Diagnosis of old anterior myocardial infarction in emphysema with poor R wave progression in anterior chest leads

GRAHAM J HART, PETER A BARRETT, PETER F BARNABY, ELIZABETH H CLARK, NORMAN R LYONS, JOHN J BURKE

From the Departments of Cardiology, Respiratory and Nuclear Medicine, The St George Hospital, and the Department of Medicine, University of New South Wales, Sydney, Australia

SUMMARY The electrocardiograms of patients with emphysema may suggest associated old anterior myocardial infarction. Sixteen patients with the physiological characteristics of emphysema were studied, who also showed poor R wave progression in the anterior chest leads, so that RV3 was ≤ 4 mm. A thallium-201 cardiac scan consistent with previous anterior (septal) myocardial infarction was present in seven patients. In these patients there was no significant increase in RV3 amplitude when recorded one interspace below the conventional site. In the nine patients with a thallium-201 cardiac scan negative for old anterior myocardial infarction, RV3 amplitude increased from 2·2 ± 0·4 mm to 6·4 ± 1·2 mm. Patients with or without associated old anterior myocardial infarction could be better diagnosed by consideration of RV3 amplitude as recorded from one interspace lower, as compared with conventional electrode placement. All five patients with RV3 (lower interspace) < 2 mm had associated anterior infarction, and all seven patients with RV3 (lower interspace) > 3 mm did not. This simple manoeuvre is recommended in patients with emphysema and an electrocardiogram suggestive of old anterior myocardial infarction.

The electrocardiogram of some patients with emphysema may suggest previous anterior myocardial infarction, with small or absent initial R waves in the anterior chest leads, resulting in QS complexes or "poor R wave progression" in these leads.1-3

In such patients there is difficulty in deciding whether these electrocardiographic appearances are the result of emphysema or previous anterior myocardial infarction, or whether in fact both conditions are present.

Such electrocardiographic appearances in emphysema have been ascribed to hyperinflation with descent of the diaphragm.4-6 This results in a relatively low position of the heart so that the anterior chest leads now have a superior orientation, and so reflect the dominantly negative QRS complexes normally associated with such an orientation.

The value of recording the anterior chest leads in a lower intercostal space and noting the presence or absence of increase in R wave amplitude, to exclude or confirm associated anterior myocardial infarction, has been suggested in the past.4 7 Such studies, however, have involved patients with a wide variety of cardiopulmonary diseases, or have not adequately documented the respiratory pathophysiology. In addition, the criteria for the presence or absence of anterior myocardial infarction have usually been clinical, and therefore subject to the error inherent in this difficult clinical differentiation. Moreover, the actual changes in R wave amplitude in such patients with emphysema with and without associated anterior myocardial infarction have not been quantified.

In this study, 16 patients with the physiological characteristics of emphysema, who also showed small or absent initial R waves in the electrocardiographic chest leads V1 to V3 were considered, and these leads were also recorded one interspace below the conventional sites. Changes in the initial R wave of the QRS complexes were noted and results were correlated with the diagnosis of anterior myocardial infarction made by thallium-201 cardiac scanning.
Methods

Sixteen patients were studied with a clinical diagnosis of emphysema, and poor R wave progression in the anterior chest leads so that the amplitude of the R wave in lead V3 was 4 mm or less. None had clinical data adequately documenting previous myocardial infarction. No patient had undergone cardiac catheterisation. None of the patients had had chest pain suggestive of angina within the previous three months. No patient had another cause for this electrocardiographic appearance, such as left bundle-branch block or the Wolff-Parkinson-White syndrome, and none had electrocardiographic evidence of myocardial infarction at other sites. All patients had radiological evidence of hyperinflation. Their ages ranged from 44 to 79 years (mean 66 years). There were 11 men and five women.

Respiratory Function Tests

Forced expiratory volume in one second (FEV₁) and forced vital capacity (FVC) were measured using an Autospirometer (AS-700 Minato Medical Science Co. Ltd.) and the maximum of three measurements taken. Lung volumes were measured in a Morgan constant volume body plethysmograph. The diffusing capacity for carbon monoxide (DLCO) was measured by the single breath technique. Two readings were averaged. KCO, the uptake of carbon monoxide per unit lung volume (DLCO/alveolar volume), was calculated.

Results were expressed as percentage of predicted normal.

