Successful resuscitation in diabetic ketoacidosis:
a strong case for the use of bicarbonate

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Summary
Although severe acidosis is a life-threatening condition owing to its cerebral and cardiac effects, correction of the acidosis with alkali remains a controversial issue in diabetic ketoacidosis. A 22-year-old female patient in diabetic ketoacidosis who was successfully resuscitated after cardiac arrest is presented and a strong case is made for the administration of bicarbonate in severe cases of diabetic ketoacidosis.

The use of bicarbonate in the management of diabetic ketoacidosis has remained a controversial issue. Insulin leads to a reversal of the hydrogen ion accumulation of ketoacidosis and physicians at the Joslin Clinic have argued that alkalis are not a critical part of treatment (Bradley, 1965; Young and Bradley, 1967; Bradley, 1971). On the other hand, some workers (Asfeldt, 1965; Hockaday and Alberti, 1971; McGarry and Foster, 1972) have recommended correction of the acidosis with alkali in selected cases whilst others (Soler et al., 1973) employ bicarbonate routinely for partial correction of the acidosis. A number of considerations are put forward against the administration of sodium bicarbonate; potassium is shifted back into the cells and hypokalaemia may result (Burnell, 1956), correction of the systemic acidosis can lead to a paradoxical worsening of the CSF acidosis (Posner, Swanson and Plum, 1965; Ohman et al., 1971) which in turn increases cerebral blood flow and contributes to increased intracranial pressure (Lassen, 1966); the acidosis compensates for the low red cell levels of 2 : 3 diphosphoglycerate (Guest, 1942) and helps to maintain a normal oxygen dissociation curve, but administration of alkali results in a rise of haemoglobin affinity for oxygen and tissue hypoxia follows (Bellingham, Delter and Lefant, 1970, 1971). We feel that the following case provides strong evidence that correction of the acidosis with bicarbonate is important in diabetic ketoacidosis despite considerations to the contrary.

Case report
A 22-year-old female patient was admitted to hospital in diabetic ketoacidosis. Since the diagnosis of diabetes 9 years previously her control had never been satisfactory and emotional upsets had recently led to further deterioration. She gave a history of thirst and polyuria that had developed overnight and she had also been very sick. On examination, she was fully conscious, over-breathing, dehydrated and vomiting. Her temperature was 35°C, pulse rate 120/min and systolic blood pressure 90 mmHg.

Investigations: Blood sugar 774 mg/100 ml, pH 6.85, Pco2 35 mmHg, standard bicarbonate 6 mEq/l, blood urea 70 mg/100 ml, serum sodium 130 mEq/l, serum potassium 5.8 mEq/l. The serum reacted strongly positive when tested with Ketostix. Her ECG showed a sinus tachycardia.

Treatment was started immediately (Table 1) with 100 units of soluble insulin intravenously and an infusion of isotonic saline containing 30 mEq of potassium chloride/l. In 1 hr she received 1500 cc of isotonic saline with 45 mEq of potassium chloride and her serum potassium fell to 4.5 mEq/l. As she continued to vomit, it was decided to pass a Ryle's tube to empty the stomach and a nurse performed this procedure with a doctor in attendance. The patient struggled and pulled out the tube when in reached her throat, but there was nothing to suggest that she had inhaled vomit. A second attempt to introduce the Ryle's tube met with no resistance and it immediately became obvious that the patient had developed cardiac arrest, the ECG monitor showing ventricular fibrillation. Resuscitation commenced immediately with external cardiac massage and following endotracheal intubation manual inflation of the lungs with 100% oxygen. An initial direct current shock of 100 J was applied, but cardiac asystole ensued. Following 0.4 mg of isoprenaline intravenously ventricular fibrillation recurred. Further direct current shocks of 200 and 250 J respec-
Case reports

Table 1. Resuscitation of a ketoacidotic patient

<table>
<thead>
<tr>
<th>Time (hr)</th>
<th>Blood sugar mg/100 ml</th>
<th>Astrup pH</th>
<th>Pco2 mmHg</th>
<th>HCO3 mEq/l</th>
<th>Serum potassium mEq/l</th>
<th>Insulin† units</th>
<th>Fluids vol.‡ (l)</th>
<th>Bicarbonate mEq</th>
<th>Potassium mE</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>774</td>
<td>6.85</td>
<td>35</td>
<td>6</td>
<td>5.8</td>
<td>4.5</td>
<td>100</td>
<td>1.5</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>650</td>
<td>7.21</td>
<td>27</td>
<td>12</td>
<td>4.9</td>
<td>200</td>
<td>1.8</td>
<td>300</td>
<td>145</td>
</tr>
<tr>
<td>4</td>
<td>340</td>
<td>7.34</td>
<td>30</td>
<td>17.5</td>
<td>3.5</td>
<td>3.3</td>
<td>480</td>
<td>190</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiac rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus Ventricular fibrillation</td>
</tr>
<tr>
<td>Supraventricular tachycardia</td>
</tr>
<tr>
<td>Sinus</td>
</tr>
</tbody>
</table>

* The figures shown represent total amounts administered at the end of 1, 2 and 4 hr of treatment respectively.
† Two intravenous boluses of 100 units of insulin each were given at 0 hr and at 2 hr.
‡ The fluids given during the first 4 hr of treatment were: 1·5 l in saline, 0·3 l of 8·4% NaHCO3 and 1·5 l 11% NaHCO3.

