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Editorial

Hemodynamic Evaluation of Vasomotion: Capacitance vs Conductance

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When a young medical or physiological student is first introduced to the cardiovascular system, one of the first diagrams that he or she will see is that of a simple electrical circuit composed of a battery coupled to an array of variable resistors, all arranged in parallel. The student will then be taught Poiseuille's Law, which will be identified as the hydraulic equivalent of Ohm's Law; both laws defining 'resistance' as the ratio of a pressure or voltage difference to a flow or current. It is then suggested that the student memorize the fact that the reciprocal of the resistance of a parallel circuit (ie, systemic vascular resistance) is equal to the sum of the reciprocals of the resistance of each parallel component. Although correct, this is complex, nonintuitive, and fails to promote any deeper understanding. Having had such introductory instruction, it might never occur to the maturing physician or cardiovascular scientist that there is any other way of characterizing the relations between pressure and flow.

In general, physical variables can be classified as either 'extensive' or 'intensive.' Extensive variables include volume (and its time derivative, flow), heat, charge (and current), and conductance.[1-4] The question, 'How much?' can be asked about extensive variables. Intensive variables (respectively) include pressure, temperature, voltage, and resistance. Importantly, it is not equally meaningful to ask 'How much?' about these variables but only 'To what degree?' For example, one can say that 2 lungs have twice the conductance of 1 lung; it is less meaningful or useful to say that 2 lungs have half the resistance of 1 lung. Investigators who study the characteristics of membrane channels uniformly use conductance rather than resistance. (The metric system is a great boon with respect to calculations with extensive variables—compare solving physics problems using BTU's rather than SI units—but the metric system is quite irrelevant with respect to calculations with intensive variables.)

The difference between conductance and resistance becomes especially important when evaluating the effects of vasoactive agents. For example, a vasodilator relaxes vascular smooth muscle and thereby increases the calibre of venous or arterial blood vessels. Like changes in capacitance, changes in conductance (unlike changes in resistance) vary directly with changes in length of vascular smooth muscle. An increase in the calibre of veins increases their capacitance (ie, the volume of blood contained at a given pressure,[5] which affects cardiac preload). The increase in its calibre also increases their conductance but, because the total pressure decrease across the venous system is so small compared with the total arteriovenous pressure difference, this effect can be ignored. An increase in the calibre of arteries increases their conductance, which affects left ventricular afterload. It also increases their capacitance,[6,7] but because there is much more blood in the venous system, changes in arterial capacitance do not substantially affect cardiac preload.

In anaesthetized dogs, using a radionuclide method to measure changes in splanchnic blood volume[8] in our laboratory, Wang et al. measured the changes in venous capacitance induced by acute heart failure (microsphere embolization of the left coronary system) and the subsequent changes effected by 3 vasodilators, hydralazine, enalaprilat, and nitroglycerin.[9] As shown in Figure 1A, microsphere embolization[10] decreased venous capacitance by shifting the splanchnic venous pressure-volume curve to the left by almost 20%. This showed how veins can constrict to reduce peripheral venous pooling and thereby increase cardiac preload.[11] Using the splanchnic pressure-volume relation recorded after heart failure as a new basis of comparison, they reported that hydralazine increased venous capacitance by $<$ 5% (Fig. 1B). Enalaprilat increased venous capacitance by approximately 15%, nearly matching the pre-embolization value of venous capacitance. Nitroglycerin increased venous capacitance by 40%, substantively exceeding pre-embolization venous capacitance. To compare capacitance effects with conductance effects, systemic conductance (cardiac output divided by the arteriovenous pressure difference) was calculated.[12] Figure 1C shows the comparative, capacitance-conductance effects of the 3 agents. As anticipated, the capacitance effects of nitroglycerin were dominant, as were the conductance effects of hydralazine.