Thallium-201 Cardiac Scan

An intravenous bolus of 1 to 1.5 Ci of thallium-201 was administered at rest, the patients having fasted. Imaging was started after 10 to 15 minutes. Anterior, 45° and 60° left anterior oblique, and left lateral views were obtained in the supine position using a Toshiba GCA 102S camera with a low energy high resolution parallel hole collimator. A 20 per cent energy window was centred on 75 keV and a minimum of 300,000 counts were collected averaging 400 seconds per view. Analogue scans were obtained on a Matrix 4 Multi-imager using Nuclear Medicine B film. The scans were performed within one week of recording the electrocardiogram and were interpreted without reference to other data. From consideration of all projections, defects in thallium-201 perfusion were defined as anterior (septal), apical, lateral, inferior, or posterior.

The patients were divided into two groups: those with anterior (septal) defects in radionuclide uptake and those without.

Electrocardiograms

These were carefully calibrated (1 mV/cm). Leads V1 to V3 were recorded at the conventional site and one interspace lower. The amplitude of the R wave in lead V3 in both sites was measured to the nearest 0.5 mm. The change in R wave amplitude in lead V3 within each group was analysed according to Student's paired t test. Differences between the two groups were compared by means of the Wilcoxon rank sum test.

Values are expressed as mean ± standard error of the mean.

Results

Respiratory Function Tests (Fig. 1)

All patients had moderate to severe chronic airways obstruction, with a mean FEV₁ of 32 ± 3 per cent of predicted normal, and a mean FVC of 58 ± 3 per cent of predicted normal. Hyperinflation was
evidenced by a mean total lung capacity (TLC) of 143 ± 7 per cent of predicted normal. Diffusing capacity was reduced, with a mean D_{L}CO and KCO of 41 ± 4 per cent and 51 ± 5 per cent of predicted normal, respectively.

THALLIUM-201 CARDIAC SCAN
Seventy patients had anterior (septal) defects of thallium-201 uptake consistent with previous anterior myocardial infarction. Three of these had apical-inferior extension, and two had inferior extension. Nine patients had no defects in anterior thallium-201 uptake. Four of these, however, had apical-inferior defects, one apical alone, and one inferior alone. Only three patients had entirely normal thallium-201 cardiac scans.

ELECTROCARDIOGRAMS (Fig. 2)
(a) Amplitude RV3 in conventional site (C)
The seven patients with a thallium-201 cardiac scan diagnosis of anterior myocardial infarction had a mean RV3 (C) of 0·6 ± 0·2 mm. In the nine patients with a thallium-201 cardiac scan negative for anterior myocardial infarction, mean RV3 (C) was significantly greater at 2·2 ± 0·4 mm (p < 0·005).

Anterior myocardial infarction was present if RV3 (C) was absent (three patients) and absent if RV3 (C) was ≥ 2 mm (six patients). There was an equivocal range of RV3 (C) of 0·5–1·5 mm shown by seven patients (43%).

(b) Amplitude RV3 one interspace lower (L)
Mean RV3 (L) was 1·4 ± 0·4 mm in the seven patients with an anterior myocardial infarction on thallium-201 scanning, and 6·4 ± 1·2 mm in the nine patients without this finding. This difference was highly significant (p < 0·001).

Anterior myocardial infarction was present if RV3 (L) was < 1·5 mm (five patients) and absent if RV3 (L) was ≥ 3·5 mm (seven patients). The equivocal range of RV3 (L) of 2 to 3 mm was found in four patients (25%). Two of these four were correctly classified, however, using criterion (a) and a third using criterion (c) below.

(c) Increase in amplitude RV3 between two recording sites
In the patients with anterior myocardial infarction, the mean increase in the amplitude of the R wave in lead V3 between the two recording sites was 0·8 mm. This was not statistically significant (p < 0·1).

The mean RV3 increase of 4·2 mm in the patients without anterior myocardial infarction, however, was significant (p < 0·005), and this difference between the two groups was also significant (p < 0·005).

Anterior myocardial infarction was present if the increase in the amplitude of the R wave in lead V3 between the two recording sites was < 0·5 mm (six patients), and absent if the RV3 increase was ≥ 3·5 mm (four patients). The equivocal range of the increase in RV3 amplitude of 1 to 3 mm was found in six patients (38%).

Fig. 3a shows the electrocardiogram of a patient with emphysema and poor R wave progression in
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the anterior chest leads so that the amplitude of the R wave in lead V3 in the conventional site was 1 mm. When recorded one interspace lower, however, RV3 became 7 mm. The thallium-201 cardiac scan (Fig. 3b) was normal.

