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Reply to: return of spontaneous circulation after an out-of-hospital cardiac arrest: an acute brain injury like others?

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Despite its non-invasive nature, Transcranial Doppler Sonography (TCD) remains underused at the return of spontaneous circulation (ROSC) phase after out-of-hospital cardiac arrest (OHCA). This is probably due to the low interrater reliability of some TCD measurements and the conflicting results in previous studies[1,2]. In our study, we found that patients with poor neurological outcomes had lower diastolic flow velocity (DFV) and higher pulsatility index (PI) at early stage after ROSC than patients with good neurological outcomes [4]. Therefore, we assumed that TCD measurements may allow to estimate cerebral blood flow (CBF), cerebral vascular resistance and therefore to optimize cerebral perfusion[3].

Nevertheless, TCD measurements provide only a surrogate of the cerebral blood flow (CBF) and does not allow to affirm if the cerebral autoregulation is preserved or not[5].

As Engrand et al. have pointed out, the relationship between TCD and CBF, and the interpretation of an elevated PI are complex and not completely understood.

Indeed, several processes would occur following a recovered cardiac arrest: elevation of the CBF, metabolic disorders, imbalance of the balance supply and need for tissue oxygen, reperfusion lesions, all contributing to cell death[6].

As mentioned in our study, the CBF alteration profile in post cardiac arrest has been studied in animals, and shows four phases[7]: absence of flow during cardiac arrest, hyperemia in the first 30 minutes following ROSC, hypoperfusion in the following 6 hours and then last fixed phase with either a return to normal, or persistence of prolonged hypoperfusion, or hyperemia. We hypothesize that TCD measurement should be focused on the early phase of ROSC and may allow the development of individualized targeted therapies to improve cerebral perfusion at this phase. Different targeted therapies have been tested in animals which would improve the neurological prognosis by reducing early hyperemia of the subcortical areas[8,9].

We believe that TCD should complement other neuromonitoring tools. The tools for monitoring cerebral hemodynamics are widely used for traumatic brain injury[10], but are poorly developed in the management of the patient in post-ROSC.

Finally, results of our study strongly encourage us to carry out further investigations to understand the different mechanisms involved at early stage of ROSC in CBF modifications, as recommended for others acute brain injury situations.

Conflicts of interest statement:

On behalf of all authors, the corresponding author states that there are no conflicts of interest.

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