and effects of stimulation of brain structures in parkinsonism [2]. Phenomena of this type have been described in connection with experimental multifocal epilepsy [3] and the behavior of secondary mirror foci [5, 6].

LITERATURE CITED

1. M. N. Aliev, in: Theoretical Basis of Pathological States [in Russian], Leningrad (1980), pp. 206-209.
2. D. K. Kambarova, V. A. Ilyukhina, Yu. K. Matveev, et al., in: The Treatment of Parkinsonism. The Present State of the Problem [in Russian], L. S. Petelin, ed., Moscow (1977), pp. 15-27.
3. G. N. Kryzhanovskii, Determinant Structures in the Pathology of the Nervous System [in Russian], Moscow (1980).
4. G. N. Kryzhanovskii and M. N. Aliev, Byull. Éksp. Biol. Med., No. 4, 397 (1976).
5. V. M. Okudzhava, The Basic Neurophysiological Mechanisms of Epileptic Activity [in Russian], Tbilisi (1969).
6. W. Penfield and H. Jasper, Epilepsy and the Functional Anatomy of the Human Brain, Little, Brown, Boston (1954).
7. F. E. Bloom, E. Costa, and G. C. Salmoiraghi, J. Pharmacol. Exp. Ther., 150, 244 (1965).
8. J. D. Connor, J. Physiol. (London), 208, 691 (1970).
9. E. Fifkova and J. Marsala, in: Electrophysiological Methods in Biological Research, Prague (1967), pp. 653-695.
10. J. A. Gonsales-Vegas, Brain Res., 80, 219 (1974).
11. C. Ohye, R. Bouchard, R. Boucher, et al., J. Pharmacol., Exp. Ther., 175, 700 (1970).
12. W. Schultz and U. Ungerstedt, Exp. Brain Res., 33, 159 (1978).
13. G. R. Siggins, B. J. Hoffer, and F. E. Bloom, in: The Basal Ganglia, M. D. Yahr, ed., New York (1976), pp. 227-248.
14. R. Spehlmann, Brain, 98, 219 (1975).
15. G. Steg, in: Third Symposium on Parkinson's Disease (F. J. Gillingham and I. M. L. Donaldson, eds.), Edinburgh (1969), pp. 26-29.
16. U. Ungerstedt, T. Lyunberg, B. Hoffer, et al., in: Advances in Neurology, Vol. 9, (D. B. Calne, T. N. Chase, and A. Barbeau, eds.), New York (1975), pp. 57-65.

FUNCTIONAL ACTIVITY OF PLATELETS IN THE EARLY PERIOD AFTER ARTIFICIAL HEART IMPLANTATION

I. A. Burykina, T. N. Kovaleva, S. I. Ignatenko, N. N. Mertsalova, and A. K. Chepurov

UDC 616.12-008.1-78-089.843]-07:616.155.2

KEY WORDS: platelets; adhesion; aggregation; fibrinogen; artificial heart.

Platelets play an active part in hemostasis [5, 6, 8, 9, 12] and, because of their specific property of adhesion, they are the first blood elements to react to changes arising in the blood vessels [8, 11, 12].

Since the role of platelets and their adhesion-aggregation properties after implantation of an artificial heart have not been studied, it was decided to remedy this deficiency.

EXPERIMENTAL METHODS

Experiments were carried out on 10 calves weighing 90-100 kg into which artificial hearts of Soviet manufacture were implanted. To determine the state of the platelets methods of ad-

Laboratory of Physiology, Research Institute of Transplantology and Artificial Organs, Ministry of Health of the USSR, Moscow.

(Presented by Academician of the Academy of Medical Sciences of the USSR N. N. Malinovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 92, No. 12, pp. 659-661, December, 1981. Original article submitted June 20, 1981.

TABLE 1. Changes in Adhesion, Aggregation, and Number of Platelets and in Fibrinogen Concentration in Early Stages of Work of an Artificial Heart

