

Acid-base Balance in Acute Gastrointestinal Bleeding*

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Summary

Acid-base balance has been studied in 21 patients with acute upper gastrointestinal bleeding. A low plasma bicarbonate concentration was found in nine patients, accompanied in each case by a base deficit of more than 3 mEq/litre, indicating a metabolic acidosis. Three patients had a low blood pH. Hyperlactataemia appeared to be a major cause of the acidosis. This was not accompanied by a raised blood pyruvate concentration. The hyperlactataemia could not be accounted for on the basis of hyperventilation, intravenous infusion of dextrose, or arterial hypoxaemia. Before blood transfusion it was most pronounced in patients who were clinically shocked, suggesting that it may have resulted from poor tissue perfusion and anaerobic glycolysis. Blood transfusion resulted in a rise in lactate concentration in seven patients who were not clinically shocked, and failed to reverse a severe uncompensated acidosis in a patient who was clinically shocked. These effects of blood transfusion are probably due to the fact that red blood cells in stored bank blood, with added acid-citrate-dextrose solution, metabolize the dextrose anaerobically to lactic acid. Monitoring of acid-base balance is recommended in patients with acute gastrointestinal bleeding who are clinically shocked. A metabolic acidosis can then be corrected with intravenous sodium bicarbonate.

Introduction

It has long been recognized that a metabolic acidosis may accompany traumatic shock (Cannon, 1918; Cournand *et al.*, 1943). This was first described in battle casualties during the first World War. A metabolic acidosis has also been shown to accompany shock of purely haemorrhagic origin induced experimentally in dogs (Root *et al.*, 1947). Despite these findings, and the clinical observation that a severe haematemesis may be followed by "air hunger" (Deller, 1948), acid-base balance has been little studied in acute gastrointestinal bleeding, though some clinical studies of lactic acidosis have included isolated cases of shock due to gastrointestinal bleeding (Davidson *et al.*, 1946; Huckabee, 1961; Peretz *et al.*, 1964).

Patients and Methods

Acid-base balance was studied in 21 patients (17 men and 4 women) with acute upper gastrointestinal bleeding. Their ages ranged from 20 to 85 (mean 55) years. Eleven had a chronic duodenal ulcer (accompanied by a chronic gastric ulcer in one patient), two had an acute gastric ulcer, one a stomal ulcer,

one a probable Mallory-Weiss lesion, one a small-bowel reticulosis, and one had thrombocytopenia. In four patients the cause of bleeding was not identified. Arterial blood samples were taken within 36 hours of hospital admission, with the patients breathing air. No patient received any drugs known to cause a lactic acidosis. Though patients were supine at rest when the blood samples were taken, conditions were not truly basal, as some of them were anxious and not all were fasting. In 14 patients an arterial blood sample was taken before blood transfusion, and in seven the first sample was taken during blood transfusion.

The blood samples were analysed immediately for pH, carbon dioxide tension (PCO_2), and oxygen tension (PO_2) with a Radiometer electrode system at 37°C. Plasma bicarbonate concentration and base deficit were derived from pH and PCO_2 by use of Siggaard-Andersen's (1963) nomogram. An aliquot of the blood sample was deproteinized at the bedside with ice-cold perchloric acid. Blood lactate and pyruvate concentrations were measured by means of a lactate dehydrogenase enzyme assay, the reagent kits supplied by Biochemica Test Combination being used as specified by the manufacturers (Boehringer und Soehne, 1967).

For the purpose of this study arterial hypoxaemia has been defined as an arterial oxygen tension of less than 80 mm Hg, and clinical shock as the presence of cold, sweaty extremities in combination with a systolic blood pressure of less than 100 mm Hg.

Results

Values for pH, PCO_2 , and plasma bicarbonate concentration in the initial blood samples from all 21 patients are shown in Fig. 1. Nine patients had a plasma bicarbonate concentration below 20 mEq/l. and all nine had a base deficit of more than 3 mEq/l. This was accompanied in each case by a PCO_2 below

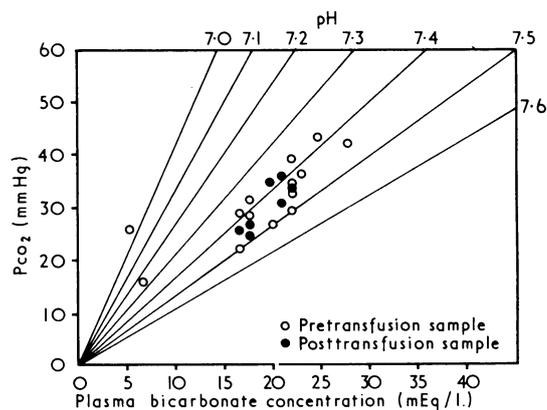


FIG. 1—Arterial pH, PCO_2 , and bicarbonate concentration.

36 mm Hg. In most cases the pH remained within normal limits (7.35-7.45), but in two patients who were severely shocked and semicomatose on admission the pH was 6.94 and 7.25 in the initial blood sample. A pH of 7.23 was recorded subsequently in a third patient during an episode of severe recurrent haemorrhage.

