Relation between Metabolic Acidosis and Cardiac Dysrhythmias in Acute Myocardial Infarction

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In the past 30 years mortality in acute myocardial infarction had remained virtually unchanged, varying between 30 and 40 per cent. Recently, with the advent of the coronary care unit, it has become possible to increase the patient's chance of survival, partly by the more energetic treatment of cardiogenic shock, but largely by the prompt suppression of the minor dysrhythmias which are such a common feature of the early phase of acute myocardial infarctions. These lesser dysrhythmias, as well as reducing circulatory efficiency, may herald the graver disturbances of rhythm such as ventricular tachycardia and ventricular fibrillation, and their prevention would, therefore, be an important factor in improving the prognosis in this disease.

Experimental and clinical experience suggest that extreme metabolic acidosis may be a factor in producing dysrhythmias, even where respiratory compensation has resulted in a normal arterial pH. Metabolic acidosis is known to occur in the early stages of an acute myocardial infarction (Mackenzie *et al.*, 1964; Neaverson, 1966; Kirby and McNicol, 1966), but it is rarely severe except in the presence of profound cardiogenic shock; nevertheless, it could contribute to the development of dysrhythmias.

PATIENTS AND METHOD

Studies were made on 21 patients all of whom had presented with persistent ischaemic cardiac pain; 19 had electrocardiographic changes of recent myocardial infarction and 2 others had diagnostic serial electrocardiographic changes. The serum hydroxybutyric dehydrogenase and/or the aspartate transaminase were raised in all cases. Of the patients, 19 were men whose ages ranged from 41 years to 74 years (mean 60 years) and the 2 women were aged 44 years and 70 years.

The heart rate and rhythm were monitored for at least 72 hours and one-minute electrocardiographic recordings were made hourly. Arterial blood samples were obtained by brachial artery puncture on admission, 24 hours later, and at the onset of any dysrhythmia. A recording was made at the same time and the patient's blood pressure was also noted. The arterial base deficit and blood pH were measured using the Astrup micro-electrode technique (Astrup *et al.*, 1960). Metabolic acidosis due to an actual or potential accumulation of hydrogen ions results in a low pH, PCO_2 , and bicarbonate. Since as a result of compensatory mechanisms a severe metabolic acidosis may be accompanied by a less severe reduction in blood pH, the degree of acidosis was assessed on the value of the base deficit, that is the excess acid concentration of whole blood in mEq/l. over blood of a pH of 7.4 at a PCO_2 of 40 mm. Hg. The patients were graded on the basis of their history, clinical, and electrocardiographic findings, according to the Peel prognostic index (Peel *et al.*, 1962).

RESULTS

Nine patients had a base deficit of more than 2.5 mEq/l. on admission. The presence of metabolic acidosis was associated with a poor early prognosis, 4 of 9 of these patients dying within a week of admission, compared with 1 of the 12 remaining patients (Table I). In the succeeding 3 weeks in hospital 3 more patients died, one of a pulmonary infarct and the other 2 of further episodes of myocardial infarction. The significance of this high mortality rate is discussed later. Of the 9 patients with metabolic acidosis, 7 survived the first day of admission. In 4 the acidosis corrected itself spontaneously within 24 hours; one patient who had been given an intravenous infusion of sodium bicarbonate was alkalotic; and in 2 patients a degree of metabolic acidosis just outside the normal range was present.

The presence of metabolic acidosis on admission

Received November 1, 1967.

FIG.—The relation of hypotension and dysrhythmia to the acid-base state.

TABLE I
7-DAY SURVIVAL IN ACUTE MYOCARDIAL
INFARCTION

Prognostic index	Metabolic acidosis		Others	
	No. patients	No. died	No. patients	No. died
1-12	4	1	7	0
13+	5	3	5	1
Total	9	4	12	1

was found to be invariably associated with hypotension and cold extremities (Fig.). Nine patients had metabolic acidosis and hypotension on admission, while of the 12 patients in a normal acid-base state only one was hypotensive at the time. Of 9 patients who had metabolic acidosis on admission, 6 also had a dysrhythmia at the time, as did 6 of the 12 in a normal acid-base state. Further episodes of dysrhythmia in the first 3 days were much commoner in patients who were acidotic on admission (8 of 9) than in the remainder (4 of 12). The range of dysrhythmias was wide in both groups, with ventricular premature contractions predominating. Sinus bradycardia present in 3 cases was always accompanied by metabolic acidosis; ventricular fibrillation and asystole resulting in circulatory arrest were also predictably accompanied by acidosis.

In all, 21 samples were obtained from 12 patients who developed episodes of dysrhythmia after admission. There appeared to be no close correlation between the onset of dysrhythmias and the patient's acid-base state at the time. In 8 samples there was evidence of metabolic acidosis, and in 11 the results were within normal limits. The 2 remaining samples showed the presence of a metabolic alkalosis, and in these 2 patients previous acidosis had been treated with intravenous sodium bicarbonate. In

all, 3 patients with cardiac dysrhythmias and a marked base deficit were treated with an intravenous infusion of bicarbonate. The general condition of the patients improved, but the dysrhythmias continued and had to be corrected by other means. The relation between these results and the blood pressure is of interest (Table II). Of the normal samples, 2 of 11 were from patients who were hypotensive at the time, while of the 8 with metabolic acidosis 7 were hypotensive at the time. In the 2 patients with metabolic alkalosis the blood pressure was normal. This suggests that where metabolic acidosis occurs at the time of a dysrhythmia, it is probably as a result of circulatory insufficiency rather than the cause of the dysrhythmia itself.

