Introduction:
Blood pressure management in post cardiac arrest (CA) patients ensures sufficient cerebral perfusion to avoid secondary brain injury. In previous studies, lower mean arterial pressure (MAP) was associated with poor clinical outcome. However, the relationship between hypotension and severity of hypoxic-ischemic encephalopathy (HIE) remains uncertain.

Methods:
We retrospectively analyzed MAP during the first week in three CA patient groups: (1) Patients surviving with no/mild HIE – cerebral performance category (CPC) 1. (2) Patients surviving with severe HIE – unresponsive wakefulness syndrome (CPC 4). (3) Deceased patients with post-mortem brain autopsy. In these autopsy patients, we used the Selective Eosinophilic Neuronal Death (SEND) classification to quantify the histopathological severity of HIE.

Results:
We included 354 autopsy patients, 118 had histopathological severe and 236 patients no/mild HIE. Between autopsy patients with no/mild HIE and severe HIE, there was no difference in mean MAP during the first week. However, no/mild HIE autopsy patients with post-CA regain of consciousness had a mean MAP of 75 mmHg compared to 65 mmHg in those never conscious before death. We found no difference in MAP between 216 patients with full neurological recovery (CPC 1) and 57 surviving with unresponsive wakefulness syndrome (CPC 4). Hypothermia induced small reversible hypotension in all groups.

Conclusion:
Contradicting previous studies, we found no difference in post-CA MAP of patients with histopathological and clinical evidence of presence and absence of HIE. No/mild HIE patients with post-CA regain of consciousness before death showed high-normal blood pressure stability. This may indicate a blood pressure effect on neuronal function rather than structural integrity. Importantly, extracerebral causes for loss of consciousness and mortality need to be considered.