NORADRENALIN AND ADRENALIN CONTENT IN THE HUMAN MYOCARDIUM AFTER ACCIDENTAL AND SUDDEN DEATH

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To investigate the mechanisms of sudden death the authors determined the noradrenalin and adrenalin content in the myocardium of various parts of the heart by a fluorometric method; parallel determinations were made in the myocardium of persons dying accidentally. A heterotopic distribution of noradrenalin in the various parts of the heart after sudden death was found compared with its distribution after accidental death. Differences in the content of adrenalin and noradrenalin in the various parts of the heart of the two groups indicate that death was due to different mechanisms. KEY WORDS: sudden death, trauma, catecholamines, myocardium.

The mechanisms lying at the basis of sudden death are complex, varied and, probably, highly individualized and depend on many conditions, genetic factors, previous diseases, psychoemotional stress, and disturbances of the activity of enzymic and hormonal processes.

Among metabolic disorders which have been suggested, those affecting catecholamines (dopamine, noradrenalin, and adrenalin), play an important role [1-3, 6, 8-11, 13, 15].

This paper describes the results of determination of the noradrenalin and adrenalin content in the myocardium of two groups of persons, dying suddenly and as a result of trauma.

EXPERIMENTAL METHOD

Tests were carried out on 23 human hearts, 15 of them from persons dying suddenly. Autopsy showed that during life these persons suffered from chronic and acute ischemic heart disease, myocardial infarction, and acute cardiovascular failure. The time between death and the investigation varied between 2 and 24 h. Eight persons died from trauma between the ages of 3 and 52 years; the time between death and the investigation varied from 12 to 24 h. The adrenalin and noradrenalin content also was determined in the myocardium of 8 bulls killed by electric shock. The time between slaughter and the investigation was 30 min.

Catecholamines were determined by a fluorometric method [7]. Samples for investigation were taken from the left atrium, the anterior wall of the left ventricle (the middle third), the apex of the left ventricle, the ventricular septum (upper third), the right atrium (at the junction between the superior vena cava and the lateral border of the right atrium), and the right ventricle (middle third). The results were subjected to statistical analysis.

EXPERIMENTAL RESULTS AND DISCUSSION

In nearly all cases the adrenalin level was considerably lower than the noradrenalin level in the hearts of both groups of subjects (Table 1). A lower content of adrenalin in the myocardium of the atria and ventricles was observed in the traumatic group than in the sudden death group.

Considerable differences both between different parts of the heart and between the two groups were found in the noradrenalin content. Comparison of the results shows a heterotropic distribution of noradrenalin between the left and right portions of the heart in both groups (Table 1).

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TABLE 1. Adrenalin and Noradrenalin Content (in μg/tissue) in Different Parts of Human Heart after Sudden and Accidental Death (M ± m)

	Left atrium		Left ventricle		Apex of left ventricle		Ventricular septum		Right atrium		Right ventricle	
	A	NA	A	NA	A	NA	A	NA	A	NA	A	NA
Accidental death	0,02±0,004	0,075±0,023	0,022±0,008	0,14±0,047	0,025±0,016	0,163±0,05	0,028±0,016	0,254	0,005±0,0015	0	0,009±0,008	0,16±0,08
Sudden death	0,036±0,027	0,2±0,06	0,032±0,008	0,2±0,05	0,021±0,006	0,132±0,04	0,026±0,005	0,25±0,053	0,013±0,005	0,18±0,06	0,021±0,006	0,013±0,03
P	0,5	0,001	0,3	0,4		0,2			0,6	0,01	0,2	0,1
P_1						0,02		0,001		0,02		
						(3,6)		(4,6)		(5,6)		

Legend. P_1) statistically significant changes in NA level in different parts of the heart after sudden death. Here and in Table 2: A) adrenalin; NA) noradrenalin.

TABLE 2. Concentrations of Noradrenalin and Adrenalin (in μg/g) in Conducting System of Heart

	Sinoatrial node		Atrioventricular node		Left branch of bundle of His	
	HA	A	HA	A	HA	A
Accidental death	3,2	2,1	2,9	0,027	0,17	0,058
Sudden death	5,7	1,04	0,32	0,15	0,32	0,105

TABLE 3. Noradrenalin Concentration (in μg/g) in Human Myocardium

Pathology	After sudden death	Noradrenalin concentration	Citation
Mitral incompentence	Auricle of left atrium	1.15	[5]
Fallot's tetralogy	Right ventricle (children aged 8-13 years)	0.67-0.85	[2]
Functional failure	Myocardium of ventricles	0.33	[14]
Mitral stenosis	Auricle of left atrium:		
	before commissurotomy	0.52	[4, 12]
	after commissurotomy	0.16	
After death from trauma	Myocardium of right ventricle	0.16	
After sudden death	The same	0.013	

After accidental death, for example, the noradrenalin level in the myocardium of the ventricular septum was 0.254 μg/g, whereas in the myocardium of the anterior wall of the left ventricle its concentration was lower by almost half; in the myocardium of the right atrium noradrenalin was absent, whereas in the myocardium of the left atrium its concentration was 0.075 μg/g. After sudden death the noradrenalin concentration in the myocardium of the right ventricle was one-fifteenth of that in the myocardium of the anterior wall of the left ventricle; the same degree of heterotopia with respect to noradrenalin was found between the myocardium of the ventricular septum and the right ventricle. Consequently, after sudden death heterotopia for the noradrenalin concentration between different parts of the heart was more marked than after accidental death.

