THE LACTATE LEVEL AS AN INDICATOR OF THE SEVERITY OF THE CONDITION AFTER MASSIVE BLOOD LOSS

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UDC 616.151.11-02:616-005.1]-07:616.153.472.3-074

Comparison of the clinical and experimental material suggests that the maximal increase in the blood lactate level (70-80 mg%) in the early recovery period after clinical death from blood loss does not itself indicate a possible lethal outcome. However, if a state of clinical death does not develop, the critical blood lactate level can serve as a prognostic sign. A long-lasting raised lactate level can also serve as the basis for an unfavorable prognosis.

KEY WORDS: lactacidemia; blood loss; clinical death.

Lactacidemia is regarded as an indicator of the irreversibility of tissue changes; the critical lactate level at the extremal moment is considered to be 70-80 mg % [2, 4, 5].

The lactate level was determined experimentally in terminal states and in the postresuscitation period and the results were compared with those of corresponding determinations in patients after massive blood loss.

EXPERIMENTAL METHOD

Thirteen dogs, each receiving 4-8 mg/kg pantopon and 10 mg/kg pentobarbitol, were used. Slow bleeding was carried out for 2 h (the blood loss was 50 ± 5.3 ml/kg body weight; the arterial pressure was kept at 40 mm Hg). Bleeding continued until the onset of clinical death, which continued for 1 min. During resuscitation blood with adrenalin (0.3-0.5 ml) was injected intra-arterially and artificial respiration performed. Three groups of patients with massive blood loss of different pathogenesis also were investigated (period of blood loss not less than 2 h): group 1: patients in whom the course of recovery was favorable, whose breathing was unassisted throughout the period of treatment (16 patients, blood loss of 1.5 to 3 liters);

TABLE 1. Indices of Metabolism in Dogs in Pre- and Post-resuscitation Periods (M ± m)

Index studied		Original data	After 2 h of hypotension	Postresuscitation period			
				5 min	1 h	3 h	6 h
Lactate (mg %)	A	14,6±1,3	51,2±4,6	77,9±2,3	47,6±3,1	16,1±2,2	12,3±1,7
	B	13,4±1,2	39,0±1,2	75,4±2,7	43,8±4,7	14,0±1,9	11,8±2,0
	C	16,9±2,5	65,4±3,5	81,5±4,2	52,3±4,3	18,7±3,3	12,7±3,1
Pyruvate (mg %)	A	0,94±0,14	0,90±0,12	1,70±0,16	1,23 −0,10	0,42±0,07	0,39±0,09
Lactate/pyruvate	A	15,5±5,7	66,5±15,3	57,2±10,5	44,3±18,0	38,3±7,8	31,6±5,7
Organic acids (meq/liter)	A	9,8 ±1,8	26,3±3,3	28,5±4,1	24,7±2,9	22,9±2,3	19,5±2,1

Legend. A) In all animals; B) in surviving animals; C) in dying animals.

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TABLE 2. Indices of Metabolism in Patients after Massive Blood Loss (M ± m)

Index studied	Group No.	Time after blood loss				
		0–12 h	12–24 h	2 days	3–4 days	over 5 days
Lactate (mg%)	1	26,1±6,9	16,5±4,6	8,2±0,7	9,1±0,6	10,3±0,9
	2	59,1±5,0	14,4±1,1	10,1±1,8	7,0±0,5	9,4±0,7
	3	—	30,5±1,5	26,4±7,4	21,8±6,8	—
Pyruvate (mg%)	1	0,59±0,16	0,57±0,18	0,43±0,07	0,41±0,04	0,40±0,04
	2	0,63±0,02	0,24±0,01	0,25±0,02	0,20±0,04	0,33±0,05
	3	—	1,15±0,09	0,31±0,02	0,26±0,02	—
Lactate/ pyruvate	1	34,9±8,5	20,2±1,1	23,4±5,1	26,4±3,4	30,1±4,8
	2	93,5±3,6	69,8±19,6	40,0±5,9	36,6±9,5	34,8±7,3
	3	—	27,0±5,6	87,0±24,9	96,9±26,4	—
Organic acids (meq/liter)	1	15,4±4,9	17,0±0,6	12,5±1,8	12,1±1,2	13,4±1,8
	2	22,1±0,9	—	13,0±0,9	10,2±0,7	14,8±1,6
	3	—	25,2±2,7	19,3±1,1	16,4±1,1	—

