1. Title (length 26):
C. TWAR & Atherosclerosis

2. Writing Group (list individual with lead responsibility first):
(lead) Melnick                Folsom                               Sorlie
           Shahar             CCSC representative          URC representative

3. Timeline:
As soon as the antibody test results are available.

4. Rationale:
Results from a case-control study of acute myocardial infarction patients, chronic coronary heart disease patients, and controls indicated a higher antibody response in both case groups as compared to controls (Saikku, Leinonen, et.al. Lancet 1988;2:983-6). These data are suggestive of an association between Chlamydia TWAR and atherosclerosis, in that C. TWAR is a clinically important agent in the "response to injury" hypothesis in a manner similar to that postulated for certain herpes viruses. Alternatively, it may be that C. TWAR-associated pneumonia and concurrent respiratory distress precipitates cardiac distress in an individual with preexisting atherosclerosis or that C. TWAR finds an ecologic niche in developing atherosclerotic plaques independently of any pathogenic process.

5. Main Hypothesis:
Baseline serum IgG antibodies to Chlamydia pneumonia strain TWAR will be increased in ARIC cases relative to paired ARIC controls.

6. Data (variables, time window, source, inclusions/exclusions):
Dependent variables: wall thickness (case vs. control status)
Independent variables: C. TWAR antibody levels (IgG & IgM geometric mean titer)
Covariates: Smoking, pulmonary function, respiratory signs and symptoms. Other risk factors such as increased SBP, lipids, and fibrinogen will also be considered. Pairs will have been matched on age, race, sex, time of exam, and field center.

Keywords: Infectious agents, CHD, case-control, lung, wall thickness