

USE OF LYMPHOCYTE SUCCINATE DEHYDROGENASE ACTIVITY TO PREDICT
THE COURSE OF THE POSTRESUSCITATION PERIOD

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The enzyme status of the leukocytes is used in clinical and experimental cytochemistry to predict the course of pathological processes and states [1, 3, 4, 6].

The aim of this investigation was to determine whether correlation exists between the enzyme status of lymphocytes before and after clinical death, and to what extent the cytochemical picture and characteristics of the course of the postresuscitation period can be predicted from the initial enzyme status.

EXPERIMENTAL METHOD

Experiments were carried out on 26 noninbred male rats weighing 163 ± 18 g, anesthetized by intraperitoneal injection of pentobarbital (25 mg/kg body weight). Clinical death for 4 min was caused by acute blood loss, and the animals were resuscitated by the method in [5]. Blood for preparing blood films was taken from the caudal vein before the experiment, at the end of clinical death, and 5, 15, and 90 min after resuscitation. Succinate dehydrogenase (SDH) activity of the lymphocytes was determined as the number of granules of reaction product per lymphocyte, on the basis of examination of 50 cells in each film [2]. Mean activity, coefficients of variation and asymmetry, and diversity of the cells according to enzyme activity were calculated by computer, correlations between cytochemical parameters and the duration of hypoxia, the time of restarting the heart, and the appearance of the corneal reflex, the time of resuscitation, and the length of survival after resuscitation were analyzed. Only statistically significant coefficients of correlation, exceeding the critical level for the number of animals used ($r = 0.39$) were accepted in the investigation.

TABLE 1. SDH Activity of Lymphocytes during Clinical Death and after Resuscitation ($M \pm m$, $n = 25$)

Parameters	Stage of experiment				
	before clinical death	clinical death	postresuscitation period, min		
			5	15	90
Typical activity	$16,1 \pm 0,21$	$9,9 \pm 0,29^*$	$15,1 \pm 0,86$	$10,9 \pm 0,67^*$	$12,7 \pm 0,47^*$
Variation of lymphocytes according to enzyme activity	$32,4 \pm 1,84$	$59,2 \pm 2,21^*$	$43,6 \pm 3,00^*$	$57,7 \pm 3,36^*$	$48,6 \pm 2,82^*$
Equilibrium state of number of cells with low and high activity	$0,202 \pm 0,065$	$0,245 \pm 0,068$	$0,304 \pm 0,085$	$0,459 \pm 0,127$	$0,540 \pm 0,144^*$
Sufficiency of cells with typical enzyme activity	$-0,085 \pm 0,141$	$-0,205 \pm 0,131$	$+0,304 \pm 0,174$	$+0,609 \pm 0,218^*$	$+1,487 \pm <0,237^*$
Diversity of cells according to enzyme activity	$0,693 \pm 0,010$	$0,694 \pm 0,006$	$0,699 \pm 0,010$	$0,683 \pm 0,009$	$0,699 \pm <0,009$

* $p < 0.05$ for comparison with initial level.

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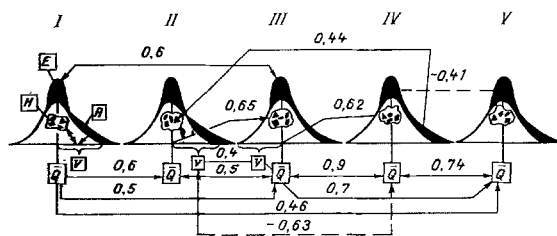


Fig. 1. Correlations of peripheral blood lymphocyte enzyme status during resuscitation. Parameters of distribution of lymphocytes by SDH activity: \bar{Q}) mean activity, V, A, and E) coefficients of variation, asymmetry, and excess respectively, reflecting heterogeneity and balance of pools with high and low enzyme activity, sufficiency of cells with typical enzyme activity; H) entropy of information (measure of diversity). Continuous and broken lines denote significant positive and negative correlations respectively ($P < 0.05$). I) Before death; II) clinical death; III) 5 min of resuscitation, IV) 15 min of resuscitation; V) length of survival.

