

dependent of blood pressure reduction.”

The effect of valsartan alone and of valsartan plus HCTZ on CRP levels was “remarkably consistent” across every patient subgroup examined, including patients who were also treated with statins and those who were not, he said. This last observation suggests that the ability of valsartan alone to lower CRP levels is independent of the action of statins, and so the two effects might be additive.

Although Dr. Ridker acknowledged that CRP-lowering may be a class effect for all ARBs, he stressed that so far the effect has been proved only for valsartan: “We’ve been [wrong] about class effects

for statins; it may not be as simple as we once thought.” ARBs were hypothesized to have an effect on CRP because of angiotensin II’s proinflammatory effects. (ARBs block the angiotensin II receptor.)

It’s unclear what effect HCTZ might have on inflammatory processes. The drug is known to boost insulin resistance, the incidence of diabetes, and serum levels of plasminogen activator inhibitor-1. All of these activities track with elevated CRP levels, but it’s too soon to say whether this explains the study’s results, he said.

Even if HCTZ is eventually shown to block a beneficial reduction in CRP levels, and despite the drug’s other adverse

effects, physicians will probably find it hard to avoid using the drug in patients with refractory hypertension.

“In many patients with hypertension, it’s extremely hard to get them to their goal pressure without a diuretic,” commented Dr. Joel M. Neutel, medical director of clinical pharmacology at the Orange County Research Center in Tustin, Calif.

“If a patient is on two vasodilator drugs, such as an ARB and a calcium channel blocker, the third drug almost has to be a diuretic. We need diuretics to treat hypertension. You use other drugs to balance their negative effects,” Dr. Neutel said. ■

Aldosterone May Worsen Sleep Apnea

BY MITCHEL L. ZOLER
Philadelphia Bureau

NEW YORK — A link between aldosterone, hypertension, and obstructive sleep apnea was made in a study with 71 patients.

“We found an extraordinarily high prevalence of obstructive sleep apnea in patients with [treatment-] resistant hypertension,” and serum aldosterone levels were significantly related to the severity of sleep apnea,” Dr. David A. Calhoun said at the annual meeting of the American Society of Hypertension. “We went in thinking that obstructive sleep apnea was driving aldosterone release, but now we think that a high serum level of aldosterone somehow contributes to worsening sleep apnea,” said Dr. Calhoun, a hypertension specialist at the University of Alabama, Birmingham.

The link may be mediated by increased salt and water retention or perhaps by a change in flow resistance.

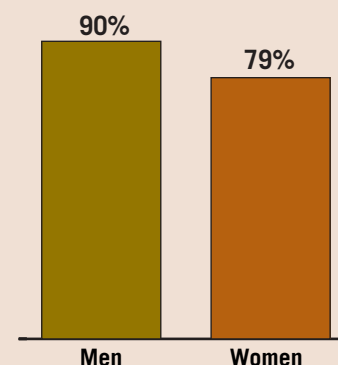
Dr. Calhoun and his associates have begun a study to explore the implications of their findings for patient management. They are withholding continuous positive air pressure, a standard treatment for obstructive sleep apnea, from patients with the disorder and are instead treating them with spironolactone, an aldosterone antagonist. The goal is to see whether spironolactone alone is effective at relieving sleep apnea.

The current study involved a consecutive series of 41 men and 30 women who were referred to the hypertension clinic at UAB because of treatment-resistant hypertension. Their mean blood pressure was 156/88 mm Hg despite treatment with an average of four antihypertensive drugs.

The patients were assessed for obstructive sleep apnea by diagnostic polysomnography. The overall prevalence of obstructive sleep apnea was 85%, with a prevalence of 90% in the men and 79% in the women. The average apnea-hypopnea index for all patients was 24 apnea events per hour.

The patients with sleep apnea also had high serum and urine levels of aldosterone. Those with the most severe sleep apnea had the highest levels, Dr. Calhoun said. ■

Obstructive Sleep Apnea In Hypertensive Patients



Note: Based on a study of 71 patients with treatment-resistant hypertension.
Source: Dr. Calhoun

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