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Lacticacidosis: A Clinically Significant Aspect of Shock

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ABSTRACT

A study was made of the metabolic acidosis of hypotensive shock in 25 patients in an attempt to elucidate its etiology and to determine if the degree of acidosis might be a good parameter for the evaluation of treatment and prognosis.

Blood lactate was elevated (> 1.3 mEq./l.) in 24 of 25 patients in hypotensive shock. There was a good correlation ($r = 0.83$, $p < 0.01$) between rising blood lactate and decrease in serum bicarbonate and arterial pH, early in shock. These data indicate that the metabolic acidosis of early shock is largely due to lactate ion. Evidence is presented that high blood lactate levels early in shock are indicative of poor prognosis.

THE association of metabolic acidosis and increase in blood lactate with shock in both animals and man has been well documented in the literature.[1-6, 13] Whether the acidosis occurring in shock is due to accumulation of lactate radicals alone, or whether other acid radicals are also involved has not been shown. A study was made of patients in clinical shock to clarify this point.

Study of both clinical and experimental shock is hindered by the absence of an adequate index of severity and an adequate method of early assessment of the results of treatment in individual patients. The degree of shock at any time is usually judged by measurement of blood pressure. This, however, is as much dependent on the extent of peripheral vasoconstriction as on the cardiac output, and in the presence of marked vasoconstriction, gross underperfusion of tissues may be present in the absence of significant hypotension. A better index of the severity of shock might be provided by measurement of blood lactate. Excessive reduction in tissue blood flow would be expected to cause tissue hypoxia with an increase in lactate production.[7] Furthermore, lactate is largely metabolized in the liver and reduction of hepatic flow might also contribute to an elevation of blood lactate. A systematic study of blood lactate was thus made to test whether it correlated well with the severity of the shock syndrome.

MATERIALS AND METHODS

In this study shock was defined as a systolic blood pressure below 90 mm. Hg with tachycardia and sweating. There was usually an associated peripheral vasoconstriction. Lacticacidosis was arbitrarily designated as a blood lactate level in excess of that found in humans at rest (1.33 mEq./l., 12 mg./100 ml.) associated with an arterial pH less than 7.3 when corrected to a pCO_2 of 40 mm. Hg.

This report is based on experience with 25 patients with shock. The shock was due to myocardial infarction in 14, internal hemorrhage in three, poor-risk postoperative patients in four, and one each due to mitral stenosis with intractable heart failure, barbiturate intoxication, acute hepatic necrosis, and respiratory insufficiency due to pneumothorax in an emphysematous patient. There were no cases in which bacteremia was thought to play a significant role.

The patients were seen as early in shock as possible, usually within one hour of the onset of hypotension, and arterial blood was drawn under anaerobic conditions for pH, pCO_2 and bicarbonate determinations by the Astrup method, and for blood lactate determinations by the method of Barker and Summerson.[8] For the lactate determination the trichloroacetic acid was kept at 10° C. until the blood was added. It was then immediately centrifuged and the supernatant quick-frozen until the colorimetric determinations were done.

Eight patients were followed with repeated pH, pCO_2, bicarbonate and blood lactate determinations during the course of their shock and during the recovery phase.

RESULTS

Blood lactate in the sample taken early in shock was elevated in 24 of the 25 patients studied. This blood sample was usually taken within the first hour and in no instance later than three hours after the development of the clinical shock. Most of these patients had a metabolic acidosis.

Fig. 1A shows the relationship between rising blood lactate and decrease in serum bicarbonate and blood pH in 18 patients who had not received any sodium bicarbonate prior to the arterial punc-

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Canad. Med. Ass. J.
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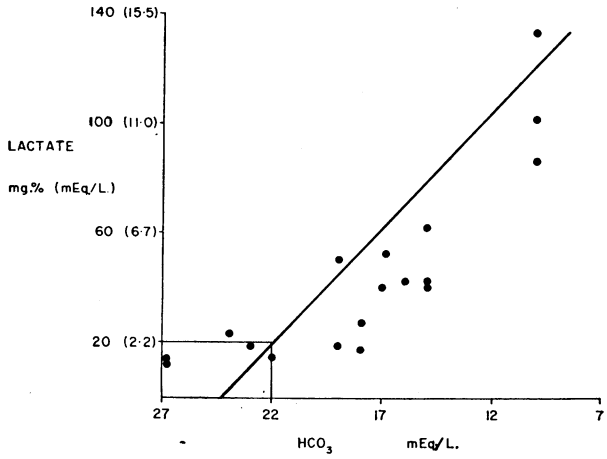

Fig. 1A.—Correlation between rising blood lactate and decreasing serum bicarbonate in 18 patients with the shock syndrome.

ture ($r = 0.83$, $p = <0.01$). The pH in Fig. 1B is corrected to a pCO_2 of 40 mm. Hg to correct for the effects of underventilation or overventilation which occurred in some of these patients. It is clear from these data that early in shock, prior to the accumulation of other acid radicals from renal failure and other sources (e.g. hyperchloremia), the acidosis is largely or entirely due to lactate.

