Coexistence of Hypertensive and Coronary Arterial Disease

THE preceding article is a cogent discussion by Prisant et al.\(^1\) of coronary heart disease, a frequently coexisting cardiovascular problem in the patient with hypertension. This association assumes great clinical significance when the coronary artery disease becomes symptomatic and the presence and extent of these two clinical conditions must be established. Of particular importance in this discussion is the acceptance of the fact that coronary artery disease (i.e., atherosclerosis) frequently coexists with hypertension, and therefore a clinical need exists for the establishment of a clear-cut diagnosis and definition of the degree and extent of vascular disease.

Of major importance to the clinical investigator concerned with further understanding the coexistence of these two independent, yet interdependent, diseases is the need to provide the means for earlier recognition of the coexistence of coronary atherosclerosis with hypertensive heart disease — even before the presence of significant atherosclerotic occlusive disease of the coronary arteries is discerned by the usual invasive diagnostic techniques.\(^2\)\(^-\)\(^3\) This objective is extremely important and implies the search for answers to the following questions:

1. What is the mechanism(s) whereby hypertension accelerates atherogenesis and the progression of atherosclerotic cardiovascular disease?
2. How may the natural history and pathophysiology of hypertensive heart disease be affected by atherosclerotic coronary artery disease? Similarly, how may the natural history and pathophysiology of coronary artery disease be affected by hypertensive diseases?
3. If left ventricular hypertrophy, per se, is an independent risk factor of cardiovascular morbidity and mortality, is this due to cardiac dysrhythmias, to the hypertension-induced ventricular hypertrophy, or to coronary vascular insufficiency (on the basis of either atherosclerosis or the physiologically relative capability of the coronary circulation to satisfy the increased oxygen demand of the hypertension)?
4. If left ventricular hypertrophy secondary to arterial hypertension is a risk factor in and of itself, could this be a clinical corollary to the silent ischemia syndrome that has been described for patients with coronary heart disease?\(^4\)
5. What is the role of antihypertensive therapy in altering the course of hypertension, the associated left ventricular hypertrophy, and the relationship of hypertension to coronary atherosclerosis and coronary arterial insufficiency?

At the present time there are no complete answers to these questions. We recognize that the explanation of the interrelationship between hypertension and atherosclerosis by simple mechanisms of pressure-mediated filling of the vascular intima with lipid\(^5\) is inadequate in the light of present knowledge of endothelium-derived pressor and depressor substances and growth factors that actively determine the metabolism of the vascular wall itself.\(^6\) This area, with many new possibilities for experimental and clinical investigations, surely will provide more information and new questions that will no doubt lead to a better understanding of the relationship of these two major and common cardiovascular problems.

It is incumbent on all clinical investigators who are concerned with pursuing a greater fundamental understanding of hypertensive heart disease to be clear about just
what is being studied and described. Is it related to the hypertensive disease in its broadest concerns? Is it the effects of hypertensive disease on the heart? Or is it the second disease, atherosclerosis, that frequently and coexistently affects the heart and the blood vessels? In this respect, it is just as important for these complicating factors to be fully appreciated as it was two decades ago when we came to realize the importance of excluding drug therapy and its influence in obtaining meaningful hemodynamic and other physiological data. In addition, it is not possible to clarify the effects of ischemia that may be associated with hypertension-induced hypertrophy and its associated increased myocardial oxygen demand if the ischemia is also produced by atherosclerosis-mediated events of the coronary arterial tree. This very difficult question has been actively (or passively) ignored or, at best, discounted in many previous studies, but as we become more committed to the diagnosis of early cardiac involvement in both diseases, we must consider atherosclerosis and hypertension as two independent factors affecting the heart. This clearly is necessary if we are going to understand the impact of one problem on the other.

The issue raised concerning left ventricular hypertrophy as a risk factor is of particular pertinence at this time. Epidemiological studies have clearly pointed out that ventricular hypertrophy is an independent risk factor. Studies from this laboratory have demonstrated the increased frequency of ventricular dysrhythmias in untreated persons with essential hypertension and associated left ventricular hypertrophy. These findings must be considered, particularly in the light of antihypertensive drug therapy, since certain side effects of these therapies may enhance that risk. Answers to these questions may help further our overall understanding of the interpretation of therapeutic trials and our understanding of the history of the two diseases, particularly as we consider the impact of the two problems on silent myocardial ischemia and sudden death in hypertension and coronary artery disease.

We have already learned that, in designing pharmacological studies involving antihypertensive drugs, we must be careful to exclude antihypertensive drug therapy for a sufficient length of time prior to initiating another agent for study. Newer and major considerations have emerged that relate to the heart and its available coronary circulation. Further, if previous therapy induced a regression of cardiac mass, we must also learn about its effects on coronary blood supply. And, conversely, when that new agent was instituted, was myocardial chemical and structural makeup the same as before administration of the first drug? We already know that in a very short time after the institution of an antihypertensive agent there may be an induced synthesis of a new ventricular isomyosin. Do different myosins require different blood supplies, and are they associated with different ventricular performance characteristics? Clearly, when the problem of coronary heart disease is presented as a fait accompli in a specific patient with problems as described by Prisant et al., the clinical management they outline is available today. But in the final analysis, if we are to make major new inroads into this common problem and to understand further the nature of the coexistence of these two diseases, the questions involve greater fundamental understanding of pathophysiology and prevention. This demands new basic and clinical information to permit the elucidation of the complex interrelationships between these two frequent clinical problems. However, in this regard, experimental counterparts are difficult to develop since atherogenesis is not a characteristic of our usual experimental hypertensive models. Thus, the question of how we can diagnose coronary heart disease in hypertensive patients seems far easier to answer than the underlying questions that this problem generates.

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