Introduction:
Brain tissue hypoxia (brain tissue oxygen tension, PbtO2<20mmHg) is common after subarachnoid hemorrhage (SAH) and associated with poor outcome. Recent data suggest that brain oxygen optimization is feasible and may reduce the time with brain tissue hypoxia to 15% in patients with severe traumatic brain injury.[1] Little is known about the effectiveness of protocolized treatment approaches in poor-grade SAH patients.

Methods:
We present a retrospective analysis of prospectively collected data of 105 poor-grade SAH patients admitted to 2 tertiary care centers where PbtO2<20mmHg was treated using an institutional protocol. Treatment options were left to the discretion of the treating neuro-intensivists including augmentation of cerebral perfusion pressure (CPP) using vasopressors if necessary, treatment of anemia and targeting normocapnia, euvolemia and normothermia. The dataset used for analysis was based on routine blood gas analysis for hemoglobin data matched to 2 hourly averaged data of continuous CPP, PbtO2, temperature and cerebral microdialysis (CMD) samples over the first 10 days of admission.

Results:
Patients were admitted with a GCS of 3 (IQR 3-4) and were 58 (IQR 48-66) years old. Overall incidence of brain tissue hypoxia was 25%. During this time we identified associated episodes of CPP<70mmHg (27%), hyperglycolysis (CMD-lactate>4mmol/L, CMD-pyruvate>120µmol/L; 26%), pCO2<35mmHg (19%), metabolic distress (CMD-lactate-to-pyruvate-ratio>40; 18%), PaO2<80mmHg (14%), Hb<9g/dL (10%), and temperature>38.3°C (4%) (Figure). Of these variables only hyperglycolysis was significantly more common (37%) during episodes of normal PbtO2 (75% of episodes).

Conclusion:
Our results present clinical data of protocolized PbtO2-targeted therapy and show that there is room for further optimization. A larger cohort with predefined interventions is needed to proof the effect on longterm outcome after SAH.

References:
The black bars represent the percentage of abnormalities of factors shown in the x-axis during brain tissue hypoxia.