Comment: Revisiting Lassen cerebral blood flow constancy with hysteresis and 3D

Roh et al.[1] present a unique case of severe, prolonged hypercapnic respiratory failure that triggered refractory global cerebral edema (GCE). This case report is interesting because edema was not abatable by hyperventilation, despite adequate reversal to a normocapnic state, and responded only to aggressive osmotherapy.

The relationship between cerebral blood flow (CBF) and arterial partial pressure of carbon dioxide ($PaCO_2$) may thus be represented visually by an S-curve with a delayed return in response to prolonged hypercapnia (figure 1). At a certain threshold of $PaCO_2$ that is sustained for a substantial duration, a state of "vasoplegia" might be triggered, whereby reversal of $PaCO_2$ levels is no longer effective in decreasing CBF. In reality, regulation of CBF is a complex process[2] that can be further comprehended by examining correlations between cerebral perfusion pressure (CPP) and CBF, and also between $PaCO_2$ and CBF, with a 3D interplay (figure 2). It is worth emphasizing that autoregulation, vasoreactivity, and blood–brain barrier impermeability are nuanced physiologic functions, not always directly related,[3] and each can lead to dysregulated CBF and to GCE. Serial measurements of CBF, CPP, and permeability, by neuroimaging and intracranial probes, would have been ideal to scrutinize the effects during hyperventilation and osmotic treatment in this patient.

Readers should not extrapolate these therapeutic suggestions for a noninjured brain with acute on chronic hypoventilation to common cases of brief exposure to severe hypercapnia. Finally, the fluid-attenuated inversion recovery image depicts GCE with an intriguing predilection for subcortical U fibers and periventricular CSF spaces, suggesting venular hypertension, rather than diffuse white and gray matter involvement, as expected in global cerebral capillary vasoplegia.

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Figure 1 Effect of arterial partial pressure of carbon dioxide ($PaCO_2$) onto cerebral blood flow (CBF) in usual circumstances and in this acute on chronic prolonged hypercarbic case triggering hysteresis

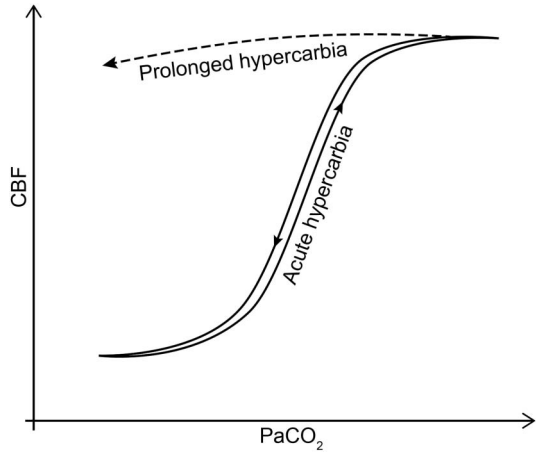

Figure 2 Three-dimensional representation of the interplay among cerebral perfusion pressure (CPP), arterial partial pressure of carbon dioxide ($PaCO_2$), and cerebral blood flow (CBF)