LETTERS TO THE EDITOR

Comments on "Diastolic-Systolic Coronary Flow Differences Are Caused by Intramyocardial Pump Action in the Anesthetized Dog" which appeared in Circ. Res. 49: 584–593, 1981

In recent years, the effect of ventricular systole on coronary blood flow has usually been understood in terms of the vascular waterfall concept, the major premise of which is that coronary vessels will display the hydraulic characteristics of collapsible tubes when subjected to extramural compression (Downey and Kirk, 1975). Spaan et al. (1981) have suggested an intriguing alternative explanation which they call the intramyocardial pump. They believe that flow decreases in systole because ventricular contraction forces blood from intramyocardial vessels into upstream arteries, thereby inhibiting antegrade flow. Flow increases in diastole because blood surges back into these previously compressed vessels when the ventricle relaxes. This to-and-fro motion causes the phasic flow differences during the cardiac cycle.

Spaan and colleagues support their argument by claiming that portions of their data are incompatible with the waterfall concept. For instance, the authors state that backflow is not possible in the waterfall model because “—the diodes in the electrical analog model of Downey and Kirk, which are essential for the working of their model, would actually prevent such a flow.” Surely, it is not too much to say that the attitude apparent in this statement shows an extravagant respect for the heuristic power of an electrical analogy. If the waterfall model actually describes reality, it is because coronary vessels behave like collapsible tubes. Whether or not they behave like diodes is irrelevant.

Furthermore, the authors argue, the waterfall model can not explain the data on phasic and mean coronary resistance shown in Figure 6. Putting aside the question as to why these data support the intramyocardial pump and not the waterfall concept, the question remains as to how one calculates resistance “by the waterfall model without diodes.” I assume that Spaan and colleagues use diodes to represent the coronary back pressure. Does “without diodes” mean that the back pressure is zero? What is a waterfall model with zero back pressure?

I am concerned that Spaan and colleagues misconstrue the waterfall model. In their introduction, they wrote: "If extravascular compression were to affect coronary flow, solely by a time-varying resistance..." The specific context in which this phrase appears gives one no choice but to believe that this is how the authors view the workings of a putative vascular waterfall. When Downey and Kirk (1975) used the waterfall concept to explain the effect of ventricular systole, they explicitly stated that the increase in extravascular compression caused by systole would be apparent as an increase in the back pressure opposing coronary artery flow. The parallel pressure-flow relations they observed whenever perfusion pressure exceeded peak ventricular pressure are prima facie evidence that their waterfall model was applicable. Systole decreased coronary flow by increasing the back pressure, not by changing coronary resistance.

Of course, the situation in the beating heart generating its own perfusion pressure is more complicated. Since systolic intramyocardial pressure in the left ventricular subendocardium exceeds local microvascular pressure, vessels that were perfused during diastole will be emptied of blood in the way described by Spaan and colleagues. It seems reasonable to assume that these vessels will not be perfused so that the total cross-section of the bed will be smaller and resistance correspondingly greater than in diastole. Thus, the mechanism for flow reduction in systole viewed in terms of the waterfall concept will involve both an increase in the back pressure of perfused vessels and an increase in resistance.

Spaan and colleagues would no doubt disagree with this assessment, but the full consequences of their intramyocardial pump, apparent in the following “thought experiment,” should be considered. Given: a maximally vasodilated coronary bed and a constant perfusion pressure. Let ventricular systole be prolonged until all flow transients associated with intramyocardial pumping have passed. What would be the level of steady state systolic flow? I believe that the authors would answer mean flow. Now let diastole be prolonged until all transients have passed. What would be the level of steady state diastolic flow? Would it be also equal mean flow? Although distinctly counterintuitive, the conclusion is inescapable that, during the steady state, systolic and diastolic flows (and pressures) are identical. I wonder if Spaan and colleagues are really prepared to argue that ventricular systole has no effect whatsoever on coronary flow when transients associated with their intramyocardial pump have disappeared. As I see it, the problem the authors should address is the extent to which flow transients from intramyocardial pumping obscure the real inhibiting effects of ventricular systole on coronary blood flow.

