CHANGES IN THE MINERAL METABOLISM AFTER PRIMARY AND REPEATED BONE TRAUMA

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UDC 616.71-001-008.92

The results of the study of reparative regeneration after repeated trauma have shown that the stimulation of regeneration after preliminary traumatic injury bears the character of a regular biological process [1-4, 6-8]. However, the mechanism of this phenomenon is not clear, because no objective quantitative data concerning metabolism in the conditions of primary and repeated posttraumatic regeneration are available.

The object of this investigation was to study the changes in mineral metabolism after primary and repeated, strictly graded bone trauma accompanied by a reliable biological control.

EXPERIMENTAL METHOD

Experiments were carried out on 47 female Wistar rats weighing 110-130 g. Trauma was inflicted to the middle third of the right femur of the group of animals in the form of two cuts with a No. 3 drill as far as the medullary cavity, and 38 days later the identical trauma was repeated. The wound was sutured after primary and repeated injury.

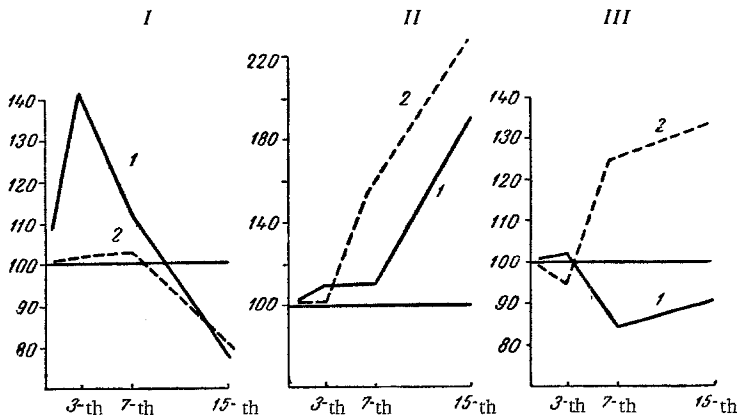
For comparison, an identical injury was inflicted on the femur of another group of rats. As controls animals undergoing single or double mock operation was used (incision of the skin and separation of the muscles followed by suture).

On the 3rd, 7th, and 15th days of the experiment the rats received an injection of $Ca^{45}Cl_2$ solution into the left femoral vein in a dose of 0.1 mg calcium/100 g body weight, and 30 min later (measured by a seconds counter) they were decapitated. The concentration of potassium and sodium in the blood serum was determined by flame photometry. The total and radioactive calcium of the blood serum and the calcified tissues was estimated. The specific activity of the serum and bone tissue were expressed in pulses /min/mg calcium in relation to 10,000 pulses/min radioactive isotope injected per gram body weight.

Concentration of Sodium, Potassium and Calcium Ions (in mg %) in Blood Serum after Primary and Repeated Bone Trauma (M ± m)

Day of expt.	Group	Sodium	Potassium	Calcium
3-th	Control	320±10	25,4±0,8	10,81±1,05
	Primary trauma	300±5	26,2±1,2	7,28±0,50
		P=0,2	P=0,5	P<0,02
	Control	346±15	22,5±0,5	9,50±0,54
	Repeated trauma	254±4	34,2±1,6	9,90±0,38
		P=0,01	P=0,01	P=0,5
7-th	Control	315±13	33,6±1,6	11,84±0,44
	Primary trauma	353±8	30,6±1,0	9,31±0,27
		P=0,05	P=0,2	P=0,002
	Control	310±20	31,8±4,9	9,86±0,83
	Repeated trauma	206±23	38,3±4,0	9,62±0,18
		P<0,02	P=0,4	P=0,8
15-th	Control	334±5	28,0±3,0	8,30±0,57
	Primary trauma	320±8	26,1±1,4	10,12±0,26
		P=0,2	P=0,6	P<0,02
	Control	334±7	29,0±0,8	8,36±0,43
	Repeated trauma	332±6	31,2±0,8	8,56±0,42
		P=0,8	P=0,1	P=0,7

Department of Pathological Physiology, Moscow Medical Stomatological Institute (Presented by Active Member of the Academy of Medical Sciences of the USSR N. A. Fedorov). Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 63, No. 2, pp. 54-56, Feburary, 1967. Original article submitted May 12, 1965.



Metabolism of Ca^{45} during primary and repeated posttraumatic regeneration. I) Blood; II) injured bone; III) intact bone; along the axis of abscissas) days of experiments; along the axis of ordinates) specific activity (in %); 1) primary trauma; 2) repeated trauma.

EXPERIMENTAL RESULTS

The results given in the table show that the concentration of sodium and potassium in the blood serum was essentially unchanged after primary bone trauma: the difference between the experimental and control values was not statistically significant. After repeated trauma, on the other hand, the concentration of sodium in the blood fell on the third day (64.9% of the control level, $P < 0.01$) and on the 7th day (66.7%, $P < 0.02$). On the 15th day the sodium level had returned to normal. The potassium concentration in the rats' blood after repeated bone trauma showed a significant increase on the third day of the experiment (152%) but subsequently was indistinguishable from the control level.

The total calcium concentration in the blood serum changed in a different manner. Whereas after primary bone trauma it fell sharply (on the 3rd day, 67.4% of the control value, $P < 0.02$), and then increased on the 15th day of the experiment (122.3%, $P < 0.02$), after repeated trauma the fluctuations in the blood calcium concentration were very small and the difference between the values in the experimental and control groups was not statistically significant.

The state of the calcium metabolism was reflected more accurately by the values of the specific activity (see the figure). The specific activity of the blood serum rose sharply on the 3rd day after primary bone trauma (141.8 % compared with the control value) and then fell gradually, to 77.5% of the control value on the 15th day (the difference is significant, $P < 0.05$). After repeated bone trauma the specific activity of the blood on the 3rd and 7th days was indistinguishable from the control level, and on the 15th day of the experiment it showed a slight decrease, although at this time also the difference was not significant.

The changes in metabolism of Ca^{45} in the femur were still more marked. The specific activity of the bone tissue after primary trauma showed its greatest increase after 15 days (192.5% of the control level, $P < 0.001$). After repeated trauma the rate of calcium metabolism in the femur increased sooner (on the 7th day 156.5% of the control level, $P < 0.001$) and more intensively than after primary trauma (on the 15th day 231.5%, $P < 0.001$).

The difference in calcium metabolism in the opposite uninjured femora in response to primary and repeated trauma was interesting. Whereas after primary trauma the specific activity of the opposite, intact femora developed regular phasic changes, as the author's earlier experiments [5] showed, after repeated trauma the rate of calcium metabolism in the intact bones was significantly increased by the 7th day (125%, $P < 0.05$) and the 15th day of the experiment (134.4% of the control level, $P < 0.02$).

Objective evidence was thus obtained for the first time showing that during repeated posttraumatic regeneration of bone a complex reorganization of the mechanisms of regulation of mineral metabolism takes place in the animal body: whereas the fluctuations in the concentration of potassium and sodium in the blood after repeated bone trauma were more marked than after primary trauma, the calcium level, on the contrary, was more stable. The rate of elimination of Ca^{45} from the blood during the 30 min from the time of administration of the isotope until the time of sacrifice of the animal likewise was indistinguishable from its values in the control series ($P > 0.05$). Meanwhile, both in the injured and in the uninjured opposite femur, following repeated trauma the rate of calcium metabolism increased by comparison with that after primary trauma.

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