Plasticity of Pyramidal Neuron Dendrites in Layer V of Rat Brain Sensorimotor Cortex during the Postischemic Period

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UDC 616.831-036.882-08-092.9

Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 116, № 7, pp. 94-96, July, 1993
Original article submitted February 11, 1993

Key Words: *sensorimotor cortex; pyramidal neurons; ischemia; plasticity*

Since hypoxia is known to play an important role in the pathogenesis of many diseases, we thought it of interest to study its effect on nervous tissue. Model animal experiments help elucidate theoretical and clinical aspects in investigations of compensatory rehabilitative processes in the central nervous system (CNS). It is currently believed that dendrites play the leading role in providing integrative activity and in elaborating central programs in the brain [4]. Many scientists have analyzed structural changes taking place in neurons and synapses after total-body ischemia [1,5], but there have been no reports about changes in pyramidal neuron dendrites over the course of the rehabilitative period after a terminal state.

The aim of this investigation was to elucidate the regularities governing pyramidal neuron dendrite restructuring in layer V of the rat brain sensorimotor cortex in the postischemic period.

MATERIALS AND METHODS

Experiments were carried out with 24 outbred white rats weighing 170 to 210 g. Total ischemia was induced by a 10-min clamping of the cardiovascular bundle under ether narcosis [2]. The animals were resuscitated by indirect massage of the heart and artificial ventilation of the lungs. The brain was taken from intact animals at the end of the tenth minute of ischemia and then 90 min and 1, 3, 7, 30, and 90 days after recirculation onset (three animals per term). The sensorimotor cortex (SMC) block was impregnated after Golgi and then 15 neurons of SMC layer V were sketched from frontal sections at magnification 600 in each group. Fifteen parameters of dendritic geometry were quantitatively analyzed using the A.S.M. system (Leitz, Germany) according to a previously described method [3]. Dendritic spines (DS) were counted on apical dendrites passing through layers I-II and III-IV, on oblique dendrites in layer III-IV, and on basal dendrites in layer V of the same neurons using an Ortholux microscope at magnification 600 per 100 μ of dendrite length. The reliability of the differences in parameters were estimated using the Kolmogorov-Smirnov test and the results were presented as the median.

RESULTS

All parameters of dendrite geometry in the sensorimotor cortex that were measured and estimated in the postischemic period after short-term total

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0007-4888/93/0007-0883$12.50

TABLE 1. Morphometric Characteristics of Pyramidal Neuron Dendrites in Layer V of Rat Brain Sensorimotor Cortex in the Postischemic Period

Parameter	Control	Postischemic period					
		90 min	1 day	3 days	7 days	30 days	90 days
Scl	729 (600–922)	829 (628–998)	714 (655–1037)	805 (646–1051)	929 (783–1047)	772 (616–951)	728 (560–769)
d	5 (5–6)	5 (4–6)	5 (4–6)	5 (4–6)	5 (5–6)	5 (4–6)	5 (5–6)
Rcomp	660 (623–710)	655 (644–664)	660 (652–681)	640 (629–663)	651 (646–668)	660 (630–673)	668 (646–672)
$Rcomp_1$	245 (219–269)	224 (205–235)	198* (171–210)	198* (181–215)	165** (151–183)	211 (195–264)	213 (195–252)
Ad	6.0 (5.1–7.3)	6.0 (4.8–7.2)	4.6** (3.9–4.9)	4.9* (4.0–5.3)	4.9* (4.0–5.2)	6.0 (4.6–6.6)	6.1 (5.2–7.0)
Ac	32 (29–37)	30 (28–35)	24** (23–28)	26* (22–28)	26* (24–28)	28 (25–39)	34 (31–39)
Nd	189 (164–220)	154 (131–170)	139 (114–173)	130** (111–163)	134** (120–154)	172 (148–203)	191 (164–217)
xFMB	0.56 (0.33–0.80)	0.42 (0.32–0.76)	0.43 (0.30–0.71)	0.45 (0.33–0.69)	0.48 (0.20–0.66)	0.53 (0.40–0.75)	0.60 (0.41–0.75)
Vdt	0.50 (0.41–0.69)	0.39 (0.30–0.50)	0.32** (0.23–0.41)	0.30** (0.22–0.39)	0.23** (0.21–0.26)	0.39 (0.30–0.65)	0.47 (0.32–0.70)
Ldt	8.4 (7.0–10.6)	6.3 (4.4–7.1)	5.3** (4.2–5.9)	4.2** (3.1–5.0)	3.7** (3.0–4.1)	7.0 (6.1–8.2)	8.2 (6.9–9.8)
Fdt	3.0 (2.0–3.5)	2.1 (1.8–3.2)	3.0 (1.1–4.0)	2.0 (1.6–3.1)	2.0 (1.0–3.1)	3.0 (2.1–4.0)	3.1 (2.0–4.0)
Ers	30 (24–36)	28 (26–32)	30 (24–33)	27 (23–30)	27 (24–28)	28 (25–32)	34 (29–37)

