1.a. **Full Title:** C-reactive protein concentration and its relation to the prevalence, extent, and severity of adult periodontal disease.

b. **Abbreviated Title** (Length 26): CRP-Periodontal Disease

2. **Writing Group** (list individual with lead responsibility first):
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3. **Timeline:**
   Begin C-reactive protein measurements in fall 1997; begin statistical analysis and manuscript writing in early 1998.

4. **Rationale:**
   C-reactive protein (CRP) is an acute phase reactant and nonspecific marker of inflammation, infection, and tissue damage. Prospective epidemiologic studies have found that baseline C-reactive protein concentration measured in healthy adults predicts myocardial infarction, ischemic stroke (Ridker et al., 1997), and fatal CHD (Kuller et al., 1996). The determinants of CRP are unknown; subacute inflammation of the coronary and other arteries, chronic bacterial or viral infections (Helicobacter pylori, Chlamydia pneumoniae, or cytomegalovirus), and bronchial inflammation due to smoking may increase hepatic production of CRP. Cardiovascular disease risk factors such as body mass index, LDL-C, low HDL-c, and fibrinogen are also positively associated with CRP (Mendall et al., 1996). Adult periodontal disease may be another important determinant of C-reactive protein: CRP may rise due to the systemic effects of lipopolysaccharide (endotoxin) released by oral pathogens and serve as an important link between periodontal disease, atherosclerosis, and its clinical sequelae. Ebersole et al. (1997) recently found statistically significantly higher levels of CRP in 35 patients with moderate to severe adult periodontitis (9.1 mg/l) than in 40 normal controls (2.2 mg/l).
   Visit four of ARIC includes a dental examination: ARIC is collecting gingival crevicular fluid, plaque samples, and serum samples to determine levels of inflammatory mediators, oral pathogens, and bacterial-specific antibody titers.