Continuously were used, in each case cardiac asystole resulting and ventricular fibrillation recurring following intravenous isoprenaline. An intravenous bolus of 120 mg of lignocaine did not affect the ventricular fibrillation. At this stage external cardiac massage and inflation of the lungs were continued whilst infusing rapidly 100 cc of 8·4% sodium bicarbonate with 40 mEq of potassium chloride. Another shock of 250 J was applied, but asystole resulted and intravenous isoprenaline had to be used to stimulate the myocardium once more. As the cardiac monitor again showed ventricular fibrillation a further 200 cc of 8·4% sodium bicarbonate with 60 mEq of potassium chloride were infused using continuous ECG monitoring to follow the cardiac response. After this infusion of bicarbonate a direct current shock of 250 J converted the ventricular fibrillation into a supraventricular tachycardia and an effective cardiac output was re-established exactly 1 hr after the start of this cardiac emergency. When a further 1500 cc of 1% sodium bicarbonate with 45 mEq of potassium chloride were infused over the next 2 hr a spontaneous return to sinus rhythm occurred. The pH was now 7.34, Pco2 30 mmHg, standard bicarbonate 17·5 mEq/l, serum sodium 147 mEq/l and serum potassium 3·5 mEq/l.

After this near disaster there were no further setbacks in this patient's progress. X-rays of her chest showed clear lung fields but her SGOT rose to maximum of 1540 units 24 hr after the cardiac arrest. She was discharged home fully recovered within 1 week.

Discussion

Resuscitation of ketoacidotic patients who develop cardiac arrest has received little attention in the literature so far, although Abramson and Arky (1966) reported success in one of their patients who was resuscitated following open chest cardiac massage. Recent reports (Soler et al., 1973; Jewett, 1973) confirm that unexpected cardiac arrest does claim the lives of some ketoacidotic patients and suggest that this occurrence may be more frequent than previously recognized. Such cases may have been mislabelled in the past as episodes of delayed vasomotor collapse (Daughaday, 1958; Greenaway and Read, 1958; Hudson, Brick and Martin, 1960). There are a number of possible causes for cardiac arrest in this diabetic emergency. Changes in potassium balance alter the electrophysiological properties of the heart and hypokalaemia may lead to a variety of arrhythmias including ventricular fibrillation (Fisch, 1973). In addition, hypoxia and hypothermia, which sometimes complicate severe ketotic cases as well as the acidosis, all lower the myocardial threshold to ventricular fibrillation (Covino and Beavers, 1957; Gerst, Fleming and Malm, 1966).

The essential principles of resuscitation are the same for the ketoacidotic patient as for any other patient. After cardiac arrest the sooner the airway is established and external cardiac massage commenced the greater are the chances of successful resuscitation. In this case we were helped by prompt recognition of the cardiac arrest and by the patient's lean build which enabled us to apply effective external cardiac massage. It was also very useful to know the patient's serum potassium immediately prior to the cardiac arrest as potassium balance is always of critical importance in ketoacidosis. Although this patient's serum potassium was normal, her total body potassium must have been depleted at this early stage of treatment and when infusing large doses of bicarbonate we were careful to administer potassium in order to avoid a catastrophic drop in the serum potassium. Direct current shocks used in an attempt to revert the ventricular fibrillation led to asystole until the acidosis had been partially corrected with alkali. Other instances of metabolic acidosis from various causes have been described in which repeated counter shocks have been ineffective in restoring cardiac output until the acidosis has been reversed (Brooks and Feldman, 1962; Harden and Macken-
Case reports

467

zie, 1963; Stewart, 1964). It is also interesting that when our patient developed supraventricular tachycardia the dysrhythmia spontaneously reverted to sinus rhythm following further administration of bicarbonate. The use of bicarbonate leads to a restoration of intracellular potassium (Burnell et al., 1956) which may account for the return to normal cardiac rhythm (Fisch, 1973).