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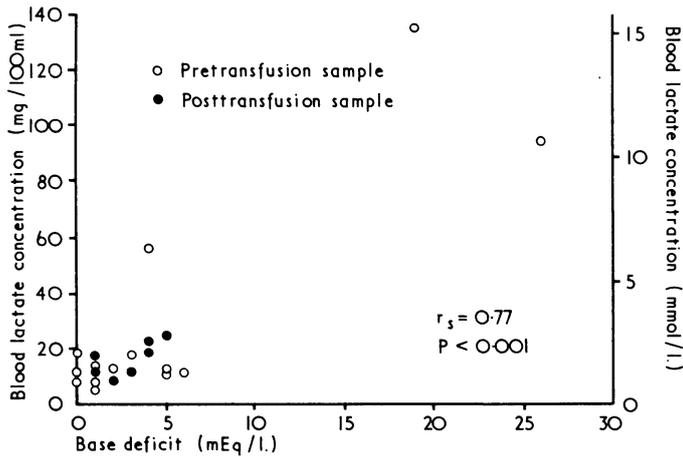


FIG. 2—Correlation between base deficit and blood lactate concentration.

The association between base deficit and blood lactate concentration in the same 21 patients is shown in Fig. 2. All nine patients with a base deficit of more than 3 mEq/l. had a blood lactate concentration of more than 7 mg/100 ml (0.8 mmol/l.). There was a significant correlation between base deficit and blood lactate concentration (with Spearman's rank correlation test, $r_s = 0.77$; $P < 0.001$). Blood lactate concentration has been expressed in mmol/l. in addition to mg/100 ml to allow direct comparison with base deficit concentration in mEq/l. The increase in blood lactate (11 and 15 mmol/l.) would account for much of the acidosis in the two patients with the most severe acidosis (base deficit 26 and 19 mEq/l. respectively).

Blood pyruvate concentration was measured in 12 samples from 10 patients. It was below 1.3 mg/100 ml in all 12, including four having a blood lactate concentration of more than 30 mg/100 ml. All the samples had a lactate: pyruvate ratio of more than 10:1. There was no significant correlation between blood lactate and pyruvate concentration ($r = +0.028$).

Arterial P_{O_2} was measured in the initial blood sample of 19 patients. Nine patients had arterial hypoxaemia (P_{O_2} 62-80 mm Hg, mean value 68 mm Hg). Seven of these had chronic bronchitis, one had bronchopneumonia, and the ninth had no known chest disease but was aged 85. Ten patients did not have arterial hypoxaemia (P_{O_2} 87-125 mm Hg, mean value 99 mm Hg). Only two of these had known chest disease. The mean arterial lactate concentration was lower in patients with arterial hypoxaemia (20 ± 13 mg/100 ml) than in those without arterial hypoxaemia (34 ± 46 mg/100 ml) ($0.2 < P < 0.5$).

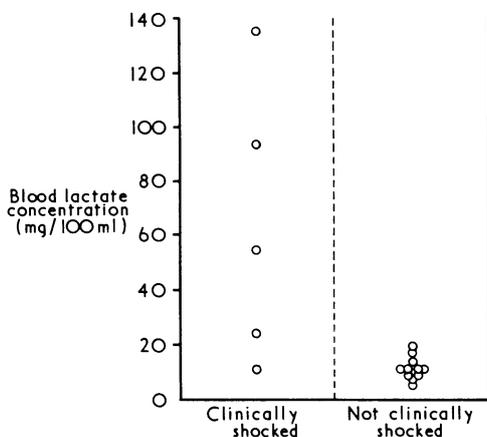


FIG. 3—Relationship between blood lactate concentration and the presence of clinical shock (pretransfusion blood samples only).

The relationship between blood lactate concentration and the presence of clinical shock in patients studied before blood transfusion is shown in Fig. 3. Represented are the 14 untransfused patients shown in Figs. 1 and 2, together with two additional patients on whom acid-base data are not available. The mean blood lactate concentration in those who were clinically shocked was 65 mg/100 ml and in those who were not clinically shocked it was 11 mg/100 ml ($P < 0.005$).

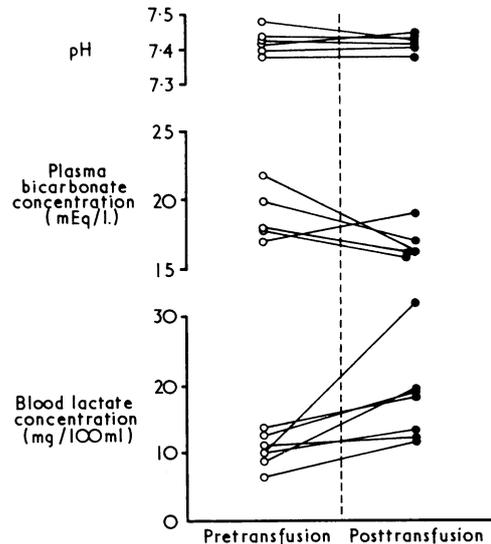


FIG. 4—Effect of blood transfusion.

