Angiotensin AT1-receptor blockers and cerebrovascular protection: do they actually have a cutting edge over angiotensin-converting enzyme inhibitors?

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Résumé en anglais	First, an update of the vascular systemic and tissue renin–angiotensin–aldosterone system is provided to explain how it is regulated at the systemic and tissue levels, and how many angiotensin peptides and receptors can be modulated by the various antihypertensive drugs. Second, experimental data is presented to support the hypothesis that antihypertensive drugs that increase angiotensin II formation, such as diuretics, AT1-receptor blockers and dihydropyridines, may have greater brain anti-ischemic effects than antihypertensive drugs that decrease angiotensin II formation, such as β-blockers and angiotensin-converting enzyme inhibitors, because they increase activation of angiotensin AT2 and AT4 receptors. Indeed, these trigger brain anti-ischemic mechanisms by favouring cerebral blood flow (angiogenesis and recruitment of pre-existing collateral circulation, specifically in the ischemic brain where AT2 receptors are overexpressed) or by directly increasing neuronal resistance to anoxia. Third, we review most of the large primary and secondary stroke prevention trials as well as the ACCESS acute stroke trial in which antihypertensive drugs were evaluated. With the exception of the secondary stroke prevention trial PRoFESS, most trials support the hypothesis that angiotensin II-increasing drugs confer specific blood pressure-independent brain ischemia protection when compared with angiotensin II-decreasing drugs or placebo. A careful analysis of the PRoFESS trial, however, reveals study design limitations, the main one being that diastolic BP (<80 mmHg) in the first month post-stroke may have been too low in at least one third of the population with baseline systolic blood pressure less than 130 mmHg, because a high dose of telmisartan was given after a very short post-stroke delay (median 15 days) without discontinuation of the baseline antihypertensive treatment.
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