Introduction:
Routine nursing procedures (NP) can interfere with blood pressure and cardiac output and may therefore alter cerebral hemodynamics in critical illness. This may be a risk factor of sepsis-associated encephalopathy.

Methods:
20 sedated and mechanically ventilated pigs were randomized to fecal peritonitis or controls (n=10, each). After 8 hours of untreated peritonitis, the animals were resuscitated for 76 hours (resuscitation period). NP [assessment of sedation (AS), tracheal suctioning (TS), change in body position (CP), lung recruitment maneuver (RM)] were performed at baseline and 8h, 32h, 56h and 72h after start of RP. Systemic and cerebral hemodynamics and O₂ saturations were recorded continuously.

Results:
After sepsis induction, mean arterial pressure (MAP) decreased by 15 (7-22) mmHg, cardiac output by 1.3 (0.7-2.2) L/min and arterial lactate increased by 0.2 (0.1-0.5) mmol/L. Intracranial pressure (ICP) was not affected. In controls, NP decreased MAP (max. 23±11mmHg) and increased ICP (max. 3.0±1.4mmHg). Cerebral perfusion pressure (CPP, max. 26±11mmHg) and carotid arterial flow decreased (max. 79±27ml/min). Thus, cerebral O₂ delivery (DO₂, max. 0.58±0.33ml/min/kg) and superior sagittal sinus O₂ saturation (SssO₂, max. 20±15%) decreased markedly, despite maintained arterial O₂ saturation (all p<0.05). The alterations recovered within 1 min after end of NP. The effects of NP on CPP, DO₂ and SssO₂ were attenuated during the resuscitation period. NP with most impact on SssO₂ were TS, CP and RM. In septic animals, NP led to similar changes as in controls. Higher SssO₂ values in sepsis – both before and after NP - were the result of numerically higher cerebral DO₂ and lower O₂ consumption (VO₂).

Conclusion:
NP impaired cerebral O₂ transport similarly in control and in septic animals, but the changes were short lasting and attenuated during the course of treatment. In septic animals, cerebral VO₂ was numerically lower than in controls, despite numerically higher DO₂.