Sodium MRI after focal cerebral ischemia shows that the presence of collateral circulation does not affect the accumulation of brain tissue sodium, [Na⁺]

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Introduction

Brain $[Na^+]$ has been proposed to assess insult duration in evolving stroke (1). Stroke location and the type of local blood supply may modify this relation. We used sodium MRI (2) to test the hypothesis that the time course of $[Na^+]$ in cortical stroke (COR), where collateral circulation is present (3), is different from that in caudoputamen stroke (CP), where collateral circulation is absent (4).

Methods

Four Sprague-Dawley rats (~300 g) were prepared under endotracheal isoflurane anesthesia (0.8-2.5%), O_2 and N_2O (30%:70%), and volume controlled artificial ventilation (MR-1, CWE, Inc.). The femoral artery and vein were cannulated with PE-50 polyethylene catheters. Mean arterial blood pressure (MABP) was continuously monitored from a femoral artery, using a strain gauge transducer,



COR ROI CP ROI

Figure 1. Images of the rat head in the supine position. The brain is at the bottom of the image. Left Selected partition from 3D sodium image 5.4 hr after ischemia (5.3 min, 398 projections, NEX=10, TR/TE=100 ms/0.4 ms, 0.48 mm³ voxel size). The ROIs for the ischemic cortex (COR) and ischemic caudate putamen (CP) are shown. (**Right**) The ischemic region is clearly visible in the right hemisphere as high intensity in the DWI (TR/TE = 2 s/120 ms, b = 1500 s/m², 0.24 mm³ voxel size) 6.8 hr after ischemia. The circular structures at the top of the images are sodium calibration standards.

Table 1: Rate of [Na ⁺] increase in ischemic brain				
	Cortex, COR		Caudoputamen, CP	
Animal	Slope (%/hr)	р	Slope (%/hr)	р
1	15.3 ± 1.9	< 0.001	14.3 ± 1.4	< 0.001
2	8.6 ± 2.2	0.001	10.1 ± 2.1	0.001
3	30.2 ± 2.5	< 0.001	22.4 ± 2.3	< 0.001
4	21.9 ± 3.8	< 0.001	22.0 ± 2.4	< 0.001
Mean	19.0 ± 2.6		17.2 ± 2.1	



and recorded on a polygraph. Arterial blood gases (PaO₂, PaCO₂) and pH were monitored before and after stroke and at the end of the MR scan. Focal ischemia was induced by insertion of an intraluminal suture through the internal carotid artery (5). Infarct visualization was accomplished using ¹H DWI imaging. 3D sodium images were obtained on a 3 T whole body scanner (GEMS) using a dual-tuned $(^{23}Na/^{1}H)$, dual-quadrature

Figure 2. Time course of [Na⁺] for the ischemic cortex (red circles) and ischemic caudoputamen (blue squares). Each point in the graph corresponds to a sodium image collected in 5.3 minutes.

birdcage RF coil (2). [Na⁺]

maps (twisted projection, B1 corrected) were acquired every 5.3 min. $[Na^+]$ was measured as percent change from the average in normal cortex vs. time after occlusion (T_a) in hr. The experimental procedure included $[Na^+]$ measurements interspersed with DWIs over a period of ~4 hr. The locations of ROIs for the sodium slope calculation for the CP and COR areas are shown in Fig. 1.

Results

Fig. 2 shows $[Na^+]$ time courses in the ischemic cortex and caudate putamen with similar slopes from animal #4. The rates of $[Na^+]$ increase for COR and CP strokes (see Table 1) for each animal show no differences between the COR and CP regions.

Conclusions

The rate of $[Na^+]$ increase for cortical and caudoputamenal stroke was similar. Thus, the $[Na^+]$ time course can describe the progression of stroke for cortical as well as for subcortical regions, suggesting that it is independent of the potential for collateral arterial supply. Possible explanations for the similar time course

of $[Na^+]$ in cortical and caudoputamenal stroke include: 1) partial occlusion of the vessels by intraluminal suture permitted some residual flow; 2) low resolution of Na MRI resulted in "partial volume" effect of sampling from two adjacent regions; and 3) residual flow in the ischemic caudate putamen.

References

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