

Alkalosis in Burns in Children

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Summary

The acid-base changes in 14 children with severe burns were studied for varying periods after resuscitation. A long-continued metabolic alkalosis was found, which may be due to increased adrenocortical activity.

Introduction

The changes in acid-base state shortly after severe burns have been extensively studied and it is well recognized that a metabolic acidosis develops rapidly during the shock phase. However, relatively little attention has been given to the later changes in acid-base metabolism. In this investigation we did serial estimations of the acid-base state after initial resuscitation. Since little information was available about the range for blood pH, standard bicarbonate, and PCO_2 in normal children, we took a single blood sample, before elective surgery, in a group of children in order to obtain a comparable series of normal values for our age range.

Patients and Methods

Over a period of 12 months we made serial, usually daily, estimations of the acid-base state on 14 children admitted to Sheffield Children's Hospital with burns of 10% or more of their body surface. The average period of study was 40 days, the shortest being five days and the longest 50 days. The age range was from nine months to eight years and 57% of the children were between one and four years. We took arterialized capillary blood samples during the mid-morning and the pH was measured with Radiometer pH meter (Siggaard Andersen *et al.*, 1960), making temperature corrections when necessary (Siggaard Andersen, 1963). The standard bicarbonate and carbon dioxide tension (PCO_2) were derived according to the method and nomogram of Siggaard Andersen and Engel (1960). Estimations of plasma sodium, potassium, and urea were also made at the same time as the acid-base determinations.

All patients were resuscitated during the first 48 hours after the burn with reconstituted freeze-dried plasma in amounts according to the formula of Muir and Barclay (1963). We excluded from this series all patients with known or suspected burns of the respiratory tract. Local treatment of the burned area was by exposure followed by the application of 0.2% chlorhexidine cream. Children with very extensive burns usually required intravenous fluids during the first week, mainly 0.45% sodium chloride in 2.5% glucose. The total fluid intake was adjusted to maintain a minimum output of urine equivalent to 25 ml an hour, making appropriate reduction for the age and size of each child. No alkalinizing or acidifying preparations or fluids were used, with the exception of the citrate contained

in the plasma. The alkalinizing effect of this citrate would not have influenced acid-base metabolism after the period of resuscitation (E. Lenton, unpublished). Hyponatraemia and hypokalaemia were corrected as soon as they were observed. Two children with extensive burns (55% and 60% of body surface) died.

During the same period 94 children who were admitted for elective operation for tonsillectomy, herniotomy, and various plastic and ophthalmic conditions had a single preoperative estimation of their blood pH, standard HCO_3 , and PCO_2 . The same methods were used as in the children with burns. The age range of these children was from three months to 13 years, with 42% between the ages of one and four years.

Results

The values for blood pH, standard HCO_3 , and PCO_2 obtained in the 94 control patients are shown in Fig. 1; the means were as follows: pH 7.388 (S.D. 0.027), standard HCO_3 22.6 mEq/litre (S.D. 1.75), and PCO_2 37.8 mm Hg (S.D. 4.4). The mean pH is slightly lower than the accepted normal for adults and the range for standard HCO_3 is similar to that found in children by Vokac and Vavrova (1968), taking into account that they used venous blood. The mean values for pH, standard HCO_3 , and PCO_2 for the first 28 days after the burn are shown in Fig. 2. The PCO_2 values are symmetrically distributed about the mean for the controls, but pH and standard HCO_3 are considerably raised. It should be emphasized that the value given for each day is the mean of a number of results from different patients and therefore there is not the same chemical and biological

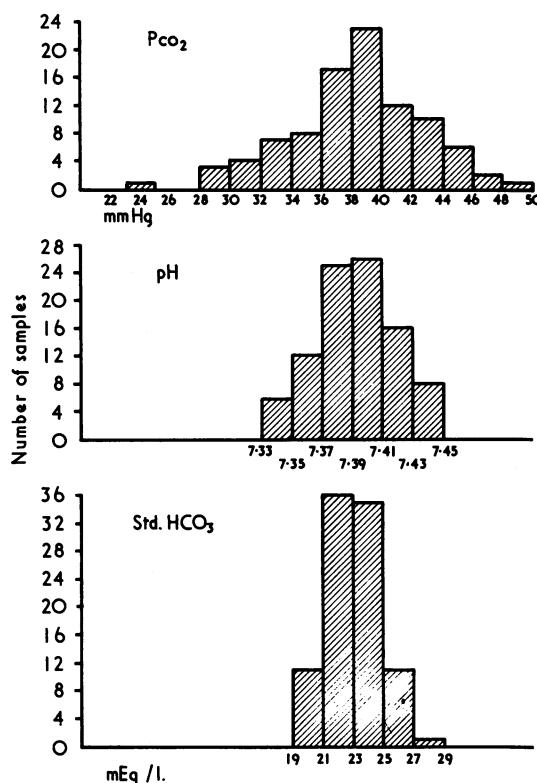


FIG. 1—Values for blood pH, standard HCO_3 , and PCO_2 in 94 control patients.

