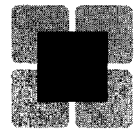

Hypertension revisited: the concept of relative hypertension in congestive heart failure

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■ Although hypertension has been defined numerically as blood pressure levels above 140/90 mmHg, there is no doubt that a blood pressure level within the "normal" range increases the stress on a diseased heart. For this reason, vasodilator therapy is often successful in treating congestive heart failure. The authors propose the term *relative hypertension* to illustrate the latter situation.

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ACCORDING to the World Health Organization Expert Committee on Hypertension,[1] normal adult blood pressure refers to systolic levels equal to or below 140 mmHg and diastolic levels (fifth phase of Korotkoff sounds) equal to or below 90 mmHg. Blood pressure levels between 140/90 and 160/95 are defined as *borderline hypertension*, whereas a diastolic blood pressure between 95 mmHg and 104 mmHg reflects *mild hypertension*. Moreover, the Framingham study[2] showed a 9.5% average annual incidence of cardiovascular death in men aged 45–54 years with blood pressure levels greater than 160/95 mmHg. This incidence was 4% when blood pressure was between 140/90 mmHg and 160/95 mmHg and 2.9% for those with pressures less than 140/90 mmHg. Based on these criteria and findings, blood pressure control has helped reduce cardiovascular complications such as strokes,[3–8] left ventricular hypertrophy, and heart failure.[3,9–11]

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Nevertheless, the current definition of hypertension needs revising to take the status of the heart into consideration. The standard definition of hypertension applies to the blood pressure level when cardiac function is normal. It omits the whole sector of cardiac dysfunction in which a normal (by standard definition) pressure will generate high stress on the dilated left ventricle. Moreover, during treatment of hypertension, normalization (by standard definition) of blood pressure may compromise coronary perfusion when left ventricular hypertrophy and/or coronary artery disease is present.[12] On the other hand, low levels of systolic blood pressure that could be otherwise described as "hypotension" are well tolerated by patients with heart failure treated with vasodilators.

THE CONCEPT OF AFTERLOAD AND LEFT VENTRICULAR STRESS

Afterload has traditionally been defined as the level of diastolic blood pressure or mean arterial pressure.[13] It is evident, however, that the correct determination of ventricular afterload includes left ventricular pressure, inner radius, and wall thickness.[14] In particular, peak systolic stress and end-systolic stress have been used extensively in recent years as measures of afterload.[15–17]

The widespread measurement of afterload has been

TABLE 1
RELATIONSHIP BETWEEN SYSTEMIC BLOOD PRESSURE AND LEFT VENTRICULAR (LV) WALL STRESS IN NORMAL, DILATED, AND HYPERTROPHIED HEARTS

	Normal	Dilated	Hypertrophied
Hypertension (150/100 mmHg)			
LV wall thickness	N	↓	↑
LV internal diameter	N	↑	↓
LV stress	↑	↑↑↑	N or ↓
Normotension (120/80 mmHg)			
LV wall thickness	N	↓	↑
LV internal diameter	N	↑	↓
LV stress	N	↑↑	N or ↓
Hypotension (90/65 mmHg)			
LV wall thickness	N	↓	↑
LV internal diameter	N	↑	↓
LV stress	↓	↑ or N	↓↓↓

N = normal

fostered by the availability of noninvasive techniques to determine left ventricular chamber dimensions.[15] Also, it has been demonstrated repeatedly that auscultatory brachial systolic pressure is closely related to left ventricular systolic pressure in the absence of outflow tract obstruction.[15,18] This noninvasive approach has allowed left ventricular systolic pump performance to be evaluated relative to the mechanical load imposed on the heart. Thus, an inverse correlation was found between indices of left ventricular systolic function (such as percent fractional shortening of the left ventricle) and end-systolic stress,[16,17] corroborating the value of therapeutic reduction of afterload in patients with impaired cardiac performance.

According to this relationship (*Table 1*), left ventricular hypertrophy has been subdivided into *compensatory* (with normal left ventricular stress), *inadequate* (with increased left ventricular stress), and *inappropriate* (with decreased left ventricular stress) hypertrophy.[19] The dilated heart may be subdivided into the categories *compensated* (with normal left ventricular stress) or *inadequate* (with increased left ventricular stress). The important information provided by such an evaluation balances to a great extent the cost of the test.