Parameter	Statistical index	Initial data	Anesthesia	Time of work of artificial circulation			Time of work of artificial heart, h				
				5 min	1 h	2 1/2 h	1	2—5	6—10	11—24	25—35
Adhesion, %	$M \pm m$	41,0±1,9	47,0±1,9	45,0±5,9	35,0±2,1	25,5±5,3	25,0±3,2	42,0±6,9	—	49,0±7,1	57,0±6,2
	n	19	19	12	17	13	16	10		9	7
	P		<0,02	>0,1	>0,1	<0,01	<0,01	>0,1		>0,1	<0,02
Aggregation, % optical density	$M \pm m$	51,0±4,3	57,0±4,8	19,0±4,8	18,0±6,2	17,0±1,4	18,0±3,4	31,0±1,7	44,0±5,3	58,0±6,2	69,0±5,1
	n	17	18	12	16	11	12	9	7	8	8
	P		>0,1	<0,01	<0,001	<0,001	<0,001	<0,001	>0,1	>0,1	<0,02
Number of platelets, $\cdot 10^9$/liter	$M \pm m$	364,0±31,0	416,0±33,0	232,0±30,0	153,0±27,0	123,0±13,0	119,0±7,0	160,0±8,6	185,0±21,2	200,0±32,0	380,0±34,0
	n	21	25	20	18	14	10	12	8	8	9
	P		>0,1	=0,1	<0,001	<0,001	<0,001	<0,001	<0,001	<0,01	<0,01
Fibrinogen, g/liter	$M \pm m$	4,59±0,16	4,51±0,17	3,86±0,33	3,63±0,57	3,03±0,26	3,03±0,13	3,09±0,13	2,70±0,25	3,20±0,37	3,68±0,32
	n	25	26	24	20	14	15	12	8	12	12
	P		>0,1	>0,1	>0,1	<0,001	<0,001	<0,001	<0,001	>0,1	>0,1

hesion [2] and aggregation (a photometric method with continuous recording on the KSP-4 potentiometer) [4] were used. The aggregating agent was a solution of ADP. Platelets were counted in the phase-contrast microscope. The fibrinogen concentration was determined at the same time [1].

EXPERIMENTAL RESULTS

While the animals were under anesthesia, by the usual technique, an increase was observed in the adhesive activity of the platelets compared with initially — up to 47 ± 1.9% ($P < 0.02$); the increase in the aggregating activity of the platelets under these circumstances was not significant. The platelet count reached $(416 \pm 33.0) \cdot 10^9$/liter compared with $(369 \pm 31.0) \cdot 10^9$/liter and the fibronogen concentration was not significantly changed (Table 1).

The artificial circulation, which was in operation for 1.5-2.5 h, caused a significant decrease in the platelet count accompanied by a decrease in their functional activity. The aggregating activity of the cells during the first 5 min of work of the artificial circulation apparatus (ACA) fell to 19 ± 4.8% optical density ($P < 0.001$) and it remained at this level until the end of the artificial circulation. Adhesion of the platelets decreased gradually, and toward the end of the work of the ACA it did not exceed 25.5 ± 5.3% ($P < 0.01$). The changes noted above were accompanied by a fall in the fibrinogen concentration to 3.03 ± 0.26 g/liter ($P < 0.001$) and in the number of platelets to $(123 \pm 13.6) \cdot 10^9$/liter ($P < 0.001$).

Working of the artificial heart for 1 h caused no changes in the quantitative or functional indices of the platelets and the fibrinogen level compared with data obtained at the end of the artificial circulation. The next 2-5 h of work of the artificial heart led to some increase in aggregation (to 31 ± 1.7% optical density) and adhesion (to 42 ± 6.9%), but even after the changes observed they remained below the initial values. The fibrinogen concentration and platelet count increased but not significantly.

After working of the artificial heart for 6-10 h aggregation of the platelets continued to rise to 44 ± 5.3%, the platelet count at this time was $(185 \pm 21.2) \cdot 10^9$/liter. The fibrinogen concentration fell to 2.70 ± 0.25 g/liter. After working of the artificial heart for 11-24 h the adhesive and aggregating activity of the platelets continued to rise, the platelet count was increased to $(200 \pm 32.0) \cdot 10^9$/liter, and the fibrinogen concentration reached 3.20 ± 0.37 g/liter. Compared with the initial data, however, these changes were not significant ($P > 0.1$).

Observations made 25-40 h after the beginning of working of the artificial heart showed a further increase in the adhesive and aggregating properties of the platelets to 57 ± 6.2 and 69 ± 5.1% optical density respectively ($P < 0.02$). The fibrinogen concentration regained its initial values and the platelet count rose to $(380 \pm 34) \cdot 10^9$/liter ($P < 0.01$).

Changes in the number and functional activity of the platelets and the decrease in the fibrinogen concentration observed during the artificial circulation were evidently due to the special features of hemostasis during operation of the ACA [3, 7]. The traumatizing effect of ACA may also have led to a decrease in functional activity of the platelets [7, 10].

The increase in the adhesive and aggregating properties of the platelets which began after the second hour of working of the artificial heart continued until the observations ended. Platelet aggregation was observed to depend directly on the fibrinogen concentration.

The increase in functional activity of the platelets in the early stages after implantation of the artificial heart thus led to a disturbance of the suspension stability of the blood and created the risk of development of thrombosis in the later stages of the experiment.

