting only the effects of variations in transmural pressure; and (2) indirect evidence (Ref. 2). It is very possible that myogenic action may be found to be the effector of renal blood flow autoregulation. At present, however, no positive evidence that this is the situation has been demonstrated. In order to obtain evidence either for or against this hypothesis, it is seen that a new experimental approach is needed. To indicate the direction of such an approach, a model of the system to be studied would be of obvious help.

II. THE MODEL

Utilizing the steady-state and transient characteristics of a renal autoregulatory adjustment, a model relating vessel radius and wall tension was derived. (Ref. 3) This model is shown in Fig. 1.

Tc, Ts, Tv, and Tm are the tensions due to the contractile, spring, viscous, and inertial elements, respectively, and TA is the tension producted by the transmural pressure at the level of the afferent arteriole.

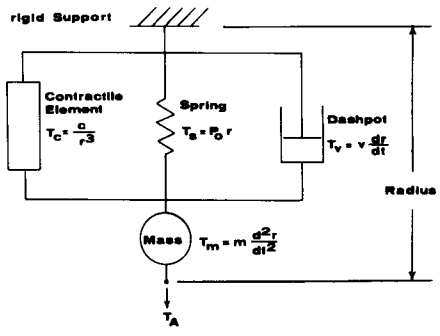

Figure 1
THE MODEL

The equation describing this model is:

$$TA = \frac{C}{r3} + Por - \frac{v\,dr}{dt} - \frac{m\,d2r}{dt2}$$

This equation is a non-linear, second-order differential equation, the solution of which cannot be expressed in closed form.

III. RESULTS

The model was evaluated by comparing transient, autoregulatory flow paterns of experimental animals, in which step changes in renal artery pressure had been produced, to the responses generated by an analog computer, programed to simulate the model. A representative comparison is illustrated in Fig. 2.

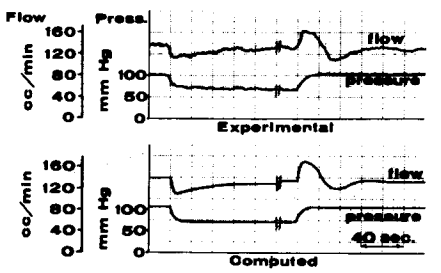

Figure 2
TRANSIENT AUTOREGULATORY PATTERNS

The values of c and Po were obtained from the steady-state characteristics of the particular response pattern to be simulated. The constants m and v were adjusted until a best fit was obtained. In general, an increase in v damped the oscillation and extended the period of oscillation, whereas an increase in m heightened the oscillation and also lengthened the period.

Due to the non-linear nature of the contractile element, the frequency of oscillation decreased with time after the simulated step change in pressure. Thus the system had no natural frequency.

In the experiments compared with the model, it was found that a slightly dilated bed would have a lower mass and viscosity on contraction than a more constricted bed. The more dilated bed also had a higher viscosity on relaxation, indicating that it was unable to dilate rapidly. In all beds, viscosity during relaxation was greated than the viscosity value during contraction. This is in agreement with the known fact that some smooth muscles contract faster than they relax.

IV. CONCLUSIONS

This is neither evidence for nor against the myogenic hypothesis, but merely shows that a model built around the hypothesis can produce the necessary responses. It is hoped, however, that by utilizing this model in an experimental design or the analysis of data, conclusions can be reached concerning the validity of such a mechanism and its value to the renal bed.

V. ACKNOWLEDGMENTS

This work was supported by PHS grants GM 1044 and GM 32,046. The assistance of Drs. H. D. Green and H. E. Schmid, Jr., in obtaining and interpreting the experimental data is gratefully acknowledged.

REFERENCES

1. Guyton, A. C., Langston, J. B., and Navar, G.: Theory for renal auto-regulation by feedback at the juxtaglomerular apparatus. Circ. Res. (Supp. I) 15:I-187, 1964.
2. International symposium on autoregulation of blood flow, Indianapolis, Ind., Circ. Res. 14 & 15 (Supp. I), 1964.
3. Hutchins, Phillip M.: A mathematical model of transient adjustments during renal autoregulation. Master's thesis, The Bowman Gray School of Medicine of Wake Forest College, 1966.