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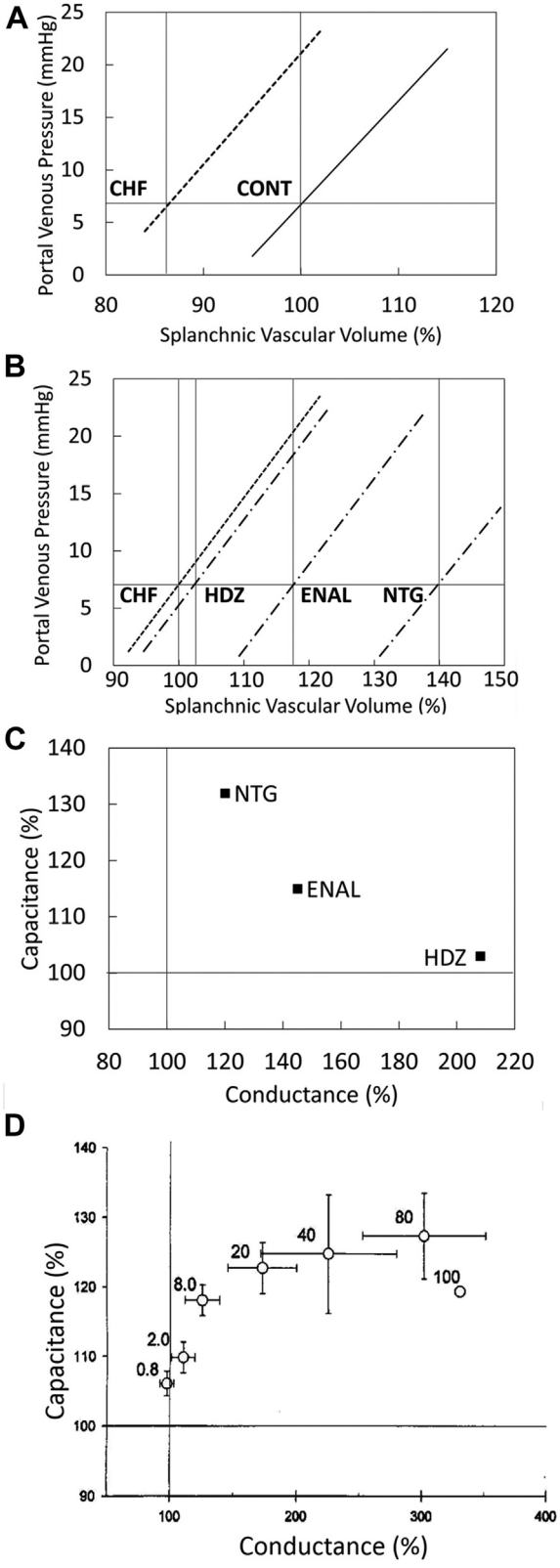

Figure 1. (**A**) Venous capacitance decreases by approximately 15% after induction of congestive heart failure (CHF). Arbitrarily, volumes were compared at a portal venous pressure of 7 mm Hg. Data from Wang et al.[9] (**B**) Effects of hydralazine (HDZ), enalaprilat (ENAL), and nitroglycerin (NTG) on venous capacitance in the presence of CHF. Data from Wang et al.[9] (**C**) Comparative capacitance-conductance effects of HDZ, ENAL, and NTG on venous capacitance in the presence of CHF. Data from Semeniuk et al.[12] (**D**) Comparative capacitance-conductance effects of increasing infusion dosages (μg/kg/min) of NTG on venous capacitance in the presence of CHF. Reprinted from Chihara et al.[13] with permission from Elsevier. Cont, control.

To compare the capacitance-conductance effects of nitroglycerin as a function of increasing dosage, Chihara et al. used the same experimental model.[13] As shown in Figure 1D, at low doses the capacitance effects of nitroglycerin were dominant. However, as the dose was increased further, the incremental increase in capacitance was modest and the effects on systemic arterial conductance were very large (at the highest doses, for a given arteriovenous pressure difference, systemic arterial flow increased 3-fold).

The physiology of vasovagal syncope provides an interesting insight into the conceptual struggle between venous capacitance and arterial conductance as the cause of hypotension.[14,15] Common-faint patients have usually been upright for a while, then develop progressive presyncope, often ending with visual blurring and syncope. Many patients notice a rapid hot flush preceding syncope. Patients can abort the progression from presyncope to syncope by lying down, squatting, or isometrically contracting lower limb muscles.

Throughout the mid-20th century the dominant concept was that progressive presyncope was due to increases in arterial conductance and therefore hypotension. This was on the basis of numerous experiments, mostly either focused on specific muscle beds or with sampling intervals too long to detect events unfolding over seconds to a few minutes.[16] This was first challenged by Manyari et al.,[17] who noted that patients with vasovagal syncope had abnormal and diminished forearm venoconstriction during mental stress. This led to the hypothesis that hypotension during progressive presyncope might be due to impaired venoconstriction, increased capacitance, and decreased cardiac preload.

Reports in the past 20 years have repeatedly challenged the increased-conductance concept, at least as the sole cause of vasovagal syncope.[14,15] Agreement now exists that the first and prolonged phase of common faint is reduced preload, most likely due to a failure of venous capacitance to respond to the challenge of orthostatic stress. The terminal phase in most patients might indeed be an abrupt increase in arterial conductance, although in a minority it is a further decrease in cardiac output due to profound bradycardia.

This concept elegantly explains the clinical symptoms. The early presyncope is due to decreased preload and cardiac output, causing progressive hypotension. The visual blurring and blacking out is due to either occipital lobe or retinal artery hypotension, and the penultimate final flush is due to increased arterial conductance in cutaneous beds.

The fight-or-flight reaction probably involves constriction of veins (increasing preload) and arteries (increasing perfusion pressure). Again, it might prove to be of more than academic interest to know the degree to which both effects are involved.

Vasodilators cause vascular smooth muscle to lengthen and vasoconstrictors cause vascular smooth muscle to shorten. Changes in capacitance are directly related to changes in venous smooth muscle length and changes in conductance are

directly related to changes in arterial smooth muscle length. No vasoactive agent is completely specific in that it affects only veins or only arteries. For optimal therapeutic results, the physician should know the relative potency of each agent on veins and on arteries.

Disclosures

The authors have no conflicts of interest to disclose.

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