CHOLINESTERASE ACTIVITY AND ACETYLCHOLINE CONCENTRATION IN THE HEART IN EXPERIMENTAL ADRENALIN MYOCARDIAL DEGENERATION

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UDC 616.127-003.8-092.9-008.948

In experimental adrenalin myocardial degeneration, changes in the concentration of acetylcholine and in the intensity of its enzymic hydrolysis in the heart depend on the dose of adrenalin injected. If the dose of adrenalin is small, the cholinesterase activity falls progressively, and the acetylcholine concentration rises in the recovery period. With an increase in the dose of adrenalin, a temporary increase in cholinesterase activity and decrease in the acetylcholine concentration are observed.

The object of the present investigation was to determine the concentration of acetylcholine and the intensity of its enzymic hydrolysis in the heart of normal rats and rats with myocardial degeneration induced by adrenalin.

The presence of myocardial degeneration was judged from changes in the ECG. The rats were decapitated. The acetylcholine concentration in the heart was determined by Rothschuk's method [7] and expressed in μg/g tissue. The total cholinesterase (without differentiation into specific and nonspecific) activity in the heart was determined by the method of Fleischer and Pope in the modification of Pushkina and Klimkina [2] and expressed as the quantity of acetylcholine hydrolyzed in μmoles/h/g tissue.

The results given in Table 1 show that in degeneration induced by injection of adrenalin in a dose of 0.1 mg/100 g body weight a significant increase in the acetylcholine concentration was found only toward the end of the recovery period (on the 20th day), when the ECG of the animals was almost completely back to normal. With an increase in the dose of adrenalin to 0.3 mg/100 g, a decrease in the acetylcholine con-

TABLE 1. Changes in Acetylcholine Concentration and Cholinesterase Activity in the Heart in Adrenalin Myocardial Degeneration

Exptl. conditions	Acetylcholine			Cholinesterase activity		
	number of animals	$M \pm m$	P	number of animals	$M \pm m$	P
Control	10	1,30±0,13	—	20	78,15±2,10	—
Adrenalin 0,1 mg/100 g						
2nd day	10	1.41±0,15	>0,05	10	63,13±4,70	<0,01
5th »	10	1,15±0,08	>0,05	20	65,09±3,59	<0,002
20th »	10	1,86±0,09	<0,01	10	50,62±4,88	<0,001
Adrenalin 0,3 mg/100 g						
2nd day	10	0,96±0,08	<0,05	10	61,03±1,63	<0,001
5th »	10	0,99±0,14	>0,05	10	90,62±1,63	<0,001
20th »	10	1,10±0,12	>0,05	10	69,96±2,39	<0,01

Department of Pathological Physiology, Ternopol' Medical Institute. (Presented by Academician V. V. Parin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 72, No. 7, pp. 22-23, July, 1971. Original article submitted September 29, 1970.

centration was observed after 24 h, but later its concentration rose to almost the control level. In these animals on the 20th day the changes in the ECG were much more marked than in the rats of the preceding series.

The cholinesterase activity in the heart of the animals receiving adrenalin in a dose of 0.1 mg/100 g fell progressively. In rats receiving 0.3 mg/100 g adrenalin, a decrease in the cholinesterase activity was observed on the 2nd day, an increase on the 5th day, and a further decrease in the activity of the enzyme to below the control level later (Table 1).

This increase observed in the acetylcholine concentration in the heart during the period of recovery from adrenalin-induced myocardial degeneration evidently depends to some degree on a decrease in the activity of its enzymic hydrolysis. On the basis of existing views regarding the opposite effects of adrenalin and acetylcholine on some aspects of myocardial metabolism [1-3, 6, 8], the increase in acetylcholine concentration can be regarded as a physiological response with the object of normalizing metabolic processes in the myocardium and its functions. With an increase in the dose of adrenalin, this response is inhibited to such an extent that initially the acetylcholine concentration in the heart is lowered.

LITERATURE CITED

1. V. V. Parin and F. Z. Meerson, Zh. Évolyuts. Biokhim. i Fiziol., 5, No. 2, 158 (1969).
2. N. N. Pushkina, Biochemical Methods of Investigation [in Russian], Moscow (1963), p. 202.
3. K. Gollwitzer-Meyer, K. Kramer, and E. Kruger, Pflüg. Arch. Ges. Physiol., 237, 639 (1936).
4. J. Gremels, Arch. Exp. Path. Pharmak., 169, 689 (1933).
5. W. Raab, Rev. Canad. Biol., 22, 217 (1963).
6. W. Raab, in: Current Problems in Cardiovascular Pathology [Russian translation], Moscow (1967), p. 137.
7. K. E. Rothschuk, Pflüg. Arch. Ges. Physiol., 258, 406 (1954).
8. E. Sonnenblick, Jr., I. Ross, and E. Broumwold, Am. J. Cardiol., 22, 328 (1968).