The adrenalin level in the myocardium of the left and right halves of the bovine heart was between 0.03 and 0.04 μg/g, whereas in the myocardium of the ventricular septum it was 0.07 μg/g. The lowest noradrenalin level was found in the myocardium of the right ventricle, namely 0.05 μg/g. It varied between 0.09 and 0.1 μg/g in the myocardium of the left ventricle, the apex, and the ventricular septum, and in the myocardium of the left atrium the noradrenalin concentration was 0.18 μg/g.

The results of the investigation thus demonstrate differences between the noradrenalin and adrenalin concentrations in the human myocardium after sudden death and in the human and bovine myocardium after death from trauma.

Differences in the levels of noradrenalin and adrenalin were found in all parts of the heart, reflecting heterogeneity of conduction, excitation, and mechanical activity of the myocardium and, probably, of its conducting system. These differences in the catecholamine concentrations explain the frequently observed fact of the appearance of electrical activity and mechanical contractions in some parts of the heart during experimental attempts to revive the activity of human hearts after death from various diseases [1].

In the few determinations of catecholamines that were made in the sinoatrial and ventricular nodes and in the left branch of the bundle of His a high concentration of adrenalin and noradrenalin was found after both accidental and sudden death. As Table 2 shows, the catecholamine level fell considerably from the sinoatrial node toward the periphery, and this may perhaps be an important factor determining the velocity of conduction of atrioventricular impulses.

These facts suggest that the mechanisms of traumatic and sudden death are different.

The noradrenalin concentration in the myocardium of the ventricular septum was the same after both acci-

dental and sudden death, and was higher (0.254 and 0.25 μg/g respectively) than elsewhere in the heart. This may indicate the onset of fibrillation in the myocardium of the ventricles both during sudden death and during traumatic shock, for high catecholamine concentrations lead to the appearance of multiple pacemakers and to their heterogeneity, and increase the conduction velocity in the atria, the atrioventricular node, and the ventricles, thereby facilitating the development of fibrillation.

It will be clear from Table 3 that the noradrenalin concentration in the myocardium in the case of valvular defects was 2 to 3 times higher than in the case of functional failure; after operative stress and general fatal trauma the noradrenalin level was reduced even lower, and the lowest noradrenalin concentration in the myocardium was observed after sudden death.

The following conclusions can be drawn from these results: 1) in sudden death the noradrenalin concentration falls sharply in different parts of the myocardium by comparison with its level in the myocardium of persons dying from diseases of the heart valves, cardiac failure, operative stress, and general trauma; 2) in sudden death there is a marked heterotopic distribution of noradrenalin in the myocardium of different parts of the heart. A high level of heterotopia is also evidently found in the noradrenalin concentration in the sinoatrial and atrialventricular nodes. The possibility cannot be ruled out that dissociation between the noradrenalin concentrations in different parts of the myocardium and the nodes of the conducting system disturbs the regulation of their synchronous activity, blocks cellular metabolism, and so constitutes one component of the mechanism of sudden death.

LITERATURE CITED

1. S. V. Andreev, Restoration of the Activity of the Human Heart after Death [in Russian], Moscow (1955).
2. S. V. Andreev and I. D. Kobkova, The Role of Catecholamines in Health and Disease [in Russian], Moscow (1970).
3. Yu. I. Detsik, I. I. Birka, M. T. Monastryrskaya, et al., Kardiologiya, No. 7, 130 (1976).
4. I. S. Zavodskaya, O. N. Zabrodin, N. I. Zaskal'sko, et al., Kardiologiya, No. 2, 110 (1974).
5. B. N. Manukhin and A. V. Sumarokov, Pat. Fiziol., No. 1, 49 (1964).
6. É. Sh. Matlina and V. V. Men'shikov, Clinical Biochemistry of Catecholamines [in Russian], Moscow (1967).
7. V. V. Men'shikov, Methods in Clinical Biochemistry of Hormones and Mediators [in Russian], Moscow (1969).
8. M. E. Raiskina, in: Proceedings of the 4th All-Union Congress of Pathological Anatomists [in Russian], Moscow (1967), pp. 172-175.
9. H. Selye, Essays on the Adaptation Syndrome [Russian translation], Moscow (1960).
10. G. M. Solov'ev and E. Sh. Matlina, Kardiologiya, No. 1, 140 (1973).
11. E. I. Chazov, L. F. Nikolaeva, and M. Ya. Ruda, Kardiologiya, No. 2, 5 (1977).
12. C. A. Chidsey, E. Braunwald, and A. G. Morrow, Am. J. Med., 39, 442 (1965).
13. A. S. Leon, Am. J. Med. Sci., 262, 9 (1971).
14. W. Raab, Am. J. Cardiol., 5, 579 (1960).
15. A. Richtarik, H. Hift, and E. Valdiovia, Arch. Int. Pharmacodyn. Ther., 159, 44 (1966).