

## **AHA's 56th Council for High Blood Pressure Research Conference**

The American Heart Association's 56th Annual Fall Conference and Scientific Sessions of the Council for High Blood Pressure Research was held September 25-28 in Orlando, Florida. The program of this conference included oral and poster presentations submitted by clinical and basic researchers from 20 countries worldwide. A few of them, with interesting implications for treatment, are reported below.

### **No Dip in Nocturnal Blood Pressure Associated With Higher Clotting Risk**

Hypertensive patients whose blood pressure does not "dip" during the night have year-round significantly higher levels of plasma fibrinogen than those whose blood pressure does dip, Spanish researchers reported. They believe that this increase in plasma fibrinogen may account for the association between lack of nocturnal decline and an increase in end-organ-damage and cardiovascular events. Ramon C Hermida, PhD, and colleagues at the University of Santiago investigated seasonal variations in fibrinogen levels over a 1-year period in 741 patients with mild or moderate hypertension ( $\geq 140/90$  mm Hg /  $< 180/110$  mm Hg). Fibrinogen levels remained within the normal range (160-400 mg/dL) overall, and the readings were highest during winter. Among the 376 patients identified as "non-dippers" (decrease in nocturnal blood pressure of  $< 10\%$ ), mean fibrinogen levels were 21 mg/dL higher over the year than in the non-dippers ( $P < .002$ ). These results emphasize the importance of 24-hour blood pressure control and the need to identify non-dippers by ambulatory blood pressure monitoring in the prevention of coronary events and death, according to Dr. Hermida.

### **Resistant Hypertension Caused by Obstructive Sleep Apnea?**

A retrospective analysis of patients with resistant hypertension diagnosed with primary hyperaldosteronism conducted by researchers from the University of Alabama at Birmingham revealed a much greater prevalence of confirmed or suspected obstructive sleep apnea (OSA) compared with patients without primary Hyperaldosteronism. A total of 78 patients with resistant hypertension (defined as uncontrolled hypertension despite the use of 3 or more antihypertensive agents) were evaluated for OSA (Epworth symptom survey score  $> 10$ ). OSA was diagnosed or suspected in 63% of patients. Primary Hyperaldosteronism (plasma renin activity suppressed to  $< 1$  ng/h and urinary aldosterone elevated to  $> 12$  mcg/24 h) was diagnosed in 27% of patients with known or suspected OSA, but in only 14% of those without known or suspected OSA. The odds ratio of a patient with OSA having primary Hyperaldosteronism was 2.3 compared with patients without OSA. David Calhoun, MD, and colleagues concluded that the high prevalence of OSA in patients with primary Hyperaldosteronism suggests that OSA may cause resistant hypertension by induction of hyperaldosteronism.

Note from DV:

Hyperaldosteronism is a disease caused by an excess production of the normal adrenal hormone, aldosterone. This hormone is responsible for sodium and potassium balance, which then directly controls water balance to maintain appropriate blood pressure. Hyperaldosteronism causes high blood pressure and a low serum potassium. The serum sodium is usually in the normal range. This is a rare disease and has been considered an unusual cause of hypertension. It is usually considered by physicians if they find an unexpectedly low potassium in a person being treated for hypertension. It generally causes no other specific symptoms or signs.