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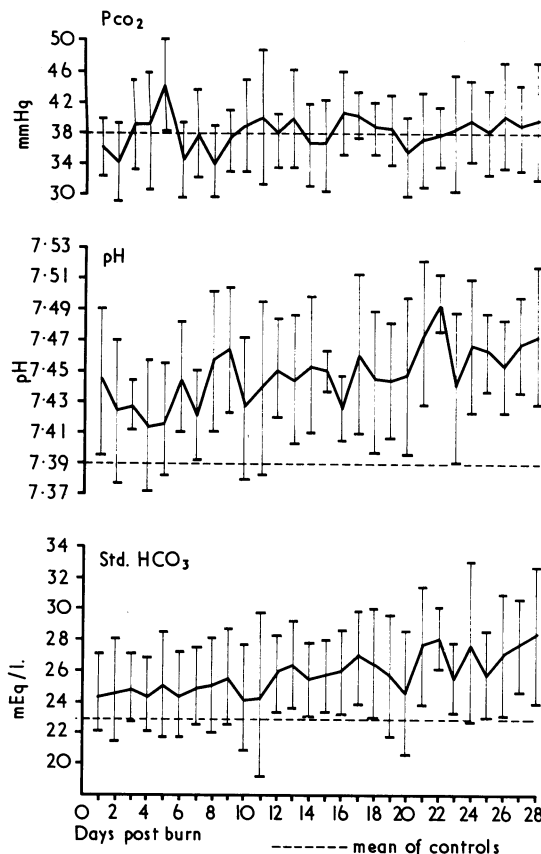


FIG. 2—Mean values for pH, standard bicarbonate, and Pco_2 , plus and minus one standard deviation, for the first 28 days after the burn.

relationship between pH, standard HCO_3 , and Pco_2 as exists when these values are obtained from a single blood sample. Nevertheless a detailed analysis of the serial results in each patient showed a progressively increasing metabolic alkalosis, with occasional periods of respiratory alkalosis superimposed. In some children this metabolic alkalosis persisted until the burn was almost healed. In Fig. 3 are shown the typical changes

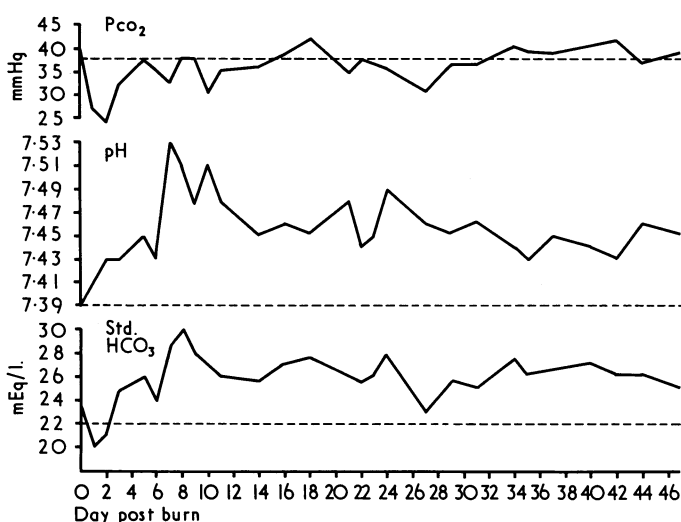


FIG. 3—Changes found in 3-year-old girl with a 22% scald.

which occurred in a 3-year-old child with a 22% scald. No systematic study of urine pH was made in these cases, but random samples examined during the period of investigation showed a pH between 6.5 and 7.5.

Discussion

Part of the reaction to injury is a metabolic alkalosis, which is thought to be due to increased adrenocortical activity. Moore (1955) showed that a similar metabolic alkalosis can be produced by the injection of corticotrophin into healthy volunteers. In contrast to an operation or a single episode of trauma a burn represents a state of prolonged or repeated stress. The plasma 17-hydroxycorticosteroids are considerably raised in these patients (Hume *et al.*, 1956). The metabolic alkalosis which we have observed may therefore be due to stimulation of the adrenal cortex. Since hypokalaemia was promptly corrected it is unlikely that potassium deficiency could have contributed to the alkalosis, though this possibility can be completely excluded only by balance studies or by tissue analysis.

The cause of the episodes of respiratory alkalosis in some cases is difficult to determine but this form of alkalosis may possibly be related to the very high excretion of catecholamines which is found in burns and which may reach levels as high as those found in pheochromocytoma (Goodall *et al.*, 1957; Birke *et al.*, 1957-8; Harrison, 1967). Whelan and Young (1953) showed that adrenaline and noradrenaline, if given intravenously, produce an increase in respiratory rate and tidal volume. However, Lundholm and Svedmyr (1966) found that intravenous noradrenaline caused a transient respiratory alkalosis, but that adrenaline produced a metabolic acidosis with stimulation of respiration due to an increased production of CO_2 .

Though it is clear that repeated estimations of the acid-base state and plasma electrolytes are essential for the proper management of patients with severe burns, we have no clinical evidence that a metabolic alkalosis, even of the severity reported here, is harmful. For this reason we made no attempt to correct the alkalosis. Nevertheless we feel that there are many aspects of the metabolic response to severe stress that require further investigation.

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