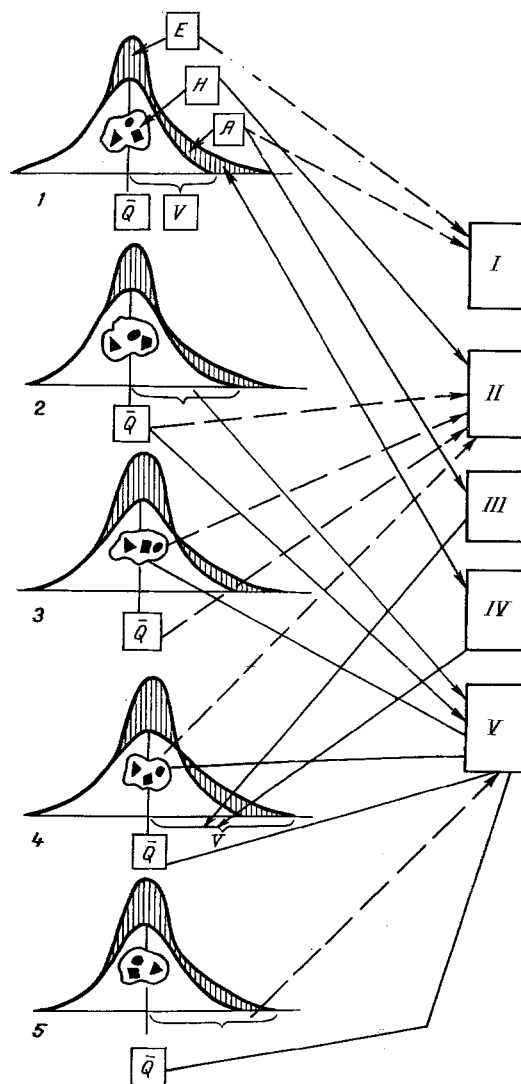


Fig. 2. Prognostic correlations of enzyme status of blood lymphocytes with signs of resuscitation and with duration of survival. 1) Before death, 2) clinical death, 3-5) 5, 15, and 90 min respectively after clinical death. Remainder of legend the same as to Fig. 1.

EXPERIMENTAL RESULTS

Data showing changes in SDH activity over a period of time are given in Table 1.

Analysis of correlation between the enzyme status of the lymphocytes in time shows that each stage was coordinated with the previous stage and largely determined the subsequent stage also (Fig. 1). Homonymous parameters did not always correlate (most frequently cross correlation was observed), but because various characteristics of the cell population were interconnected it was possible to determine the shift of these characteristics as a whole. Thus the change in SDH activity and its apparent absence of change were largely determined by the status in the previous period, and most important of all, the state just before death. For example, the increased reserve of cells with typical enzyme activity at the moment of clinical death and throughout the postresuscitation period.

The significance of the initial enzyme status also characterizes prognostic correlations: with an excess of typical cells and with the existence of individually enzymatically generative cells, the body becomes vulnerable to hypoxia (death arises more rapidly and resuscitation is more difficult, the times taken for restoration of several indices are lengthened). Characteristically, great diversity of the lymphocytes with respect to SDH activity was determined in those cases when the heart was more difficult to restart, but the duration of survival in the postresuscitation period was longer (Fig. 2). The higher the SDH activity of the lymphocytes at the moment of clinical death, the longer the animal survived after resuscitation, and the greater the decrease in SDH activity, the more difficult it was to restart the heart.

As was stated above, the diversity of the cells with respect to enzyme activity did not change throughout the period of resuscitation, but the degree of this diversity correlated with the individual course of resuscitation and with the length of survival. This indicates that it is not sufficient simply to record changes in mean values, because these changes may be found not to correlate directly with the concrete signs of resuscitation.

The results are thus evidence of the value of cytochemical analysis of the blood from the prognostic point of view, but retrognosis — the recreation of past events — is no less important. No such reconstruction is required under experimental conditions, but clinically the physician may be brought face to face with a process that has already developed, and he will then have the important task of assessing the state which preceded the pathological process, and which had a significant influence on the course and outcome of the disease. The experimental data described in this paper demonstrate that such a retrospective analysis is possible in principle.

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