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Informational Capacity of the Rat Sensorimotor Cortex in the Postresuscitation Period (Morphometric Analysis of a Neuronal Population)

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The number of neurons was counted in different functional neuronal complexes of the sensorimotor cortex in albino rats in the control and at different times after clinical death caused by asphyxia. A decrease of the number of neurons from 2.5×10^6 in the control to 1.56×10^6 toward the 30th day of the postresuscitation period was found in the sensorimotor cortex. The complexes of small neurons in the upper floor (layers II-IV) suffer badly, while the neurons of layer VI are damaged to a somewhat lesser degree and the cells of layer V are minimally altered.

Key Words: *sensorimotor cortex; neurons; postresuscitation period; morphometry*

The feasibility of rehabilitation of the integrative-trigger function of the sensorimotor cortex (SMC) in the postresuscitation period is determined by its informational capacity, which depends on the number of functioning nerve cells [4]. The sensorimotor cortex combines several functionally interconnected neuronal complexes with specific functions. The primary projection complex of neurons (layer V) accomplishes the reciprocal cortical-subcortical connections. The secondary projective-associative complex (lower third of layer III and layer IV, switches the afferent impulses from subcortical structures and redistributes them over the corresponding cortical areas. The tertiary associative complex of neurons (layer II and the upper two-thirds layer III) closes the cortico-cortical connections of the finest degree of differentiation. The deep-cortical neuronal complex (layer VI), the most ancient, weakly differentiated projection and associative system of the cerebral cortex, is identically represented over all cortical areas [2,5].

The lack of quantitative data on the morpho-functional state and informational capacity of the neuronal complexes in the neocortex hampers an understanding of the mechanisms of a postresuscitation encephalopathy and of the ability of the brain to restore its functions.

The present investigation was undertaken to determine the number of neurons in different neuronal complexes (the structural basis of informational capacity) in the SMC of albino rats in the postresuscitation period.

MATERIALS AND METHODS

Clinical death was modeled on male albino rats (45 animals) weighing 190-210 g by the clamping of an intubation tube for 6 min under ether anesthesia. Subsequent to asphyxia, attended by the cessation of the systemic blood flow, the animals were revived with closed chest massage and artificial pulmonary ventilation. The brain was fixed

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TABLE 1. Volumetric Density of Neurons in Neuronal Complexes of Albino Rat SMC in the Postresuscitation Period *(M±m)*

Time of investigation	Volumetric density of neurons in complexes, $10^3/mm^3$				
	primary	secondary	tertiary	deep-cortical	mean
Control	20.5±1.2	66.0±2.0	60.0±3.2	51.3±3.4	49.5±2.0
Postresuscitation period					
30 min	16.2±2.4	63.3±5.3	60.1±4.0	44.5±3.7	46.0±3.9
90 min	20.5±1.8	59.5±5.0	56.2±4.7	49.0±3.9	46.3±4.0
6 h	19.6±2.0	55.5±3.0*	56.5±4.3	43.1±3.3	43.6±3.4
1 day	17.5±1.2	45.2±3.0*	47.0±3.1*	50.0±3.4	39.9±2.7*
3 days	15.9±1.3*	40.9±3.8*	37.7±3.4*	37.7±3.7*	33.0±3.0*
7 days	14.5±1.1*	39.0±3.1*	36.0±3.0*	34.0±3.5*	30.8±2.6*
14 days	15.0±1.4*	38.0±3.8*	37.6±3.1*	36.2±2.0*	31.7±2.8*
30 days	14.0±0.9*	38.7±2.6*	37.0±2.5*	33.0±2.2*	30.6±2.0*

Note. * denotes $p<0.05$ in comparison to the control.

with a 1% glutaraldehyde and 4% paraformaldehyde mixture on phosphate buffer (pH 7.4) by perfusion through the ascending aorta under a pressure of 100-110 mm Hg within 30 min, 90 min, 6 h, and 1, 3, 7, 14, and 30 days after resuscitation. One cerebral hemisphere was embedded in paraffin. Oriented pieces of SMC from the other hemisphere were postfixed in osmium tetroxide, dehydrated, and embedded in an epon-araldite mixture according to routine procedures. Frontal 10-μ sections were prepared from paraffin blocks at the level of the SMC [3]. The area and volume of the entire SMC and of individual layers were determined by the line of demarcation between the regions of different cellular composition on every 20th serial section. Thereupon the SMC volumes of all sections were summated [8].