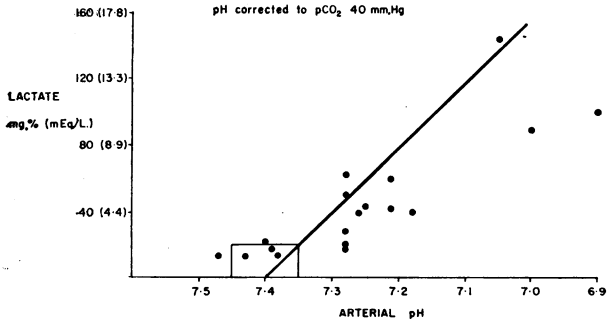

Fig. 1B.—Correlation between rising blood lactate and decreasing arterial pH in the shock syndrome.

The relationship between the initial blood lactate level and the survival was next examined in all our subjects. The survival rate was higher in those subjects whose initial blood lactate was lower (Table I).

TABLE I.—RELATIONSHIP BETWEEN CONCENTRATION OF BLOOD LACTATE EARLY IN SHOCK AND ULTIMATE PROGNOSIS

Lactate	*No. of patients*	*No. of survivors*	*% survivors*
>13.3 mEq./l. (120 mg. %)	6	0	0
8.9-13.2 mEq./l. (80-120 mg. %)....	7	1	15%
4.4-8.8 mEq./l. (40-79 mg. %).....	9	3	33%
< 4.3 mEq./l. (39 mg. %)	3	3	100%

From individual case protocols with serial examinations of blood lactate, pH, pCO_2 and bicarbonate, it became apparent that those patients with a blood lactate in excess of 8.9 mEq./l. (80 mg./100 ml.) which could not be reduced by therapy had a grave prognosis. By contrast, those patients with an initial blood lactate below 8.9 mEq./l. (80 mg./100 ml.) which rapidly dropped following commencement of therapy had a good prognosis. The above relationship held in spite of the fact that adequate amounts of sodium bicarbonate were given to all these patients to bring their pH to within, or close to, the normal range. Clinical improvement and the ability to decrease the amounts of vasopressors needed to maintain blood pressure were invariably accompanied by decreasing levels of lactate.

DISCUSSION

The occurrence of metabolic acidosis in shock is well reported in the literature, but its causes and clinical significance have not been clearly defined. These data indicate that at least in early shock the acidosis is due largely or entirely to an increase in lactate radicals. The accumulation of other acid radicals does not play an important part. It is likely that the increase in blood lactate in shock is due to tissue underperfusion with resultant increase in anaerobic metabolism, though reduced metabolism of lactate by the liver may

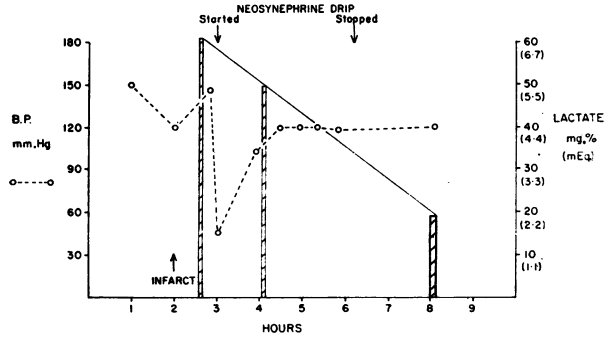

Fig. 2.—Diagram illustrating an elevated blood lactate prior to hypotension and the decrease in lactate with clinical improvement.

play a role. In either case the accumulation of lactate presumably reflects reduction in effective cardiac output and supplies a good additional index of the severity of shock. Although the numbers are small and insufficient for definite conclusions to be drawn, the apparent relationship between the blood lactate level early in shock and ultimate prognosis (Table I) supports this hypothesis. Review of individual cases shows that the rise in lactate may actually precede the appearance of hypotension. An example (Fig. 2) is the case of a 60-year-old woman who developed cardiac arrhythmia during induction of anesthesia. Her systolic blood pressure fell from 150 mm. Hg to 120 mm. Hg. The induction was stopped and she was taken to the recovery room where her blood pressure returned to 140 mm. Hg and an electrocardiogram showed evidence of a myocardial infarction. The blood lactate at this time was ele-

vated to 6.7 mEq./l. Approximately five minutes after this reading her blood pressure dropped to 45 mm. Hg. She responded well to a phenylephrine (Neosynephrine) intravenous drip and, as shown in the graph, her lactate fell off over the next several hours and she made an uneventful recovery. This response of the lactate level to clinical improvement was seen in all our patients. Those patients who did not survive maintained their blood lactate concentrations at or above the initial level in spite of clinical improvement of their blood pressure for limited periods of time.

