Blood Pressure Response to Treatment

Blood pressure control in the 1980s presents a different set of problems than it did in the 1960s or even in the 1970s. The reasons for the difference are many. In addition to a greater variety of antihypertensive agents, there are different goals of treatment, wider indications for antihypertensive therapy, and a heightened sensitivity to possible side-effects. All these aspects are interrelated. The adoption of lower diastolic pressures as criteria of normalcy and indications for treatment has entailed extension of therapy to larger segments of the population. This, in turn, is forcing a reevaluation of the acceptable "side-effects-to-benefits ratio," as greater numbers of asymptomatic patients with mild hypertension are now considered for treatment.

Recent studies suggest that normalization of diastolic arterial pressure to levels below 95 or even 90 mm Hg might offer a greater chance to reduce mortality and prevent complications. The adoption of these levels to define optimal blood pressure control has led to a widening of our concept of resistant hypertension; the term can no longer be restricted to severe hypertension or to return of blood pressure to pretreatment levels. Since reduction of arterial pressure from high to middle levels cannot — although beneficial — be considered "optimal" nowadays, then moderate elevations of blood pressure persistent despite therapy would qualify as "resistant hypertension." A reevaluation of our approach to that problem is probably in order.

Many antihypertensive agents will lead to some pressure reduction when treatment is initiated, but it is not easy to achieve optimal control and maintain it for long periods of time. Factors that can interfere with blood pressure control are many and the mosaic they form is reminiscent of the mosaic that subtends the development of hypertension itself. Some of these factors, such as volume expansion, were recognized early with the introduction of the first effective antihypertensive drugs; others were uncovered only recently. The overall picture emerging from the many communications in this symposium is beginning to outline a framework for the investigation of poor blood pressure response and a rational approach to the readjustment of antihypertensive therapy. This is particularly helpful as the problems of hypertension confronting the physician today go way beyond the initial evaluation and first-step of treatment of a newly discovered hypertensive patient.

Among the factors that may loom large in future choices of antihypertensive agents, one, the possibility of reversal of structural lesions, could open up new avenues in cardiovascular therapy. Regression of hypertensive cardiovascular hypertrophy has been clearly documented in both humans and experimental animals; much more research is needed on its determinants. If it is proven not to depend on blood pressure control alone, then reversal of structural alterations might become a second goal of antihypertensive therapy along with pressure reduction.
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