During evaluation for intermittent atypical chest pain of two weeks duration, a 59-year-old woman had an electrocardiogram performed. (Figure).

What is your diagnosis?

Explication is on pg. 218
ECG of the Month
Presentation is on pg. 217

DIAGNOSIS: Sinus rhythm with monomorphic ventricular bigeminy and ventriculophasic sinus arrhythmia. Otherwise, the ECG is normal.

Ventricular premature complexes occurring after each sinus-initiated QRS complex is the commonest form of bigeminal rhythm. In this instance, the premature complexes resemble a left bundle branch block pattern, indicating that they arise from the right ventricle.

Alternate sinus P waves are fused with the terminal portion of each ventricular premature complex and reach the atrioventricular conduction system in its absolute refractory period. This prevents their conduction to the ventricles and causes a compensatory pause before the next sinus P wave.

The sinus rhythm is slightly irregular. Because the beginning of alternate P waves is buried in a ventricular premature complex, the P-P intervals are best measured from the end of one P wave to the end of the next P wave. Such measurement reveals that the P-P intervals containing two QRS complexes (sinus initiated and premature ventricular) consistently are shorter (0.67s) than P-P intervals containing no QRS complex (0.72s). This ventriculophasic sinus arrhythmia often is most clearly seen in electrocardiograms showing sinus rhythm with 2:1 atrioventricular block but may be seen in any situation in which some sinus P-P intervals contain QRS complexes, while others do not.

Two mechanisms have been postulated for ventriculophasic sinus arrhythmia. In one, the pressure of ejected blood on the sinus node is thought to cause a momentary increase in its rate, and thus, the P-P interval containing the QRS complex is shorter than the P-P interval without a QRS. The second proposed mechanism states that the increase in carotid sinus pressure with ventricular systole causes reflex slowing of the sinus node some 200-500 ms after the peak of mechanical systole, which, of course, is well after the QRS. Thus, the slowing of the sinus rate occurs after the P-P interval containing the QRS. The second explanation has always made more sense to us. Furthermore, Prystowsky and Klein have produced sinus node slowing in a patient using neck-collar suction, and the timing of the slowing fits with the second explanation, not the first.

Our patient had no history of heart disease and in the course of her workup for chest pain had a normal atropinedobutamine stress echocardiogram. She subsequently has been treated more vigorously for her acid-reflux disease.

REFERENCES