1. Title: ACE-AGT associations with IMT in the ARIC data

2. Writing Group:
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3. Timeline:
   Analysis and writing will proceed upon receipt of IMT data from G. Heiss

4. Rationale:
   Because of the effects of angiotensin II on vascular tone, growth of smooth muscle cells, and production of extracellular matrix proteins, it seems plausible that chronic exposure to high levels of plasma ACE might result in vascular wall thickening. The ACE I/D polymorphism has been shown to correlate with plasma ACE concentrations, and, thus, the ACE I/D polymorphism may be associated with IMT.

5. Main Hypothesis:
   (1) Can variation in IMT be explained by variation at either of the ACE or AGT loci?
   (2) Can the potential role of ACE and AGT in CHD be identified with atherogenesis without MI or is the effect associated with the acute event on the diathesis of atherosclerosis?

6. Data (variables, time window, source, inclusions/exclusions):
   Subset of the Forsyth and Minneapolis ARIC samples as designed for the FHS ACE/AGT study.