Insulin, when given in sufficient amounts, leads to a reversal of ketoacidosis, but the effect is delayed. This case highlights the importance of correcting the acidosis of severely ill ketoacidotic patients with alkali. Extreme acidosis is a life-threatening condition because of its cerebral (Posner and Plum, 1967) and cardiovascular effects. In metabolic acidosis the myocardial threshold to ventricular fibrillation is lowered and is directly related to the degree of acidosis (Gerst et al., 1966). Howarth, McMichael and Sharpey Schafer (1948) reported that cardiac output is not reduced in diabetic ketoacidosis whilst total peripheral resistance is lowered. However, more recent work indicates gross deterioration of cardiac function in severe acidosis (Opie, Kadas and Gevers, 1963) and peripheral venous constriction (Harvey et al., 1966) so that fluid administration without correction of the acidosis can lead to pulmonary oedema. It has been suggested that insulin resistance is a function of the acidosis (Walker, Phear and Martin, 1963) in which case the use of bicarbonate in ketoacidosis may increase the sensitivity of the patient to insulin. Whilst in mild cases of ketoacidosis administration of alkali leads to a quicker improvement in the patient's general condition and symptomatic relief of the distressing overbreathing (Addis, Thomson and Welch, 1964), in severely ketoacidotic patients with a standard bicarbonate < 10 mEq/l correction of the acidosis with bicarbonate can be life-saving. About 185 mEq of bicarbonate are usually sufficient for partial correction of the acidosis (Zimmett, Taft and Ennis, 1970) but severe cases may require considerably more (300–500 mEq), a view supported by recent experimental work (Garella, Dana and Chazan, 1973). Although the use of alkali may cause a worsening of cerebrospinal fluid acidosis (Posner et al., 1965; Ohman et al., 1971), in our hands partial correction of the systemic acidosis with 1% sodium bicarbonate has been found remarkably safe. If bicarbonate is infused with adequate potassium replacements hypokalaemia is not a danger (Soler et al., 1972) and in practice 1% sodium bicarbonate containing 30 to 40 mEq of potassium chloride/l is suitable for routine use (Soler et al., 1973). In this cardiac emergency 8.4% sodium bicarbonate was administered to reverse the acidosis quickly, but we usually avoid this concentrated solution as it can be highly dangerous; not only can hypokalaemia ensue rapidly and prove difficult to correct, but hypernatraemia may also follow and lead to increased osmolarity and a deterioration of consciousness.

References


Diabetes insipidus and marked elevation of foetal haemoglobin in a case of acute leukaemia

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Summary
A case of acute myelomonocytic leukaemia is described in which diabetes insipidus was the presenting symptom.

Case report
A 46-year-old man first presented in Iran, in October 1972, with a history of polyuria, anorexia and fever for 2 weeks. He had lost 18 kg weight in the preceding year. On examination he was found to be anaemic (Hb 6-5 g), with hepatomegaly of 3-4 cm and a 'palpable' spleen. The urinary specific gravity was 1-002. Bone marrow at that time showed 'many abnormal cells'. A diagnosis was made of diabetes insipidus secondary to a malignant process and he was treated with 5 i.u. vasopressin tannate nocte with considerable improvement in the polyuria. This was subsequently changed to nasal pitressin t.d.s. in view of increasing pain at injection sites. He was transfused with three units of whole blood and transferred to Hammersmith Hospital for further investigation.

When first seen on 21 November 1972 he was found to have a Hb of 7-8 g, WCC 3000 (30% neutrophils, 20% blasts, 33% lymphocytes, monocytes 9% and eosinophils 8%), and platelets 192,000. Bone marrow showed acute myelomonocytic leukaemia. Alkaline denaturation showed 47% resistant Hb; Hb A2 estimation was 1-2% and electrophoresis showed an Hb A/F pattern. The patient was therefore admitted for further investigation.

During the first week in hospital the nasal pitressin stopped, and his mean urinary output was 6 l/day, average SG 1000 and average osmolality 1-05, and 260 for urine and plasma respectively. Detailed investigation of his hypothalamic-pituitary-adrenal function was carried out. A poor response to intravenous thyroid-releasing hormone was obtained, the TSH values at 0, 20 and 60 min following injection being 1-6, 2-5 and 2-8 μU/ml respectively. There was a normal basal level of luteinizing hormone and follicular stimulating hormone and following intravenous thyroid-releasing hormone was obtained, the TSH values at 0, 20 and 60 min following injection being 1-6, 2-5 and 2-8 μU/ml respectively. There was being 3-8, 22-0 and 28-0 respectively. However, serum FSH levels measured in mU/ml were 4-4, 5-9 and 7-6 respectively showing a poor and slow response of follicular-stimulating hormone. This is in keeping with the 'hypothalamic pattern of response' (Hall

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References


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doi: 10.1136/pgmj.50.585.465

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