BASIC PRINCIPLES OF EFFECTIVE HYPERTENSION THERAPEUTICS

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Effective hypertension therapeutics depends on an accurate, detailed history and physical examination as well as on key diagnostic testing. It is imperative to know, or at least have a general feel for, the prior level of blood pressure (BP) control, intensity of therapy (eg, number of drugs), compliance with the therapeutic regimen, and knowledge of the most recent therapeutic changes. Even though medical records and accurate BP readings may not be available for new patients, careful attention to their fundoscopic (arteriolar narrowing, arteriovenous nicking, silver wiring, hemorrhages) and cardiac examinations (S4 gallop, lateral displacement of the PMI), left atrial enlargement and/or left ventricular hypertrophy (LVH) on the electrocardiogram, and other manifestations of pressure-related target-organ injury provide insight into the prior level of BP control. It is also important to realize that the higher the pre-treatment BP level, the greater the risk of precipitating target-organ ischemia (eg, coronary/cerebral ischemia) in the short term.

After appropriate risk stratification, the target BP level should be determined and prominently displayed in the chart and communicated to the patient. Antihypertensive drug monotherapy rarely, on average, gives more than 15 mm Hg systolic or 10 mm Hg diastolic placebo-adjusted BP lowering. Thus, utilizing combination anti-hypertensive drug therapy is highly desirable in patients with BP >15/10 mm Hg above goal. Therapeutic inertia should be avoided, (ie, the documentation of BP levels above goal without intensification of treatment). However, in most instances, giving long-acting antihypertensive agents ~ 4 weeks to manifest their full BP response is reasonable.

Virtually all two-drug antihypertensive single-pill combinations contain either a diuretic or calcium antagonist (CA). There is no discernible racial difference in BP response to renin angiotensin system (RAS) antagonists when combined with either a diuretic or CA. And, the average lesser response often observed in African Americans to monotherapy with these RAS antagonists is no justification for avoiding their use when indicated, for example, in persons with diabetes, chronic kidney disease (CKD), and/or systolic heart failure or when used for treatment of uncomplicated hypertension in combination with either a diuretic or CA.

Patients with depressed kidney function, albuminuria/proteinuria, obesity, diabetes, evidence of pressure-related target-organ injury (eg, LVH) manifest therapeutic resistance to antihypertensive drug therapy. Interestingly, these characteristics are present more often in African American than White hypertensives. Thus, at least part of the reason African Americans have been harder to get to goal BP has to do with these markers/mediators of therapeutic resistance. The principles of effective hypertension therapeutics, however, are not race-specific.

Diuretics are key agents in complex (>2 drug) antihypertensive drug regimens. These agents block expansion of the plasma volume that occurs in patients consuming sodium freely when treated with antihypertensive agents (eg, ACE inhibitors) that expand the venous capacitance system. The optimal use of diuretics requires matching them to the level of kidney function for efficacy and avoidance of complications such as hyperkalemia. In difficult-to-control patients more than one diuretic may be required in complex regimens to

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control plasma volume expansion and thereby control BP. In selected, difficult-to-control patients, dual CA blockade (a dihydropyridine + a non-dihydropyridine CA) has proven highly efficacious in lowering BP.

In summary, these and other topics were covered at ISHIB2009 during the Hypertension Academy sessions in an effort to provide information pertaining to greater BP lowering and optimal target-organ protection in hypertensive patients.