Less Autoregulation and More Flow in Subarachnoid Hemorrhage

To the Editor:

We read with interest the article by Jaeger et al.1 published in the March issue of Stroke. In that article, tissue tension of oxygen (PtiO2) has been used as a surrogate marker of cerebral blood flow. After subarachnoid hemorrhage, in some patients a correlation (measured as the moving linear correlation coefficient, ORx) was found between changes in cerebral perfusion pressure (CPP) and corresponding changes in PtiO2, whereas in others this correlation was not detected. The absence of correlation was interpreted as a sign of preserved autoregulation, because a constant “flow” was measured despite CPP variations; more importantly, the patients who showed a correlation, measured as a positive ORx, had more delayed infarctions.

We congratulate with the brilliant use of monitored parameters to explore delicate mechanisms of vascular regulation, but we propose a few remarks.

Vasospasm is a frequent complication of subarachnoid hemorrhage, causing hyperperfusion and delayed ischemic damage. When the vessel’s caliber is pathologically reduced but not abolished by vasospasm, a CPP increase may restore, at least partially, flow.2,3 In this situation, therefore, a parallel increase of CPP and PtiO2 will indicate successful therapy rather than a deleterious loss of autoregulation. Conversely, a persistently low PtiO2 despite active increase of CPP (by vasopressors, for example) could indicate extreme spasm and failing therapy, rather than preserved autoregulation.

This has been tested in patients with acute brain damage,4 in subarachnoid hemorrhage cases,2 and in head-injured patients,4–6 in whom a significant PtiO2 increase caused by CPP augmentation has been documented and interpreted as a successful flow restoration. In the specific situation of patients with vasospasm submitted to active CPP increase, cases responsive to treatment could be categorized as cases with “lost autoregulation,” whereas cases not responding, and therefore more severe and condemned to ischemia, could be interpreted as cases with preserved autoregulation.

The second remark concerns the spatial limitation of PtiO2 monitoring. PtiO2 represent a balance between oxygen delivery and consumption in a very small volume of tissue, in the order of few cubic millimeters. We are reluctant to extend the findings of a focal measurement to the rest of the cerebral tissue, especially when delayed infarction develops distant from the probe’s position. If a high ORx is detected far from the infarction, it is difficult to explain why the tissue with impaired autoregulation will remain “healthy” but will predict infarction elsewhere.

Finally, PtiO2 depends on various physiological parameters; for example, it varies widely depending on arterial oxygen tension and temperature.7 Moreover, changes in arterial CO2 tension could affect vascular resistance. In various situations, quite common during the intensive care unit course, ORx calculation could be influenced by PtiO2 changes not exclusively related to vascular autoregulation.

We think that these remarks do not antagonize the data and the reasoning of our German colleagues. Perhaps they suggest additional ways, with potential clinical implications, of interpreting their findings.

Disclosures

None.

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