LEFT VENTRICULAR DILATION, CONGESTIVE HEART FAILURE, AND BLOOD PRESSURE

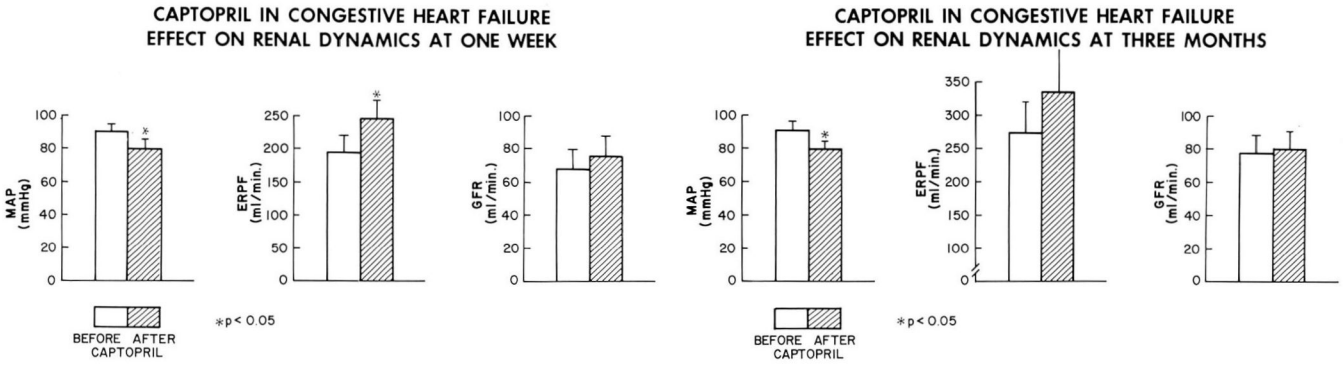
The arteriolar vascular bed is inappropriately constricted in hypertensive patients.[20] This vasoconstriction is mediated by the renin-angiotensin system, sympathetic nervous system, vasopressin, and, possibly, other activated systemic and vascular local factors. The resultant compensatory vasoconstriction maintains vital organ tissue perfusion but inappropriately increases systemic vascular resistance. This, in turn, may depress heart function, including left ventricular stroke volume, ejection fraction, and ejection velocity. Thus, afterload reduction has been used effectively to treat heart failure, especially when other conventional forms of therapy have been ineffective or insufficient to restore full cardiovascular compensation.

Historically, arterial counterpulsation was first attempted to reduce left ventricular afterload in the treatment of left ventricular failure.[21] Later, reduction of afterload in heart failure was achieved by medical treatment with the infusion of alpha-adrenergic blockers.[22] The success of this approach was followed by the use of other arteriolar vasodilators, venodilators, or the balanced venoarteriolar dilators.[23] Acutely, and in short-term therapeutic interventions, all vasodilators seem to be equally effective in unloading the diseased heart and improving cardiac function.[24] However, the long-term effects vary among the different classes of vasodilators.[25] Converting-enzyme inhibitors have been particularly effective for this purpose; their long-term effectiveness is further supported by their additional hormonal and humoral effects.[25–27]

Surprisingly, patients being treated for heart failure tolerate very low blood pressure levels without symptoms when vasodilators are added to digitalis and diuretics. In steady-state conditions, these patients do not experience symptoms related to hypoperfusion of the brain or other organs. On the contrary, renal blood flow increases during chronic captopril therapy,[28] despite reduced systolic blood pressure levels (*Figure 1*). It is not clear at this time whether the early use of vasodilator therapy in heart failure and the chronic reduction of afterload after cardiac injury will result in preservation of cardiac function, delay in progression of heart disease to congestive heart failure, or reduction in mortality.

Still unknown are the vascular complications that could be related to the relative hypertension of congestive heart failure. For example, abnormalities of arterial compliance and increased incidence of atherosclerosis,

A, B



FIGURES 1A AND B. Changes in renal hemodynamics during chronic treatment of congestive heart failure with the converting-enzyme inhibitor, captopril. MAP = mean arterial pressure. *ERPF* = effective renal plasma flow. *GFR* = glomerular filtration rate.

stroke, and coronary artery disease have not been studied in relation to the relative hypertension of congestive heart failure.

Also, the structural arteriolar changes in heart failure do not seem to be of the same nature as those seen in hypertension. Arteriolar constriction in heart failure is adaptive whereas arteriolar constriction in hypertension is usually primary. Such differences may underscore therapeutic interventions. Whereas smooth muscle relaxation may be sufficient to control heart failure, regression of vascular hypertrophy may be necessary for persistent control of hypertension.

SUMMARY

Normotensive blood pressure in patients with dilated ventricles is associated with high afterload levels and increased left ventricular wall stress. Thus, normotensive blood pressure levels represent a form of "relative hypertension" in heart failure patients. By reducing blood pressures to subnormal levels, left ventricular wall stress and afterload may normalize.

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