group 2: patients in a serious condition during the recovery period, requiring prolonged artifical ventilation of the lungs (three patients, blood loss of more than 3 liters); group 3: patients who died (three patients, blood loss over 3 liters, AVL starting from the second day). The arterial blood levels of lactate, pyruvate, and organic acids were determined.

EXPERIMENTAL RESULTS AND DISCUSSION

By the end of the second hour of hypotension an accumulation of lactate was observed in the dogs. The blood pyruvate concentration was unchanged, so that the lactate/pyruvate ratio rose sharply (Table 1). At the beginning of the recovery period a further increase in the concentration of incompletely oxidized metabolic products was found in the blood. The lactate level did not return to normal until the end of the third hour of the postresuscitation period, when the pyruvate concentration was already much below its initial value. The high lactate/pyruvate ratio and the high organic acid level were evidence of the hypoxic character of metabolism throughout the period of observation. The greatest differences in metabolism in the dying and surviving animals were observed at the end of the second hour of hypotension, but these differences were significant ($P < 0.05$) only for lactate. The high lactic acid level possibly not only reflects the severity of the condition, but may even aggravate it, by inhibiting oxidation in the tissues [2] and creating a basis for the development of irreversible changes in them.

The highest blood lactate level in the patients occurred within a few hours after blood loss (Table 2). Although this index of hypoxia was significantly ($P < 0.01$) higher than in the patients of group 1 and came close to the critical level in the patients of group 2, two of whom passed into a state of clinical death, they all survived. Later the differences in the lactate concentration diminished and its level gradually fell in all the surviving patients. The pyruvate concentration in the survivors was within normal limits during the first 12 h, which accounted for the high lactate/pyruvate ratio. Later the pyruvate level fell, more so in the patients on AVL ($P < 0.05$), in agreement with data in the literature [1, 3]. It was impossible to obtain blood samples during the first few hours after their admission to hospital from the patients who died. However, throughout the period of observation their lactate level was significantly ($P < 0.05$) higher than in the survivors, although it did not reach the critical level. The blood level of total organic acids rose in all patients during the first day of treatment. On the 2nd-5th day this index returned to normal in patients who responded favorably to treatment.

Comparison of the clinical and experimental material suggests that the use of the lactate level as a prognostic criterion after massive blood loss is justified only in cases when a state of clinical death does not develop. If the patient is revived from clinical death, a high blood lactate concentration in the early recovery period is not by itself grounds for predicting a lethal outcome. If the high blood lactate level is maintained for a long time, this could be grounds for an unfavorable prognosis.

LITERATURE CITED

1. F. Eldridge and J. Salzer, "The effect of respiratory alkalosis on blood lactate and pyruvate in humans," J. Clin. Invest., 44, 1043 (1965).
2. H. Laborit, "De la prophylaxie a la thérapeutique du choc dit 'irréversible'," Presse Méd., 74, 2079 (1966).
3. I. Leusen and G. Demeester, "Lactate and pyruvate in the brain of rats during hyperventilation," Arch. Int. Physiol. Biochem., 74, 25 (1966).

4. V. Vitec and R. Cowley, "Lactic acid in blood as indicator of prognosis in hypotensive shock," Clin. Res., 16, 519 (1968).
5. M. H. Weil and A. A. Afifi, "Experimental and clinical studies on lactate and pyruvate as indicator of acute circulatory failure (shock)," Circulation, 41, 989 (1970).