Semithin 1-m sections were cut through all cortical layers from the frontally oriented epoxide blocks. The sections were stained with toluidine blue. The numerical density of neurons was counted at 600 magnification on a 245,320 μ^2 area from each SMC level for each animal and converted to μ^2 of the section surface. Only nucleus-containing neuronal profiles were counted. The diameter of neuronal bodies was measured with a micrometer. The volumetric density of neurons in 1 mm^3 of SMC tissue was calculated taking into account the numerical density, diameter of neurons, and thickness of the section [6-8]. The data obtained were processed statistically using the Student *t* test.

RESULTS

The volume of the SMC of both cerebral hemispheres was 59.6±6.0 mm^3 in control animals. This index did not reliably change during the period following bloodflow restoration. In all cases the volume occupied by a functional complex in relation to the entire SMC volume was 10.3% for the molecular layer, 18.1% for the tertiary associative complex, 27.8% for the primary projection complex, and 25.6% for the deep-cortical complex of neurons. The mean volumetric density of neurons in the SMC of control animals was 49,500±2000 neurons per mm^3 of tissue. The highest density of neuronal distribution is typical for the secondary and tertiary neuronal complexes (Table 1).

The volumetric density of neurons in the SMC layers studied is not reliably altered 30 and 90 min after resuscitation. However, a tendency toward an increase of the number of hyperchromic neurons in the SMC (15.2%, $p>0.05$) is noted at the 90th min.

The mean volumetric density of SMC neurons remains at the control level after 6 h of reperfusion but 33.3% ($p<0.05$) of neurons have signs of hyperchromia and 9.5% ($p>0.05$) of cells signs of hypochromia. A moderate decrease of the numerical density of nerve cells by 15.9% ($p<0.05$) is found in the secondary complex.

After 1 day of reperfusion the mean volumetric density of SMC neurons drops by 19.4% ($p<0.05$). The content of hyperchromic neurons rises to 40.1% ($p<0.05$) and of hypochromic cells to 19.8% ($p<0.05$). Wrinkled neurons predominate. Neuronal deficiency is most pronounced in the secondary (31.5%, $p<0.05$) and tertiary (21.7%, $p<0.05$) complexes, i. e., in the upper floor of the SMC (Table 1).

After 3 days of reperfusion the mean volumetric density of neurons in SMC drops by 33.3% ($p<0.05$). The content of hyperchromic neurons is 11.9% ($p<0.05$) and of hypochromic cells 24.0% ($p<0.05$). The reduction of the neuron count occurs mostly in small-cell complexes (38.0%, $p<0.05$), while in the large-cell primary projection

complex the neuron deficit comprises a mere 22.4% ($p<0.05$, Table 1).

After 7 days of reperfusion the deficiency of the volumetric density of SMC neurons amounts to 37.8% ($p<0.05$); 14.9% ($p<0.05$) of preserved neurons are hyperchromic and 20.5% ($p<0.05$) are hypochromic. The neurons of the top floor are more affected, as at previous times (Table 1).

Fourteen and 30 days after revival the deficiency of the volumetric density of neurons is conserved at the previous level. Hyperchromic neurons account for 24.8% ($p<0.05$) on the 14th day and 9.8% ($p<0.05$) on the 30th day, while hypochromic neurons constitute 18.6% ($p<0.05$) and 11.3% ($p<0.05$), respectively.

Converted to the absolute number of nerve cells, the informational capacity of the SMC in control albino rats is determined by the presence of 2.5×10^6 neurons. The afferent input is accomplished by the activity of 0.72×10^6 stellate and small pyramidal neurons of the secondary projective-associative complex, while the efferent output is realized by 0.34×10^6 large and medium pyramidal neurons of the primary projection complex. The local intermodular associative relationships are executed by 0.64×10^6 neurons of the tertiary small-cell associative complex. The deep-cortical complex contains 0.8×10^6 neurons.

Only 1.56×10^6 neurons remain in the SMC 30 days after resuscitation, including 0.24×10^6 in the primary complex, 0.42×10^6 in the secondary, 0.40×10^6 in the tertiary, and 0.50×10^6 neurons in the deep-cortical complex. Consequently, the neuronal network which is responsible for perception and associative processing of information is more vulnerable during the postresuscitation period than is the network of large projection pyramidal neurons. If the informational capacity of the SMC in the control rats is taken as 1.0 according to the number of neurons, then after 30 days it will be 0.618 or 0.407 corrected for the number of pathologically altered neurons. According to the degree of morphological changes, this corresponds to a moderately severe postresuscitation brain pathology and the brain function may be partially restored thanks to the preserved neurons [1]. However, the uneven decrease of the neuron number in different functional complexes of the neocortex will probably lead to hypertrophy and hyperfunction of neurons of the cortical "input," to the formation of more powerful channels for the transmission of information than in the control, to a change of the ratio of excitation to inhibition in the SMC, and, as a result, to a new structural-functional organization and integrative-trigger activity of the brain in the late postresuscitation period.

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