central venous catheter was introduced upward until the tip of the catheter was obstructed by the bone. Then, the catheter was drawn back 0.5 cm. The catheter tip location was verified by an antero-posterior X-ray taken in the recovery room. This technique was successfully applied for 48 patients. The mean length from the skin insertion to the tip of the catheter was 13 ± 0.2 cm (mean ± SD). It is recognized that the technique is very easy and no complication or dislocation of the catheter tip occurred.

Sincerely Yours,

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References

Flow and Neuronal Density in Tissue Surrounding Chronic Infarction

To The Editor

We should like to congratulate Dr. Mies and his colleagues on a most elegant study (Stroke 14: 22–27, 1983), and in particular for their lucid discussion of the potential role of diaschisis in electrophysiological suppression in areas where flow is reduced but remains above infarction threshold.

Sincerely yours,

Yours truly,

Associate Professor of Acute Medicine

And

Professor and Chairman

References
by guest on August 14, 2017 http://stroke.ahajournals.org/ Downloaded from

The letter of Dr. Strong and colleagues refers to their recently published investigations on electrophysiological, hemodynamic, biochemical and histological changes occurring 2 hours after onset of focal ischemia.1,2 Although our data gave no indication of neuronal loss in the marginal gyrus 8 weeks after chronic brain infarction,3 it was of interest for us that indeed evidence exists for the acute histopathological changes of neurons in the marginal gyrus after shortlasting MCA occlusion concomitant with early diaschitic electrophysiological impairment. Discrete or even single neuronal cell loss, of course, will be very difficult to detect when assessed in several 0.2 x 0.2 mm areas at a magnification of 400. To correlate the histological appearance of tissue with regional autoradiographic blood flow measurements, this requires at least 1 mm2 to be well above the autoradiographic resolution and certainly is a compromise in our study. In the chronic stage of infarction, there are, however, variations of the neuron-flow relationship in at least 1 mm2 to be well above the autoradiographic resolution and certainly is a compromise in our study. In the chronic stage of infarction, there are, however, variations of the neuron-flow relationship in regions of the dorsal thalamus or in the inferior olive to be more resistant to axonal dissection when collateral branches exist compared to axonal sectioning.6 Dissection of the optic nerve (eye enucleation) is known to lead to cessation of synaptic transmission in the lateral geniculate nucleus after 3-4 days concomitant with the degeneration of synaptic terminals.7 In chronic preparations the lateral geniculate body is reduced in volume and increased in cell density (gliosis) indicating neuronal loss with subsequent fiber degeneration.8 Thalamic lesions cause Wallerian degeneration which extends to the deep layer of the cortex.9 After entorhinal lesions involving the efferent axons of the CA1 pyramidal neurons delayed degeneration of the dorsal part occurs.10 On the other hand, after white matter lesions diaschistic impairment of the EEG was reported before.11 Functional deafferentation between subcortical-cortical connections of whatever origin might very well influence the outcome of tissue surrounding chronic infarction. What we hypothesize is that white matter infarcts beneath the gray matter may contribute to the late neuronal loss when these axons are destroyed from neurons which do not collateralize or are vulnerable to axonal interruption. However, we also recognize that, for example, the development and delayed spread of brain edema within the white matter could also contribute to such changes apart from hemodynamic disturbances. The historical appearance of neuronal tissue around chronic infarctions, therefore, is likely to reflect the result of all damaging equivalents occurring during this period.

Careful studies of the interval between the acute and chronic stage of the brain infarctions are needed to supply the missing information on the pathophysiological sequelae after such an event.

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Flow and neuronal density in tissue surrounding chronic infarction.
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