Neurobiology of Passive Avoidance Impairment After Ischemia

Karasawa et al report that passive avoidance impairment after ischemia is related to damage of CA1 neurons of the hippocampus related to memory and habituation. The neurobiology is suggested by the release of dopamine1 lateralized to the right hemisphere2 subserving passive avoidance.3 This hypothesis is supported by optimal response organization at intermediate dopamine tone in a delay-dependent speeding of reaction time, indicating motor readiness, is abolished by depletion of dopamine subserving mood prompts the evaluation of ischemia-induced behavioral changes by monitoring behavioral correlates of mood, ie, speech hesitation and switching pauses.2 This method is supported by the contribution of articularatory rehearsal to short-term memory3 and by the association of >2-second speech pauses with prearticularatory repair4 and competitive and courtship activity.2 These findings suggest a causal relationship between impairment in passive avoidance and neuronal damage in the hippocampus,8 thus tending to confirm powerful isomorphisms between mind and body and the existence of deep and lawful mental structures governing human cognitive and emotional functioning reflecting properties of neuronal activity and firing.9

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References


Response

We thank Drs Hart, Halperin, and Miller and Ms Pearce for their interest in our article on the factors associated with the failure of aspirin treatment after stroke.

Unfortunately, the small sample size of patients with atrial fibrillation does not allow us to further stratify the patients to perform multivariate analysis (Cox proportional hazards model).

As for the second point, we indeed found that ischemic heart disease is a significant risk factor for aspirin failure, since even after Bonferroni correction the odds ratio for this risk factor remain statistically significant.

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Experimental Stroke Treatment With High-Dose Methylprednisolone

We read with interest the recent article by de Courten-Myers et al.1 There are several parallels between this study and a study we recently published.2 Because ischemia is a primary pathophysiological mechanism in epidural brain compression,3 both studies involve transient regional cerebral ischemia. Both the steroids in the study by de Courten-Myers et al and the hypothemia in our study are considered to protect and resuscitate by affecting several sites along the ischemic cascade.4 In both studies treatment was initiated early, 30 minutes into the insult by de Courten-Myers et al and 15 minutes into the insult in our study. The results of the two studies are remarkably similar. In neither study did treatment decrease mortality, but in both studies treatment significantly decreased the cerebral infarct size (P<.05 in the de Courten-Myers study and P=.07 for infarct and P=.03 for overt histological damage in our study). In the de Courten-Myers study treatment clearly improved cerebral blood flow during ischemia (P<.005),
Neurobiology of passive avoidance impairment after ischemia.

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