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Reply to the Preceding Letter

In Dr. Bellamy’s Letter to the Editor, we distinguish three main “charges”: (1) Our attitude towards electrical models keeps us from understanding the physical problem; (2) we misconstrue the waterfall model of Downey and Kirk (1975); (3) the intramyocardial pump model would not be able to explain the influence of cardiac contraction on mean coronary flow. Let us reply to these points in order.

One should probably make the distinction between waterfall phenomena and the electrical analog of Downey and Kirk (1975). However, a physical concept is only useful when put in a form such that it can be tested. To our knowledge, the model of Downey and Kirk is the only waterfall model exhibiting this merit. We formulated the intramyocardial pump model when we found our experimental results to be inconsistent with the waterfall model of Downey and Kirk. The type of analogy chosen for the models, electrical or hydraulic, is not relevant to this point. We could not find an alternative explanation of our experimental results based on the concept of collapsible nonelastic tubes.

“We misconstrue the waterfall model of Downey and Kirk.” This criticism is based on a fragment from the introduction of our paper and on our calculations of the relationship between the diastolic-systolic differences of coronary arterial pressure and flow at varying stenosis grade in the perfusion line. The quoted part of the introduction was not only directed to waterfall models but also to models in which the lower systolic flow is explained by an extravascular resistance component. The waterfall model predicts cessation of endocardial flow during systole when tissue pressure exceeds arterial pressure. So the overall systolic resistance of the coronary bed has increased with respect to diastole. However, we admit that a time-varying resistance concept is not applicable to the waterfall model when endocardial tissue pressures remain smaller than arterial pressures. In the discussion of our paper however, there is no reason for confusion. When we state “without diodes,” we simply mean “without diodes.” The backpressure is unaltered. Consequently, the conclusion from Figure 6 of our paper, that the waterfall model predicts larger a.c. resistances than measured, is correct. This means that the diastolic-systolic flow differences measured at constant perfusion pressure are larger (approximately 40%) than predicted by the waterfall model.

The third comment concerns the relevance of the intramyocardial pump model for the prediction of the influence of systole on the mean flow. We have treated this point very carefully in the discussion of our paper. Dr. Bellamy expects us to conclude, from our intramyocardial pump model, that coronary flow is equal during systole and diastole when both are of imaginary infinite duration. Well, let us reply by a few quotations from our paper.

A. “The presence of a large capacitance in the model makes it impossible to draw conclusions concerning the phasic aspect of the venous side of the circuit on the basis of arterial measurements. For example, transient occlusion of small veins as implied by the waterfall model are invisible to an analysis of phasic arterial pressure and flow.”

B. “So, the waterfall might still hold as a concept for how the contracting myocardium influences mean coronary flow.”

C. “...in the arrested state, the pressure difference between the vessel lumen and the surrounding tissue is larger, and as a consequence of the capacitance of the intramyocardial blood compartment, its volume will be increased. It is therefore to be expected that the resistance of the intramyocardial vessels decreases in cardiac arrest.”

We would like to add two more points to our reply. Downey and Kirk found a parallel shift of coronary arterial mean pressure-mean flow lines, when arresting the heart. A simple explanation of this shift is the waterfall model. As shown above, we do not exclude this possibility. However, other explanations are possible. “Prima-facie” evidence can be misleading. We showed earlier (Spaan, 1979; Spaan and Laird, 1981) that a linear diastolic pressure-flow line does not necessarily imply a constant diastolic resistance.

Moreover, we would like to underline a paramount conclusion of our paper. In a normal beating heart, systole and diastole are just too short to establish systolic and diastolic steady states. A shift of only 3% of intramural arterial volume between diastole and systole is sufficient to explain the diastolic-systolic flow differences. Apparently, the intramyocardial vessels are compliant enough to allow these volume changes without significant influence on intramyocardial systolic and diastolic blood pressures. Let us state, clearly, that we are not the first in showing a compliance of the intramural blood compartment. We cite in our paper three more sources, the first from 1961. All four independent estimations of capacitance values are within 15% of each other. So, an important question has now been raised for all those models of coronary circulation describing time-varying phenomena. Those models should incorporate the intramyocardial capacitance. This is not only the case when describing coronary blood flow in the beating heart, but also during long diastoles, especially when perfusion pressure changes with time.

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