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REPLY

We thank Drs. Taylor-Robinson and Thomas for their interest in our study, which found circulating *Chlamydia pneumoniae (C. pneumoniae)* DNA to be associated with coronary artery disease (CAD) in men but not women (1). The association in men was moderate, with an odds ratio of 3.2 (95% confidence interval 1.1 to 8.9), and this was stronger than that reported in most serologic studies (2). We discussed possible reasons for the lack of an association in women, but because the number of male subjects was over twice that of females, the statistical power to detect a difference was strongest for men. In our study, patients and control subjects were defined according to the presence of CAD by angiographic criteria, an approach taken by other studies of *C. pneumoniae* and CAD (2), and, indeed, by studies that have investigated other coronary risk factors. We can justifiably claim that we have found an association between *C. pneumoniae* DNA and clinically significant atherosclerosis.

Our study and that of Boman et al. (3) are still the only published reports on circulating *C. pneumoniae* DNA and CAD, and we pointed out the differences in the reported prevalence of *C. pneumoniae* DNA. In our view, the finding that circulating *C. pneumoniae* DNA was found in 46% of a healthy, blood-donating population (3) is remarkable and an extraordinarily high level for any bacterium. However, although our study was far larger than Boman's, further work is required to clarify the situation, but we cannot comment on the unpublished data of the correspondents or their observations.

It would be generally accepted that evidence of current *C. pneumoniae* infection should be found before prescribing antibiotics to patients with CAD. At present, the presence of circulating *C. pneumoniae* DNA is the most accurate method of diagnosing current infection and is therefore a means of identifying suitable patients for intervention trials. This was also a view held by the correspondents (4), and we are therefore surprised by their statement that "fortunately," current antibiotic trials are being undertaken with complete disregard for the PBMC *C. pneumoniae* status. Fortunately, investigators running such trials do realize the potential importance of such a test (5).

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The Electric Cardiographic Abnormalities Are Not Hidden!

The report by Matetzky et al. (1) stimulates the following thoughts. There are two ways to interpret electrocardiograms (ECGs). One is to memorize patterns and the other is to use basic principles of electrocardiography, including the use of vector concepts, as described by Grant (2–6).

The tracing in Figure 1A of the report by Matetzky et al. reveals a left atrial abnormality; a mean spatial QRS vector that is directed at about +20° in the frontal plane (it changes direction during inspiration and expiration) and about 45° posteriorly; a mean spatial ST segment vector that is directed at +115° in the frontal plane and at least 90° posteriorly, indicating epicardial injury of the posterior wall of the left ventricle; a mean spatial T-wave vector that is directed at +90° in the frontal plane and 80° to 90° anteriorly, indicating posterior myocardial ischemia; and a large U wave in lead I.

The point is, the ST segment vector points toward an area of posterior epicardial injury. Furthermore, one can suspect that the ST segment vector is directed toward an obstruction in the circumflex coronary artery or its branches. It should be no surprise that the ST segment is elevated in leads $V_{7–9}$, because the transitional pathway was just beyond electrode position V_6. *The ST segment abnormality is not hidden.*

The follow-up discharge tracing in Figure 1B is also interesting. The left atrial abnormality has disappeared; an S_1, S_2 and S_3 conduction defect has developed; and the first half of the QRS complex is directed at about +40° in the frontal plane and markedly anterior, producing large abnormal R waves in leads V_1

and V_2. This abnormality is due to a true posterior infarction. Electrodes located anywhere on the front half of the chest would record large R waves, and electrodes located anywhere on the back half of the chest would record Q waves. *The abnormal initial portion of the QRS is not hidden.*

The ECG shown in Figure 2 also reveals an S_1, S_2 and S_3 conduction defect and abnormal ST segment displacement. Some would argue that the ST segment displacement in the extremity leads is an artifact due to downsloping of the recording. If that is true, the mean ST segment vector points toward epicardial injury surrounding a true posterior infarction, which is usually caused by obstruction of the circumflex coronary artery, the distal right coronary artery, or their branches. If the ST segment displacement in the extremity leads is real, the mean ST segment vector is directed at 30° to 60° in the frontal plane and about 45° posteriorly, indicating epicardial injury of the left lateral and posterior portion of the left ventricle. This could be caused by obstruction in the obtuse marginal branch of the circumflex coronary artery or a diagonal branch of the left anterior descending coronary artery. *The ST segment abnormality is not hidden.*

None of the abnormalities are hidden, and true posterior infarction should be considered in both of the 12-lead tracings.

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REPLY

In our article, we describe the clinical course, echocardiographic and angiographic findings of 33 consecutive patients with acute myocardial infarction and ST segment elevation in posterior chest leads $V_{7–9}$ but *without* significant ST segment elevation on the standard 12-lead ECG. Because these ST segment elevations are not presented on the classically performed 12-lead ECG, we called them "hidden" (in quotation marks).

Like Dr. Hurst, who taught all of us through his textbook, we were also much inspired by the vectorcardiographic approach to electrocardiography so beautifully presented in Grant's book (1). However, the chest with the lungs full of air is not an ideal volume conductor, and the heart is not in the exact middle of it. Most of the time, whenever there is ST segment elevation in leads $V_{7–9}$, ST segment depression will be detected in the anterior chest leads. However, there will always be cases, as we show here, where the nonuniformity of the chest and other factors will result in distorted spread of potential, leading to the absence of reciprocal changes in leads $V_{1–3}$, despite QRS–ST-T segment changes in leads $V_{7–9}$.

We fully agree with the general statement by Dr. Hurst that there are two ways to interpret the ECG: one is to memorize patterns and the other is to use basic principles of electrocardiography. But they are *not* mutually exclusive, and sometimes the principles of volume conduction and nonuniformity and distorted spread of potential will result in the changes we presented.

The posterior chest leads $V_{7–9}$ ECG recording is simple and feasible, and the appearance of ST segment elevations in posterior chest leads $V_{7–9}$ can be detected easily by any physician, even without a deep understanding of basic ECG principles.

We believe that the early detection of ST segment elevation posterior myocardial infarction in the era of reperfusion is crucial for the identification of patients with acute posterior myocardial infarction, who may benefit from reperfusion therapy.

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