About an Epidemic of Primary Aldosteronism

To The Editor:

I agree with Calhoun’s conclusion that there is currently an epidemic of elevated aldosterone:renin ratio rather than an epidemic of primary aldosteronism (PA). I disagree, however, with the assertion that it is “inappropriate to consider idiopathic hyperaldosteronism (presumed secondary to adrenal hyperplasia) to be a type of PA” and that PA should be confirmed by a suppression test.

According to World Health Organization’s International Classification of Disease (10th Revision, version for 2007), the E26.0 category (primary hyperaldosteronism) has 2 subheadings: Conn’s syndrome and PA because of adrenal hyperplasia (bilateral). Conn’s syndrome (or aldosterone-producing adenoma [APA]) and PA because of bilateral adrenal hyperplasia (or idiopathic PA) differ in renin and aldosterone plasma levels, renin usually being lower and aldosterone usually higher in APA than in idiopathic PA, and, theoretically, in the response to dynamic tests. Stimulation and suppression tests have been designed under the assumption that the surgically remediable form of PA, APA, is associated with angiotensin-unresponsive aldosterone hypersecretion. The predicted response to stimulation tests is a fall or no change in aldosterone concentration in cases of APA and an angiotensin-dependent rise in cases of essential hypertension or idiopathic PA. However, several studies reported that 30% to 70% of patients with APA display an increase in aldosterone levels with ambulation. Stimulation tests do not help identify subjects with PA and an APA,2 a lateralized aldosterone hypersecretion on adrenal venous sampling, or a favorable BP outcome after surgery.2 Many suppression tests have been proposed for the confirmation of PA based on the principle that aldosterone levels should not be suppressed by maneuvers reducing angiotensin levels in patients with confirmed PA. To my knowledge, there is no published evidence that the response to suppression tests predicts the outcome of surgery; however, there is published evidence of the contrary.3,4 The use of suppression tests to confirm PA results in selecting patients with nonsuppressible aldosterone secretion, thereby excluding most patients with idiopathic PA and all patients with angiotensin-responsive APA. It is important to identify patients with PA, because the cardiovascular and renal consequences of hypertension are more severe in these patients than in patients with essential hypertension and similar levels of BP. In addition, PA requires relatively specific treatment, mineralocorticoid receptor inhibition using spironolactone or eplerenone and aldosterone synthase inhibition in the future. It is particularly important to document angiotensin-responsive APA, because hypertension in this condition is improved by unilateral adrenalectomy.5

Disclosures

None.

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*Hypertension*. 2008;51:e6; originally published online January 7, 2008;
doi: 10.1161/HYPERTENSIONAHA.107.104448

*Hypertension* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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