Fig. 4a shows the electrocardiogram of another patient with similar poor R wave progression in the anterior chest leads so that RV3 in the conventional site was again 1 mm. When recorded one interspace lower, however, RV3 increased to only 1·5 mm. The thallium-201 cardiac scan (Fig. 4b) showed an anterior (septal) defect in radionuclide uptake consistent with previous anterior myocardial infarction.

Discussion

Small or absent initial R waves in the QRS complexes in the anterior chest leads V1 to V3 of the electrocardiogram strongly suggest anterior myocardial infarction, but may also be present in emphysema alone.1-3

Although “emphysema” is essentially a pathological diagnosis, the term is commonly used clinically to describe patients with chronic irreversible airways obstruction, radiological evidence of hyperinflation, increased total lung capacity and residual volume, and decrease in the measurements of carbon monoxide transfer, such as Dl,CO and KCO.

The 16 patients in this study fulfilled these criteria, as well as having poor R wave progression on the anterior chest leads, so that the amplitude of the R wave in lead V3 recorded from the conventional site was 4 mm or less.

Confirmation or exclusion of previous anterior myocardial infarction was made by thallium-201 radionuclide resting cardiac scanning. This has been shown to be a reliable method for showing moderate or severe old myocardial infarction, though small
myocardial scars may not be detected. None of the patients had had chest pain suggestive of angina within the previous three months so it was felt that false positive scans caused by ischaemia without infarction were unlikely.

The seven patients with anterior (septal) myocardial infarction, diagnosed by cardiac scan, had a significantly lower amplitude of the R wave in lead V3 as recorded at both the conventional site and one interspace lower, when compared with the nine patients without anterior myocardial infarction. The increase in the amplitude of the R wave in lead V3 between the two sites was not significant in the patients with anterior myocardial infarction, but was highly significant in those without it.

This supports the view that in some patients with emphysema, hyperinflation with descent of the diaphragm results in the conventional anterior chest leads becoming “superior” leads, and thus reflecting dominantly negative QRS complexes, whether anterior myocardial infarction is present or not. When the leads are recorded lower they resume their anterior orientation. In this study the sites of the anterior chest leads were lowered one interspace, though it is possible that in some patients recording from even lower sites would be of value. Excessive lowering of the leads, however, could feasibly result in the production of an inferior orientation, with dominantly positive QRS complexes.

A correct diagnosis of previous anterior myocardial infarction could be made in our study when the R wave in lead V3 in the conventional site was absent, when its amplitude one interspace lower was 1·5 mm or less, or when the increase in the R wave amplitude between the two recording sites was 0·5 mm or less.

Anterior myocardial infarction could be correctly excluded when the amplitude of the R wave in lead V3 in the conventional site was 2 mm or more, when it was 3·5 mm or more one interspace lower, or when the increase in the R wave amplitude between the two sites was 3·5 mm or more.

Of the three criteria examined, the R wave amplitude in lead V3 recorded one interspace lower provided the best diagnostic separation between patients with and without anterior myocardial infarction, with only four patients (25%) in the equivocal range of 2 to 3 mm. Three of these four could be correctly diagnosed electrocardiographically using the other two criteria as well. In comparison, when the conventional lead V3 was considered, an R wave amplitude of 0·5 to 1·5 mm was equivocal, and yet this was a feature of 44 per cent of the whole group.

Thus when patients with emphysema and poor R wave progression in the anterior chest leads are encountered, lead V3 should also be recorded one interspace lower. Approximately 75 per cent of these patients will subsequently have an R wave of less than 2 mm or more than 3 mm in amplitude, indicating the presence or absence, respectively, of associated anterior myocardial infarction. In the equivocal range, the amplitude of the R wave in lead V3 recorded from the conventional site, or the increase in amplitude between that in the conventional site and that one interspace lower, will provide further differentiation. Nevertheless some patients will require further investigation for definitive diagnosis, by radionuclide scanning for example.

In patients with emphysema and an electrocardiogram suggestive of previous anterior myocardial infarction, however, the simple and often forgotten manoeuvre of recording lead V3 one interspace lower will frequently allow a correct diagnosis to be made.

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References

Requests for reprints to Dr P A Barrett, Department of Cardiology, The St George Hospital, Kogarah 2217, Sydney, Australia.
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