Impact of traumatic lesions on intracerebral probe positioning

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Sir: With great interest I read the work by Longhi et al., who investigated the importance of probe location on brain tissue oxygen tension ($ptiO_2$) in patients with different traumatic lesions [1]. The authors address a highly important and difficult issue, as heterogeneous traumatic lesions exhibit regionally and temporally heterogeneous functional and structural alterations. To date, the impact of probe positioning on lesion-specific diagnostic and therapeutic steps remains unanswered.

As shown by Dr. Longhi and colleagues, $ptiO_2$ is reversibly decreased pericontusionally compared to normal-appearing tissue. In general, however, this rather marginal difference will be missed at the bedside. Interestingly, pathologic $ptiO_2$ values were not reflected by abnormal $SjvO_2$, strongly indicating regional heterogeneity and supporting simultaneous measurements at different locations.

In patients with contusions, $ptiO_2$ probes were to be located within the ischemic hypodense pericontusional area. However, the exact probe location remains unclear. Different depth of insertion and distance from the lesions could be important confounding variables, since gray and white matter differ in terms of perfusion and metabolic profile. $ptiO_2$ probes were inserted during initial surgery early after trauma with small pericontusional hypodensity. Over time, contusion growth will encompass the initial hypodense region, expanding its ischemic halo, thus possibly enclosing the inserted probes [2]. This could explain the persistently low $ptiO_2$ values until day 3 after injury [1]. The subsequent significant increase could be related to the resolving contusion with improved pericontusional perfusion due to reduced perilesional edema formation. Serial perfusion CT scans might be helpful in assessing perilesional cerebral perfusion [3].

As clearly shown by the authors, normal-appearing tissue does not guarantee intact perfusion, oxygenation, and metabolism as seen under clinical and experimental conditions [4, 5]. Although tissue compression induced by epidural and subdural hematoma is reversible upon hematoma removal, metabolic brain injury can persist [4]. Thus, it is important to define "diffuse injury" more closely and consider the actual lesion types which are not differentiated by the Marshall CT classification. This might provide more detailed insight into pathologic $ptiO_2$ values as reflected by a metabolic penumbra zone [6], especially since the majority of patients suffer from different lesions existing simultaneously.

While $ptiO_2$ was collected continuously, other parameters were determined at only two time points per day. Perhaps important alterations were missed. Furthermore, dynamic influences of therapeutic measures were not considered.

All patients were hyperventilated. In patients with contusions and pericontusional ischemia, this could have induced more severe local damage, explaining their lower $ptiO_2$ values during the early phase.

Perhaps patients with focal lesions require higher CPP levels to correct decreased perilesional $ptiO_2$ values. $ptiO_2$-, microdialysis-, and $SjvO_2$-controlled CPP requirements are dynamic and require flexibility. Pathologic changes are also found beyond day 5.

Regionally heterogeneous alterations in metabolism and perfusion and the duty to protect and not harm the already injured brain force us to gain adequate insight into otherwise occult changes within the brain. This requires introduction of several probes to monitor different regions simultaneously. The future will show whether it may be unethical to confine measurements to only one location following traumatic brain injury.

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