Reversible electrocardiographic changes in severe acute asthma

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Siegler, D. (1977). Thorax, 32, 328–332. Reversible electrocardiographic changes in severe acute asthma. Previous reports have documented the occurrence of reversible electrocardiographic changes including right axis deviation, P pulmonale, right bundle-branch block, and ST-segment abnormalities in patients with acute attacks of asthma. In a further systematic study, the electrocardiographs of 63 patients admitted with severe acute asthma have been evaluated. The most consistent change was an abnormally vertical P-wave axis in 78% of the patients. P pulmonale was present in 22% and right ventricular enlargement in only one patient. Right axis deviation, right bundle-branch block, and rhythm abnormality were not present in any patient. In 11%, ST-segment or T-wave abnormalities suggesting myocardial ischaemia were noted. These abnormalities persisted for up to nine days and were unexplained. Other ECG abnormalities in acute asthma may reflect positional changes of the heart due to overdistension of the lungs. All ECG changes resolved after clinical improvement.

Reversible abnormalities in the electrocardiogram are well recognised in patients with acute attacks of bronchial asthma. Previously reported changes have included right axis deviation, prominent P waves suggesting right atrial enlargement, inversion of T waves, right bundle-branch block, and abnormalities of the ST segment (Gunstone, 1971; Re buck and Read, 1971; Da Costa and Chia, 1974). A further systematic study was prompted by observations which conflicted with previously published data. The electrocardiograms of 63 patients with severe acute asthma have been evaluated and attempts made to correlate ECG changes with physiological measurements of the severity of the attack.

Material and methods

The case records of all adult patients below the age of 50 years admitted to the medical intensive care ward of the Brompton Hospital between 1 January 1975 and 31 March 1976 with a confirmed diagnosis of status asthmaticus were studied. These were all patients known to have bronchial asthma for whom admission to hospital was necessitated by an acute attack of asthma failing to respond to their usual maintenance therapy. Patients were included in the study only if an ECG had been performed on the day of admission and thus within the first 24 hours (and usually within the first 12 hours) of their stay in hospital. Patients known to have coronary artery disease, valvular heart disease, or systemic hypertension were excluded. Sixty-three patients were thus included in the study, 34 female and 29 male, with an age range of 15 to 49 years and a mean age of 33 years. In each patient, on admission, plasma urea and electrolytes, arterial blood gases, peak expiratory flow rate, forced expiratory volume in the first second (FEV1), and forced vital capacity (FVC) were estimated. The presence and magnitude of pulsus paradoxus were noted. A standard 12-lead ECG was performed. Where this was abnormal on admission, the ECG in most cases was repeated until it had returned to normal. The ECG was examined for abnormalities of rhythm and of P- or QRS-wave axis. Right atrial enlargement was judged to be present if the height of the P wave in standard leads II and III exceeded 2.5 mm (P pulmonale). Right ventricular hypertrophy was regarded as being present if there was right axis deviation to beyond +110°, the height of the R wave in V1 exceeded 5 mm, and the ratio of R-wave amplitude to S-wave amplitude in V6 exceeded unity (Estes, 1974). The P-wave axis was abnormally vertical, ie, more than 90°, if the P
wave in standard leads II and III was taller than in standard lead I and the P wave was negative in lead aVL (Estes, 1974).

Results

For the whole group of patients the mean arterial Po2±1 SD on admission was 64 mm±15 mmHg (8·5±2 kPa). Mean arterial Pco2 was 38 mm±9 mm Hg (5·1±1·2 kPa). Mean expiratory flow rate was 110±40 litres per minute. Mean FEV1 was 0·73±0·36 litres and mean FVC 1·5±0·78 litres.

The most consistent change was an abnormally vertical P-wave axis in 48 of the patients (78%). In those patients in whom further ECGs were performed once clinical improvement had been obtained, the P-wave axis had returned to normal, usually within three to four days (Figs 1 and 2). The next most common change was the appearance of P pulmonale in 12 patients (22%), again reverting to normal in those patients who had further ECGs. Right ventricular enlargement was present in only one patient. Isolated right axis deviation was not seen in any patient. No rhythm disturbance was noted although a sinus tachycardia in excess of 100 per minute was invariable.

In seven patients (11%) abnormalities of T waves or ST segments were noted. In three of these, T-wave inversion across the chest leads from V1 to V5 was seen, and in one, electrical alternans of the QRS complexes was also present. In the other four subjects depression of the ST segments was noted: in three, this was present in leads II, III, and aVF, and in the remaining one in leads V3 to V6. The ST- and T-wave abnormalities were all reversible with clinical improvement. However, in three patients reversion to normal was slow, requiring two, four, and nine days respectively.

