# The electrocardiogram in pulmonary hypertension

## Case history

*Figure 1* shows the electrocardiogram of a 60-year-old woman who initially presented in the third decade of life with mild symptoms of dyspnoea, reduced exercise capacity and fatigue. Clinical signs included cyanosis and clubbing, right ventricular heave, palpable and loud pulmonary second sound, and a soft pansystolic murmur. Echocardiography confirmed a patent ductus arteriosus and evidence of pulmonary hypertension with right ventricular hypertrophy (RVH), significant tricuspid regurgitation and pulmonary regurgitation.

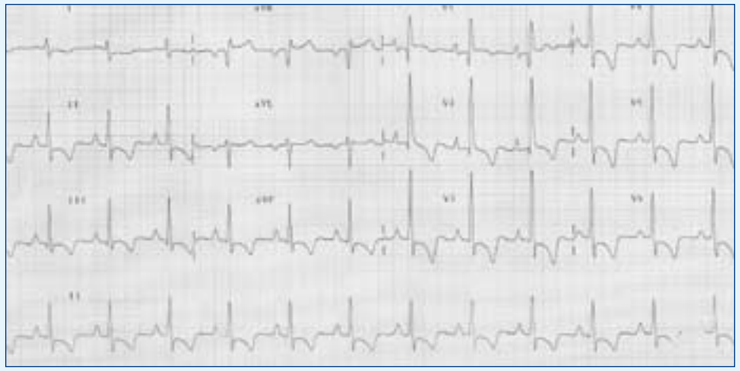
## The electrocardiogram

The electrocardiogram (ECG) demonstrates sinus rhythm with a PR interval of 200 msec. There is severe RVH and right heart strain with right axis deviation, partial right bundle–branch block and prominent R waves in the anterior chest leads (particularly V1 and V2). T wave inversion is seen in leads V2 to V6, II, III and aVF together with ST depression. A right atrial abnormality is shown by tall peaked P waves in the same leads, reflecting right atrial enlargement.

**Dr Clive Lewis** *is Specialist Registrar in Cardiology and* **Dr Pier Lambiase** *is Clinical Lecturer in Electrophysiology in the Cardiology Department, The Heart Hospital, University College London, London W1M 8PH*

*Correspondence to: Dr P Lambiase*

**Figure 1. Electrocardiogram.**



## Eisenmenger syndrome

In this case the findings are consistent with Eisenmenger syndrome of pulmonary hypertension, with a reversed central shunt. An uncorrected or unrecognized large central left-to-right shunt causes a progressive rise in pulmonary vascular resistance leading to reversal of the shunt with resultant cyanosis. The most common causes of Eisenmenger syndrome are ventricular septal defect, atrioventricular septal defect and patent ductus arteriosus, accounting for up to 80% of cases.

Pulmonary hypertension develops early, within the first few years of life and is irreversible. There is a slow symptomatic decline into adulthood with 77% survival at 15 years and 42% survival at 25 years after diagnosis. Important causes of mortality include sudden death, congestive heart failure and haemoptysis (Daliento et al, 1998).

RVH may be caused by congenital heart disease resulting in elevated right ventricular pressures either as a result of a shunt at ventricular level, outflow tract obstruction (ventricular septal defect, atrioventricular septal defect, pulmonary stenosis or pulmonary atresia) or pulmonary hypertension. Pulmonary hypertension is defined as an increase of mean pulmonary artery pressure greater than 25 mmHg at rest. Other causes of pulmonary hypertension can be divided into four main categories:

1. Pulmonary arterial hypertension, including primary pulmonary hypertension, collagen vascular disease, Eisenmenger physiology, portal hypertension, infective (e.g. human immunodeficiency virus or schistosomiasis) and drugs
2. Pulmonary venous hypertension including left-sided valvular heart disease, cardiomyopathy, pulmonary vein stenosis and pulmonary veno-occlusive disease
3. Respiratory disorders including chronic obstructive pulmonary disease, interstitial lung disease, sleep apnoea, altitude and neonatal lung disease
4. Chronic pulmonary thrombotic or embolic disease including pulmonary embolism (of any cause), in situ thrombosis and sickle cell disease (Rich, 1988).

Features of the ECG in pulmonary hypertension are largely those of RVH. However, it is recognized that up to 13% of patients with significant pulmonary hypertension have normal ECG findings (Ahearn et al, 2002). Features of RVH include right axis deviation (frontal QRS axis) greater than 80°, partial or complete right bundle– branch block, R/S wave amplitude ratio in lead V1 greater than 1, and the R wave in lead V1 greater than 0.5 mV.

Patients even with severe pulmonary hypertension often remain in sinus rhythm, although there is significant right atrial enlargement resulting in large and peaked P waves (may be best seen in lead II) and often associated with a prolonged PR interval. Additional features of right heart strain are T wave inversion in the precordial leads, with ST depression which may suggest right ventricular ischaemia.

Previous studies have suggested that ECG parameters may be predictive of the severity of right ventricular dysfunction in patients with acute pulmonary emboli. In addition, changes in the ECG may reflect improvement in right ventricular function after single-lung transplantation. The correlation between the severity of pulmonary hypertension and ECG parameters is less clear. In general, the higher the pulmonary artery pressure, the more sensitive is the ECG for diagnosing RVH. The chest X-ray is inferior to the ECG in detecting pulmonary hypertension, but it may show evidence of dilated pulmonary arteries or any signs of underlying lung disease (Widimsky, 1985).

The amplitude of the R wave in V1 of more than 1.2 mV indicates a pulmonary artery systolic pressure of more than 90 mmHg. Extreme right axis deviation (≥100°), and either a S wave in V6 ≥0.7 mV or a R/S wave amplitude ratio in V6 ≥2 correlate with a low cardiac index (<2.8 litre/min/m$^2$; Kanemoto, 1988). Other studies suggest that the mean frontal QRS axis correlated best with the severity of haemodynamic impairment (Ahearn et al, 2002). In addition, together with clinical data, the ECG can be used to predict the severity of pulmonary valve stenosis.

RVH needs to be differentiated from other conditions. The appearance of the ECG with R waves in the precordial leads, T wave inversion and ST depression may mimic posterior infarction. The high R/S amplitude ratio in lead V1 and the larger S wave in lead V5 are the most useful indices of RVH. The ECG in chronic lung disease and subendocardial anterior myocardial infarction can also be confused with RVH. The ECG changes found in pulmonary embolism are also similar to those found in RVH but they are transient and are more easily interpreted by changes on serial ECG (Bourdillon, 1978). BJHM

*Conflict of interest: none.*

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## KEY POINTS

- Pulmonary hypertension can be categorized as arterial, venous, respiratory or secondary to thrombosis.
- A combination of specific and non-specific electrocardiographic changes point to this pathophysiological process.
- Right ventricular hypertrophy may diagnosed using the following criteria:
  - Right axis deviation of +110° or more
  - R/S ratio > 1 in lead V1 and a negative T wave
  - R wave lead V1 <7 mm
  - S wave lead V1 <2 mm
  - qR in V1
  - rSR′ V1 with R′ >10 mm.



ELECTROCARDIOGRAM INTERPRETATION