Rapid progress in relation to cardiovascular effects of angiotensin 1 to 7 (Ang 1–7), the Mas receptor, and the angiotensin-converting enzyme type 2 (ACE2) is an example of basic biomedical research, which may eventually lead to an advance in care of patients.

When one of us (D.H.) first attended the meeting of the Council for High Blood Pressure Research about 1970, the future of studies of the renin/angiotensin system seemed limited. It looked like there was not much more to be learned and was not a promising area of research. 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TACE? Would systemic administration of Ang 1–7 protect the brain after brain ischemia?

The future? These studies suggest that Ang 1–7/ACE2 Mas axis may protect against stroke; an enormous word of caution, however. Many studies have observed that a wide variety of interventions have failed to reduce the size of ischemic strokes in experimental models (especially in rats and mice). But these interventions have failed to reduce the size of strokes in humans. As a minimum, this area of research is clarifying mechanisms by which endogenous Ang 1–7/ACE2 protects against stroke. We are far from knowing however whether Ang 1–7 will be the first peptide to protect against stroke in humans.

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**Disclosures**

None.

**References**


**Figure**. Brain ischemia induces neuroinflammation, apoptosis, and oxidative stress and causes brain damage. Recent studies revealed that brain ischemia may increase the circulating and local angiotensin-converting enzyme type 2 (ACE2) activity. Increased ACE2 activity may lead to increased formation of angiotensin 1 to 7 (Ang 1–7) and stimulation of Mas receptors, which may be neuroprotective. Similar effects can be obtained after local or intraperitoneal administration of an ACE2 activator (diminazene). Ang II indicates angiotensin II.
Stages in Discovery: Angiotensin-Converting Enzyme Type 2 and Stroke
Ricardo A. Peña-Silva and Donald D. Heistad

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