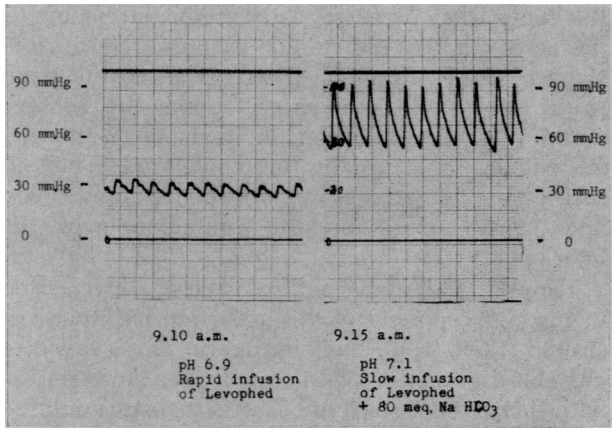

Fig. 3.—Blood pressure response before and after partial correction of metabolic acidosis with sodium bicarbonate.

Acidosis, if uncorrected, may have serious consequences. Examples of the effect of acidosis reported in the literature and observed in this series of subjects include impairment of intraventricular conduction with wide, slurred QRS complexes, refractoriness to electrical defibrillation,[9] reduced efficiency of the myocardium[10, 11] and refractoriness to pressor amines[12] (Fig. 3). Examples of each of these were encountered in this series. In each case there was a prompt response following correction of the acidosis with sodium bicarbonate.

It should be stressed that correction of the acidosis did not influence the level of blood lactate unless the circulation was improved concurrently. Thus, in one instance a pH of 6.9 was corrected over two hours to 7.4 by administration of 500 mEq. of sodium bicarbonate intravenously. During this time the lactate rose from 132 mg./100 ml. to 148 mg./100 ml. This patient did not recover. It appeared that a decrease in blood lactate could be accomplished only with improved tissue perfusion and in two instances this took place even in the presence of sustained hypotension after the administration of a sympatholytic agent, phenoxybenzamine (Dibenzyline).

SUMMARY AND CONCLUSIONS

In the early phases of shock, metabolic acidosis was common and was due largely or entirely to accumulation of lactate. This was presumably due to underperfusion of tissues and possibly to decreased metabolism of this radical by the liver.

The determination of blood lactate levels offered a valuable additional guide to the severity of shock and appeared to correlate with ultimate prognosis. High lactate levels which did not fall with treatment indicated a grave prognosis, whereas a fall in lactate followed closely on clinical improvement.

The acidosis resulting from high lactate concentration had adverse effects which could be corrected by infusion of sodium bicarbonate. This did not, however, influence the lactate level unless the peripheral circulation was improved at the same time.

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PAGES OUT OF THE PAST: FROM THE JOURNAL OF FIFTY YEARS AGO

SAVE A LIFE HERE AND THERE

The general treatment in all forms of puerperal infection is very important, and in very many cases is the only treatment called for. It should consist of providing the patient with physical and mental rest, by drugs, if necessary. An abundance of fresh air, nutritious food in sufficient quantities, and moderate free action of the bowels and other emunctories. Where it is feasible, as in hospitals, the patient should be kept on the roof under suitable arrangements, and given a sun bath for several hours a day. Where a roof is not available, a sun bath can be provided for through the employment of open windows.

The vast majority of cases will get well, treated on general and conservative principles; that it is only in about, roughly speaking, 10 per cent. of all cases that any surgical intervention comes into consideration; and that in considering surgical measures, no hard and fast lines can be laid down as to indications. Each case must be studied carefully at the bedside and closely watched as to its progress. When feasible, whatever aid is to be obtained from a bacteriological examination of the lochia and of the blood, should be sought and the result duly weighed. Then when all these conditions have been fulfilled and one feels he has the necessary skill and experience, he may be able to save a life here and there, by timely surgical intervention, otherwise it will be safer to rely upon nature's effort with the aid of palliative and supportive treatment.—H. N. Vineberg: *Canad. Med. Ass. J.*, 4: 208, 1914.