**THE IMPACT OF HYPERTENSION AND ITS TREATMENT IN HEART VALVE DISEASE**

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Heart valve diseases cause chronic mechanical (volume and/or pressure) overloads. Beneficial relief of the overloads involves replacement or repair of severely dysfunctional valves, reducing impedance to chamber outflow when valves are stenotic , or reducing volume overload when valves are regurgitant. Drugs diminish overloads but act on the arterioles, far distal to the valves, reducing chamber outflow impedance without affecting valve function. Consequently, drugs provide no clinical benefit when patients with valve disease are normotensive. Hypertension also causes mechanical overload via suboptimal interaction between vascular volume and peripheral arteriolar tone. Hypertension is relieved, with clinical benefit, by drugs’ arteriolar effects. In our society valve disease often is associated with hypertension. Indeed, hypertension is a well-documented risk marker in aortic stenosis (AS), aortic regurgitation (AR) and mitral regurgitation (MR), and is a beneficially modifiable risk factor in AR and MR. However, for hypertension, effective reduction in blood pressure (BP) is relatively similarly beneficial irrespective of the drug employed. In contrast, for valve disease, the direct myocardial effects of some antihypertensives may mitigate benefits. Thus, for example, angiotensin converting enzyme inhibitors may cause deleterious clinical effects when used to treat patients with AR and, from our most recent preliminary data, patients with MR. For AR, long-acting nifedipine minimizes hypertension while reducing progression to heart failure and ventricular dysfunction in AR. No data have demonstrated benefit of any antihypertensive drug in AS. Thus, hypertension is an important consideration for patients with valve diseases; optimal strategy to minimize its effects remains to be clarified.