Several posttransfusion blood samples contained a high blood lactate concentration in the absence of clinical shock. The effect of blood transfusion on blood lactate concentration was therefore studied in seven patients who were not clinically shocked either before or after blood transfusion (Fig. 4). The average volume of blood transfused was 1,250 ml (range 500-2,000 ml), and the average time interval between the two blood samples was nine hours (range 1-24 hours). The mean lactate concentration rose from 10 to 18 mg/100 ml ($P < 0.05$). The mean plasma bicarbonate concentration fell from 19 to 17 mEq/l. in five patients, but this fall was not significant ($0.1 \pm P < 0.2$). The pH remained within the normal range in all six patients in whom it was measured both before and after transfusion.

The effect of blood transfusion was studied in one patient who had a severe metabolic acidosis in association with clinical shock before blood transfusion. Transfusion of 2 pints (1,140 ml) of blood in one hour reversed the state of clinical shock, with a rise in blood pressure from 90/60 to 130/70. There was, however, no real change in acid-base balance; blood pH rose from 7.25 to 7.26 and plasma bicarbonate concentration from 7 to 8 mEq/l. Blood lactate concentration fell from 137 to 128 mg/100 ml. In view of the persistence of this severe degree of acidosis the blood pH was then restored to normal with 200 mEq of intravenous sodium bicarbonate.

Discussion

Nine of the 21 patients studied had a metabolic acidosis, as indicated by a base deficit of more than 3 mEq/l. This could be largely accounted for by the associated hyperlactataemia. As blood pyruvate concentration was not raised, it is unlikely that the hyperlactataemia was due to either hyperventilation or intravenous dextrose infusion (Huckabee, 1958). It could be explained on the basis of (1) poor tissue perfusion and (2) the effect of blood transfusion.

There was an association between hyperlactataemia and the presence of clinical shock in patients studied before transfusion. This suggests that the lactic acid has resulted from tissue hypoxia and consequent anaerobic glycolysis. It may, however, be more directly related to high levels of circulating catecholamines, as has been suggested in experimentally induced haemorrhagic shock (Halmagyi *et al.*, 1967). Arterial hypoxaemia cannot have been an important factor in the present study as the lactate concentration tended to be higher in the patients with a normal arterial oxygen tension. Moreover, the mild degree of hypoxaemia in the other patients (62-80 mm Hg) would not, on the basis of experimental work in animals, be expected to cause lactic acidosis, even in the presence of hypocapnia (Takano, 1968).

The results in the present study suggest that blood transfusion itself can cause a rise in blood lactate concentration. This is in accordance with experiments on animals with haemorrhagic shock, where transfusion with bank blood has caused a fall in blood pH (Nahas *et al.*, 1961). An explanation for these observations is provided by the fact that red blood cells in stored bank blood, with added acid-citrate-dextrose (A.C.D.), metabolize the dextrose anaerobically to lactic acid (Gullbring and Ström, 1956; Nahas *et al.*, 1961). It has been found that the lactate concentration exceeds 100 mg/100 ml after 10 days' storage (Gullbring and Ström, 1956), and that the pH falls to 6.2 after three weeks' storage (Nahas *et al.*, 1961). These observations may have therapeutic implications, since acidosis has been implicated (Gain, 1962) as one of the factors contributing to the high incidence of cardiac arrest following massive blood transfusion (Howland *et al.*, 1956; Le Veen *et al.*, 1960; Boyan and Howland, 1963). Infusion of alkali has been shown to decrease the mortality rate following rapid transfusion in the experimental animal (Nahas *et al.*, 1961) and in patients undergoing major surgery (Howland and Schweizer, 1965).

The results in the present investigation suggest that monitoring of acid-base status is advisable in patients with acute gastrointestinal bleeding who are clinically shocked, especially if rapid blood transfusion is contemplated. It is probably

unnecessary in patients who are not clinically shocked. A metabolic acidosis is unlikely to be rapidly corrected by blood transfusion alone, but can be corrected with intravenous sodium bicarbonate solution.

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Gas Exchange in Renal Failure

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I. Dangers of Hyperkalaemia during Anaesthesia

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Summary

Failure to maintain compensatory hyperventilation during anaesthesia in patients with metabolic acidosis results in an increase in PaCO₂, fall in blood pH, and a possible rise in plasma potassium. This sequence of events may account for unexplained operative deaths in patients in renal failure.

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Introduction

Renal failure is the commonest background for the presence of a metabolic acidosis. Anaesthesia in patients with metabolic acidosis has long been considered a serious risk, quite independent of the anaemia which is almost invariably present. Fatalities during anaesthesia have not been adequately explained. In some instances hyperkalaemia has been accepted as a cause for cardiac arrest. We have lost several patients undergoing quite minor surgery, such as replacement of an external arteriovenous shunt. In some instances an electrocardiogram immediately before anaesthesia showed no evidence of hyperkalaemia, though the plasma potassium was above the normal range.

All patients with severe renal failure are likely to have respiratory compensation for the metabolic acidosis, with the PaCO₂ well below normal. Control of respiration during anaesthesia tends to be based on the known minute volume