Discussion

The very high incidence of an abnormally vertical P-wave axis in acute asthma has not previously been described, although it is well documented as
an electrocardiographic manifestation of other forms of diffuse chronic airway obstruction. Chapman (1974) examined the ECG of a very large series of patients with diffuse airway obstruction referred for lung function testing. The series did not contain any patients with asthma. Chapman confirmed the finding of Spodick (1959a, b) that a P-wave axis of more than 90° was the only distinctive ECG finding in these patients. He showed a good correlation between a vertical P-wave axis and the degree of airway obstruction as measured by the FEV₁, as a percentage of FVC, and less good correlations with impairment of the carbon monoxide transfer factor and the presence of hyperinflation of the lungs. The cause of the vertical P-wave axis is not certain but probably reflects hyperinflation of the lungs leading to rotation of the heart to a more vertical position. It may be that for some reason the P-wave axis is a more sensitive indicator of this rotation than the QRS axis because in no patient in the present series was abnormally clockwise rotation of the QRS axis noted. Chapman (1974) also suggested that over-inflated lungs conduct the ECG complexes to the anterior chest wall less well than normal lungs, and the normal conducting pathways to the chest wall are thus modified, leading to ECG abnormalities.

The next most common abnormality was the finding of P pulmonale, suggesting right atrial enlargement, confirming the findings of Rebuck and Read (1971) in 6 out of 76 patients and those of Da Costa and Chia (1974) in 8 of 18 patients. Gunstone (1971) performed right heart catheterisation in nine patients with acute asthma. In five of the patients the ECG showed such abnormalities as P pulmonale, right axis deviation, right bundle-branch block, and T-wave inversion. The pulmonary artery pressures were no higher in these patients than in the four remaining patients whose electrocardiograms were within normal limits. Schmack et al. (1971) attempted to correlate the ECG changes in a group of patients with chronic bronchitis and emphysema with postmortem examination of the lungs and heart. They found that in all patients whose ECG demonstrated a P pulmonale, right ventricular en-
largement, P pulmonale was absent. However, in
the present series, in only one patient with P pul-
omalone were there additional ECG criteria of right
ventricular enlargement suggesting that in acute
asthma P pulmonale per se is not a manifestation
of right ventricular enlargement.

In the present study, the most unexpected and
persistent ECG abnormalities were in those
patients with T-wave and ST-segment changes
suggesting myocardial ischaemia. This confirms
the finding of Rebuck and Read (1971), who noted
similar abnormalities in 5 of their 76 patients and
of Da Costa and Chia (1974) in 7 of their 18
patients. It is unlikely that these changes repres-
ent myocardial damage due to hypercapnia or
hypoxaemia. The arterial Pco₂ was normal in all
seven subjects in the present series. In addition,
the changes persisted long after any degree of
hypoxaemia had been corrected, and in only two
patients was the initial arterial Po₂ below the
mean value for the whole group of patients. It is
of interest that Lary and Goldschlager (1974) des-
cribed seven patients in whom exercise tests were
being carried out to confirm a diagnosis of cardiac
ischaemia and in whom hyperventilation led to
‘false-positive’ exercise electrocardiograms with
the development of ST depression, elevation or
flattening without the necessity for exercise.
These authors noted that T-wave inversion had
previously been reported in similar circumstances.
Respiratory alkalosis and abnormal potassium flux
across myocardial cell membranes had already
been excluded as causes of the apparent ischaemic
changes (Barker et al., 1939; Biberman et al.,
1971) and the authors concluded that the appear-
ces could be due to autonomic nervous system
activity or to adrenergic stimulation either therapeutically or by endogenous catecholamine release
which might result in asynchronous repolarisation
on the myocardium. Hyperventilation and endo-
genous or therapeutic sympathomimetic stimula-
tion could certainly explain the appearances of
ischaemia in the first few hours of an attack of
asthma but will not account for the persistence
of these changes for several days as was seen in
the present study. The phenomenon remains un-
explained. In these seven patients as a whole
ECG changes of ‘ischaemia’ could not be cor-
related with differences in blood gases, spirometry
or magnitude of pulses paradoxus from the group
of patients as a whole. Indeed, it was not possible
to correlate any of the ECG abnormalities with
abnormalities of blood gases or spirometry. This
confirms the previous findings of Rao et al. (1974)
in 14 children with acute asthma in whom ECG
changes did not consistently correlate with physio-
logical abnormalities.

The inconsistency between the present series
and previous reports was the absence from the
present series of such changes as right axis devia-
tion and right bundle-branch block. It could be
argued that delays in recording the ECG could
lead to these abnormalities being missed. This is
a theoretical weakness of retrospective studies such
as the present one. Rebuck and Read (1971) have
pointed out that some ECG changes may disappear
within hours. However, any delay in recording the
ECG was unlikely to have made any material dif-
ference to the findings in the present study as some
gross abnormalities did in fact persist for several
days. The invariable persistence of sinus tachy-
cardia suggests further that the heart was in some
way still being influenced by the severe airway
obstruction.

Finally, it is possible that all ECG changes ex-
cept for those suggesting myocardial ischaemia
are in fact explicable on purely anatomical
grounds. Overdistension of the lungs, in addition
to altering the electrical axes, may alter the
position of the heart to such an extent that the
anterior chest leads, for instance, no longer reflect
the activity of that part of the heart which they
would normally individually represent.

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