

Country cardiograms case #66: Answer

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Sinus tachycardia is present in Figure 1, with a rate of 108/min. PR interval, QRS duration and QT interval are within normal limits. Left axis deviation is present (-80°). ST-T configuration is within normal limits, but P waves are markedly tall (3.5 mm) and peaked in leads II, III and aVF. Examination of the rhythm strip reveals a pattern whereby the amplitude of the QRS complex fluctuates substantially in a regular pattern, with a frequency of 16/min. The greatest QRS amplitude is 15 mm and the smallest QRS amplitude is 9 mm.

Two features of note are the amplitude of the P waves and the fluctuating amplitude of the QRS complexes.

The peaked, tall P waves (3.5 mm) meet criteria for right atrial abnormality (2.5 mm is the standard cutoff). 'Right atrial abnormality' is arguably a more appropriate term than the alternative 'right atrial enlargement'. Such P waves are also known as P *pulmonale*. In patients without a history of primary pulmonary hypertension, tricuspid stenosis or congenital heart conditions such as pulmonary stenosis or tetralogy of Fallot, the common cause of such P waves is chronic lung disease. They suggest the development of cor pulmonale.

The frequency of the pattern of changing QRS amplitude seen in lead V1 in the rhythm strip is the same as the patient's noted respiratory rate. The likeliest cause for this pattern is 'respiratory alternans'. This term needs to be distinguished from 'pulsus alternans' (a physical sign of alternating strong and weak arterial pulse beats) and 'electrical alternans', in which QRS amplitude or axis on the ECG varies from beat to beat, and which has classically been associated

with a diagnosis of large pericardial effusion.

The changing amplitude pattern, which is likely related to the breathing cycle, suggests a change in impedance, in which when the patient inhales, the amount of air between the heart and the chest wall increases, and hence, the QRS amplitude decreases. Diaphragmatic breathing can also cyclically change the position of the heart. While this is a physiological phenomenon and can even be construed as a healthy sign, the question arises as to why it is seldom evident to the marked degree seen in Figure 1 and whether such ECG changes may be exacerbated in patients with COPD.

In this case, the relatively slow respiratory rate, absence of fever and only slightly decreased oxygen saturation seem reassuring. The ECG findings of right atrial abnormality and substantial variation in QRS amplitude related to the breathing cycle suggest that the patient may be in more difficulty than is apparent from the traditional vital signs. Especially given this patient's relatively young age of 56 years, assessment of risk factors is indicated (including inquiring about a history of smoking and a positive family history of emphysema that might suggest a condition such as alpha-1 antitrypsin deficiency) along with further investigations such as pulmonary function testing and echocardiography.

In a remote ER setting, with relatively few diagnostic aids immediately available, the ECG has the capacity to independently provide potentially useful information that may influence management, as in this case.

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For the question, please see page 121