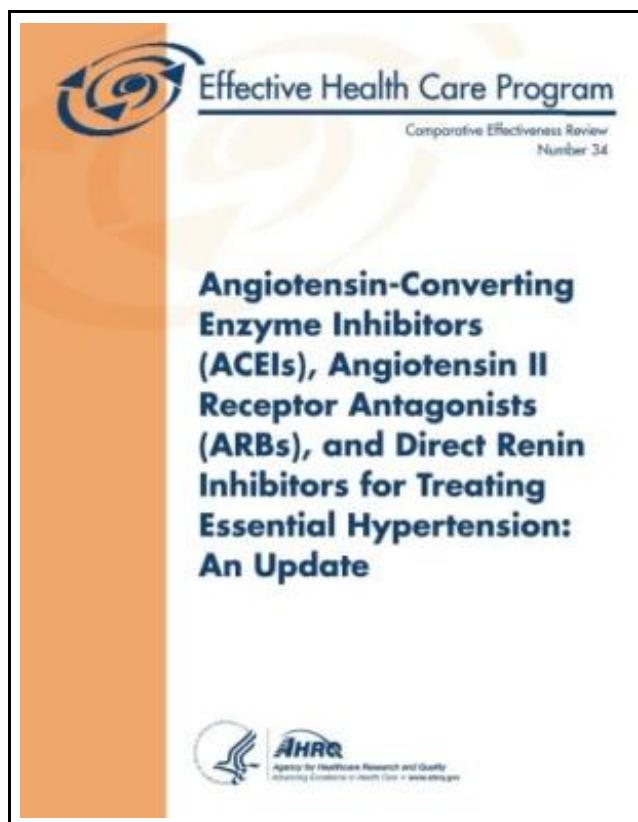


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## **ANGIOTENSIN-CONVERTING ENZYME INHIBITORS (ACEIS), ANGIOTENSIN II RECEPTOR ANTAGONISTS (ARBS), AND DIRECT RENIN INHIBITORS FOR TREATING ESSENTIAL HYPERTENSION: AN UPDATE: COMPARATIVE EFFECTIVENESS REVIEW NUMBER 34 (PAPERBACK)**



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Createspace, United States, 2013. Paperback. Book Condition: New. 279 x 216 mm. Language: English . Brand New Book \*\*\*\*\* Print on Demand \*\*\*\*\*. Almost 75 million American adults-approximately one-third-have hypertension. The prevalence of hypertension increases with advancing age such that more than half of people 55 to 74 years old and approximately three-fourths of those age 75 years and older are affected. In addition to being the primary attributable risk factor for death throughout the world, hypertension results in substantial morbidity because of its impact on numerous target organs, including the brain, eyes, heart, arteries, and kidneys. Despite the high rates of morbidity and mortality attributable to hypertension, control of the condition remains suboptimal. In addition to several effective nonpharmacological interventions-including diet, exercise, and control of body weight-many people require antihypertensive medication to lower blood pressure. Among the many choices in antihypertensive therapy, some of the most common are those aimed at affecting the renin-angiotensin-aldosterone (renin) system. The renin system is an important mediator of blood volume, arterial pressure, and cardiac and vascular function. Components of this system can be identified in many tissues, but the primary site of renin release is the kidney. The renin system can be triggered by sympathetic stimulation, renal artery hypotension, and decreased sodium delivery to the distal tubule. Through proteolytic cleavage, renin acts on the oligopeptide substrate angiotensinogen to produce the decapeptide angiotensin I. In turn, two terminal peptide residues of angiotensin I are removed by the angiotensinconverting enzyme (ACE) to form the octapeptide angiotensin II. Angiotensin II acts directly on the resistance vessels to: increase systemic vascular resistance and arterial pressure; stimulate the adrenal cortex to release aldosterone, which leads to increased sodium and water reabsorption and potassium excretion; promote secretion of antidiuretic hormone, which leads to fluid retention; stimulate thirst; promote adrenergic function;...

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