Note. Median (lower and upper quartiles); one asterisk – $p<0.05$, two asterisks – $p<0.01$ (vs. the control); Scl – cell body area, μ; d – number of all primary dendrites; Rcomp – maximal radius of apical dendrites, μ; $Rcomp_1$ – maximal radius of basal dendrites, μ; Ad – dendrite arborization; Ac – total arborization of cell; Nd – specific density of dendrite lengths in a section; xFMB – mean number of maximal branching foci per dendrite; Vdt – dendrite territory volume, mm; Ldt – total length of dendrites in entire dendritic territory, mm; Fdt – total number of maximal branching foci over entire dendritic territory; Ers – relative length of dendrites.

ischemia may be divided into two groups. Group 1 parameters remain virtually unchanged in the rehabilitative period as against the control (Table 1): Scl, d, xFMB, Fdt, Rcomp, Ers.

Group 2 parameters change as soon as one day after ischemia, this indicating plastic restructuring of dendrites. There is a reduction, in comparison with the control, of $Rcomp_1$ by 19.2% ($p<0.05$), of Ldt by 36.9% ($p<0.01$), of Vdt by 36.0% ($p<0.01$), of Ad by 23.4% ($p<0.01$), and of Ac by 25.0% ($p<0.01$), the reduction of the pyramidal neuron dendrites occurring mainly at the expense of the loss of terminal branches by oblique dendrites in layer III-IV. The number of pyramidal neuron DS decreased one day after ischemia by 25% ($p<0.05$) on oblique dendrites and by 17.1% ($p<0.05$) on apical dendrites in layer III-IV, whereas the number of DS on basal dendrites was virtually the same as previously.

The most noticeable changes in pyramidal neuron dendrites were seen on day 7 postischemia (Table 1): $Rcomp_1$ was reduced by 32.7% ($p<0.01$), Ldt by 56.0% ($p<0.01$), and Vdt by 54.1% ($p<0.01$). Pyramidal neuron dendrites were reduced at the expense of the loss of terminal branches by both apical and basal dendrites. The number of DS was reduced in comparison with the control by 27.8% ($p<0.01$) on apical dendrites in layer III-IV, by 46.1% ($p<0.01$) on apical dendrites in layer I-II, by 44.9% ($p<0.01$) on oblique dendrites in layer III-IV, and by 42.2% ($p<0.01$) on basal dendrites. Longer and thinner, in comparison with the control, dendritic spines appeared. Some spines became short and flat, this being associated with a widening of the spine neck, thus making the spines shorter.

Quantitative parameters normalized on days 30 and 90 of the postischemic period (Table 1).

Hence, the findings are evidence of a high sensitivity to ischemia of pyramidal neuron dendrites in layer V of the sensorimotor cortex, which is particularly marked on days 1, 3, and 7 of the postischemic period. Injury to, and reduction in the number of dendrites during this period may be

among the major causes of disorders in interneuronal connections and, eventually, in the summation and synchronization of cortical activity.

Marked destructive changes in pyramidal neuron dendrites in layer V of the SMC may develop nonuniformly. Our findings indicate that oblique dendrites and their spines on pyramidal neurons in layer III-IV are the most sensitive to ischemia; this is in line with the results obtained during various extreme exposures of the body [4-6]. It is on these dendrites that specific thalamocortical excitatory afferents and spinal neuron axons terminate. The decreased number of pyramidal neuron apical and oblique dendrites in layer III-IV most probably causes changes in the thalamocortical relationships and is responsible for the reduced informative value of afferent impulses from the thalamus.

On day 7 postischemia all pyramidal neuron dendrites in layer V of the SMC are maximally reduced: their length is decreased, as are their branching, the volume of dendritic territory, and the number of dendritic spines, the greatest changes being observed on apical dendrites in layer I-II and on basal dendrites. Basal dendrites are a receptor site for "chandelier" inhibitory cells and basket neurons, and nonspecific thalamocortical system fibers terminate here, too. The later reduction of dendrites in layers I-II and V may be connected with their inhibitory function. Hence, we revealed that excitatory inputs of pyramidal neurons in layer V of the sensorimotor cortex are the most sensitive to ischemia. This may account for the different reaction to ischemia of the inhibitory and excitatory elements of the cortex as a whole.

REFERENCES

1. V. G. Korpachev, V. G. Lysenkov, and L. Z. Tel', *Pat. Fiziol.*, № 3, 78-80 (1982).
2. T. A. Leontovich, *Neuronal Arrangements of Subcortical Formations of the Prosencephalon* [in Russian], Moscow (1978).
3. V. V. Semchenko, S. I. Ereniev, S. S. Stepanov, and R. I. Genne, *Arkh. Anat.*, **99**, № 7, 40-43 (1990).
4. H. Braak and E. Braak, *Progr. Neurobiol.*, **25**, № 2, 93-139 (1985).
5. K. Kawai, L. Nitecka, C. A. Ruetzler, *et al.*, *J. Cereb. Blood Flow Metab.*, **12**, № 2, 238-249 (1992).
6. A. B. Scheibel and L. Paul, *Exp. Neurosci.*, **89**, № 1, 279-283 (1985).