TABLE II
ACID-BASE STATE DURING DYSRHYTHMIA

	No. of samples	Hypotension
Normal	11	2
Metabolic acidosis	8	7
Metabolic alkalosis	2	0

DISCUSSION

The coronary care unit is potentially capable of significantly reducing mortality in acute myocardial infarction, an area where prognosis has not altered appreciably for many years. This is achieved largely by the prevention or reversal of major disturbances of rhythm. In one such unit (Lown *et al.*, 1967), the mortality was 12 per cent of 130 consecutive cases, and in only 2 cases of complete heart block was it possible to attribute the cause of death to a disturbance of rhythm. This striking result was achieved by the immediate recognition and prompt treatment of the minor dysrhythmias commonly seen in acute myocardial infarctions.

It has been shown in the past that patients with

hypotension and pulmonary congestion following an acute myocardial infarction may have a metabolic acidosis in the first 24 hours following the onset of pain (Kirby and McNicol, 1966). Experimental and clinical situations have been described where extreme metabolic acidosis has been associated with a tendency to develop dysrhythmias. Ledingham and Norman (1962) showed that in experimentally induced cardiac arrest when the accompanying acidosis was not corrected, post-arrest dysrhythmias invariably occurred. In those animals in which the metabolic acidosis was carefully corrected the incidence of dysrhythmias was reduced, even when a respiratory acidosis was superimposed by ventilating the animal with carbon dioxide mixtures. Gerst, Fleming, and Malm (1966) have shown that metabolic acidosis results in a reduced threshold for ventricular fibrillation, while metabolic alkalosis protects the heart from this dysrhythmia. Respiratory acidosis and alkalosis did not alter the fibrillation threshold, nor did the superimposition of respiratory alkalosis on metabolic acidosis return the fibrillation threshold to normal. In experimentally induced extreme acidosis a characteristic sequence of electrocardiographic changes, sinus tachycardia, electrical alternans, 2-to-1 block, and asystole has been described (Stewart *et al.*, 1965), together with a similar sequence seen in clinical practice. Reversal of the sequence occurred on correcting the acidosis. Brooks and Feldman (1962) have also described dysrhythmias accompanying post-operative metabolic acidosis, with reversion to sinus rhythm following the infusion of alkali. Harden, Mackenzie, and Ledingham (1963) described a case of ventricular fibrillation which failed to respond to repeated countershocks, but when the accompanying metabolic acidosis was corrected spontaneous reversion to sinus rhythm occurred.

The relation between metabolic acidosis, hypotension, and dysrhythmias is described in 21 patients with acute myocardial infarctions, 7 of whom died in the first 28 days of their illness. The significance of this high mortality is doubtful, since mortality rates in acute myocardial infarctions are notoriously variable, even in successive groups of the same large series. In an editorial on this subject, Grace (1967) describes a single series of acute transmural infarcts which, when divided into 5 consecutive groups of 50 patients each, showed a variation in mortality rate from 10 to 44 per cent. When 200 patients in the same series were divided into 2 consecutive groups of 100 patients the mortality rates were 16 and 35 per cent. In our experience, of the next 31 patients admitted with acute myocardial infarcts after the 21 described above, 6 died, a mortality of 19 per cent.

Metabolic acidosis present on admission was found to be associated with a poor early prognosis and an increased tendency to develop dysrhythmias in the following 3 days. In only 8 of 21 instances was metabolic acidosis found to be present at the time of a dysrhythmia, and correction of the acidosis on 3 occasions did not result in reversion to sinus rhythm. The only minor dysrhythmia always accompanied by metabolic acidosis was sinus bradycardia, present in 3 patients. This dysrhythmia frequently accompanies experimental acidosis and clinical shock, and Peretz *et al.* (1965) have suggested that this may be due to an accumulation of acetylcholine, since cholinesterase functions best at a pH of 7.5 to 8.5 and is inhibited below this level. Metabolic acidosis was found to be closely related to the presence of hypotension and cold extremities and was probably a result of peripheral circulatory insufficiency.

SUMMARY

The relation between metabolic acidosis and cardiac dysrhythmias was studied in 21 patients with clinical, electrocardiographic, and biochemical evidence of acute myocardial infarction. The base deficit was measured in arterial blood samples obtained on admission, 24 hours later, and at the onset of any dysrhythmia, and the patient's blood pressure was noted.

The close association of metabolic acidosis and hypotension was the outstanding feature of these results. Metabolic acidosis was also associated with a poor early prognosis and its incidence rose with a rising prognostic index. The presence of metabolic acidosis is thus a reflection of the severity of the infarct. The apparent predisposition of patients with metabolic acidosis to develop dysrhythmias is probably related to the greater severity of their illness rather than the direct result of the acidosis, particularly since correction of the acidosis, though improving the patient's general condition, does not correct the dysrhythmia.

This work was supported by grants from the National Heart Foundation and the National Health Service.

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