LITERATURE CITED

1. R. A. Rutberg, Lab. Delo, No. 1, 6 (1961).
2. V. A. Shestakov, Lab. Delo, No. 8, 501 (1974).
3. D. Birnbaum, H. Heilbach, E. Büchere, et al., Thoraxchirurgie, 25, 438 (1977).
4. J. V. B. Born, J. Physiol. (London), 162, 67 (1962).
5. J. R. Caen, Nouv. Rev. Franc. Hématol., 21, 81 (1979).
6. J. M. Gerrard, J. G. White, A. H. R. Rao, et al., Br. J. Haemat., 29, 657 (1975).
7. V. Hennessy, R. Hochs, S. Niewiarowski, et al., Am. J. Physiol., 232, 622 (1977).

8. J. F. Mustard, M. Packham, et al., Blood, 52, 453 (1978).
9. J. R. O'Brien, S. Jamieson, and M. Etherington, Thrombos. Diathes. Haemorrh. (Stuttgart), 31, 279 (1974).
10. V. Tamari, L. Aledort, E. Puszkin, et al., Ann. Thorac. Surg., 19, 639 (1975).
11. P. H. Walsh, Blood, 43, 597 (1974).
12. H. J. Weiss, A. L. Willis, D. Kujn, et al., Br. J. Haematol., 32, 257 (1976).

CORRELATION BETWEEN BLOOD FREE FATTY ACID CONCENTRATION AND PLATELET ACCUMULATION IN THE MYOCARDIAL CIRCULATION AFTER INJECTION OF ADRENALIN

I. P. Gerelyuk

UDC 612.123:]612.111.7:612.172.1].014.46:615.357:577.175.522

KEY WORDS: myocardium; adrenalin; free fatty acids; lipolysis; platelets.

During prolonged emotional stress disturbance of the microcirculation may play an important role in damage to the heart [5]. The writer showed previously that besides activation of the sympathico-adrenal system and an increase in the blood level of free fatty acids (FFA) the number of platelets in the microcirculation of the myocardium is increased in animals with emotional-pain stress [1]. In stress the blood adrenalin concentration is increased by more than 8 times [9].

This paper gives data on disturbance of microcirculatory-platelet hemostasis in the heart muscle following injection of adrenalin into intact animals and when its lipolytic effect is modified with the aid of nicotinic acid and heparin. Nicotinic acid is known to depress basal and catecholamine-induced lipolysis and to cause a decrease in the blood FFA concentration [8]. Heparin liberates enzymes hydrolyzing lipids from the tissues into the blood stream and, as a result, the blood FFA level rises considerably [11].

EXPERIMENTAL METHODS

Experiments were carried out on 25 rabbits. To isolate platelets blood was taken from the jugular vein. After isolation of the platelets by the usual method [6] plasma was added to the platelet residue and it was incubated with sodium ^{51}Cr-chromate (specific activity $9.2 \cdot 10^5$ Bq/ml). Labeled platelets were suspended in plasma and injected into animals. The animals then received an intravenous injection of adrenalin in a dose of 1.2 μg/kg/min over a period of 30 min. Heparin (500 i.u./kg) and nicotinic acid (5 mg/kg) were injected intravenously 10 min before adrenalin infusion. To determine the blood content in the myocardium, ^{99m}Tc-albumin was injected into the animals immediately after the end of adrenalin infusion. The animals were killed 5 min after injection of the nuclide. The blood content (V_T) in the heart muscle was calculated by the equation $V_T = (V_B \cdot C_T)/C_B$, where V_T stands for the total blood volume; C_B and C_T the radioactivity of blood and tissue respectively (relatively to ^{99m}Tc). The number of platelets in the blood was counted by a phase-contest method and the radioactivity of samples of blood and heart muscle tissue was measured, after which the numner of platelets in the myocardial circulation (P_T) was calculated by the equation: $P_T = (^{51}Cr_T/^{51}Cr_B) \cdot P_B$, where $^{51}Cr_T$ is the radioactivity of the myocardial tissue, $^{51}Cr_B$ the radioactivity of the blood; P_B the number of platelets in the blood. Accumulation of platelets (A_P) in the myocardial circulation was judged from the difference between the number of platelets actually found (P_T) and the number which ought to have been found in the myocardial circulation to correspond to the quantity of blood contained in it (V_T): $A_T = P_T - V_T \cdot P_B$, where P_T is the number of platelets in the myocardial circulation, V_T the blood volume in the

Ivano-Frankovsk Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR A. M. Chernukh.) Translated from Byulleten' Eksperimental'noi Biologii i Meditsiny, Vol. 92, No. 12, pp. 661-663, December, 1981. Original article submitted June 16, 1981.

 0007-4888/81/9212-1626$07.50