S68 Posters

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## AP-089

Sevoflurane during cardiopulmonary resuscitation improves early post-resuscitation myocardial dysfunction in the rat

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Introduction: Post-resuscitation myocardial dysfunction is an important cause of death in the intensive care unit after initially successful cardiopulmonary resuscitation (CPR) of pre-hospital cardiac arrest (CA) patients. It is well known that volatile anaesthetics reduce ischaemic—reperfusion injury in regional ischaemia in beating hearts. This effect, called anaesthetic-induced pre- or post-conditioning, can be shown when the volatile anaesthetic is given either before ischemia or in the reperfusion phase. However, up to now, no data exist for volatile anaesthetics after global ischemia due to CA. Therefore, the goal of this study was to clarify whether Sevoflurane improves post-resuscitation myocardial dysfunction after CA in rat.

Methods: Following institutional approval by the Governmental Animal Care Committee, 29 male Wistar rats (350–400 g) were randomized either to receive Sevoflurane 2.5 vol.% (Sevo group) for 5 min starting at the beginning of CPR, or to the control group without Sevoflurane. After 6 min of electrically induced ventricular fibrillation, CPR was performed. Following restoration of spontaneous circulation (ROSC), continuous measurement of ejection fraction (EF) and end-diastolic volume (EDV) was performed using a Millar catheter. All data are given as median (t-test; p < 0.05).

Results: During the first 3 h after ROSC, EF increased in the Sevo group from 24% to 38%, while animals in the control group showed no increase at all (24%; Sevo vs. control, p < 0.01). EDV values before CA were 258  $\mu$ l (Sevo) and 220  $\mu$ l (control), respectively. There was no significant change in EDV in the Sevo group (280  $\mu$ l), whereas EDV increased to 410  $\mu$ l in the control group (p < 0.01).

Conclusions: In this animal model of CA and resuscitation, administration of Sevoflurane improved two crucial parameters of myocardial function. Increased EF and lowered EDV due to the application of Sevoflurane might be a promising base for new therapeutic approach after CA.

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## AP-090

The rate of recovery of cardiac function after cardiac arrest: A theoretical study

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Introduction: During a cardiac arrest there is an initial rapid fall in arterial pressure followed by a more gradual decline until arterial and venous pressures equalize. This is accompanied by distension of the right ventricle and compression of the left ventricular cavity. The aims of this study were to develop a mathematical model of the circulation to simulate a cardiac arrest and to investigate the relative importance of fluid shifts in the circulation and ischaemia on the rate of recovery of blood pressure post-arrest. This rate of recovery is important in determining duration of CPR required after restoring normal rhythm.

Methods: The computer simulation model coupled right ventricle (RV), left ventricle (LV) and atria enclosed within the

pericardium. The circulatory system components included large to small arteries and veins as well as the capillary bed. Cardiac arrest was simulated by stopping all active cardiac contraction and incorporated changes in vascular resistance and compliance. Recovery was simulated by starting active contractions dependent on the RV and LV cavity pressures and sizes and the degree of ischaemia (increased chamber stiffness and reduced contractility). Results: The simulation mimicked clinical results with a sharp then gradual fall in aortic pressure along with RV dilation. After 10s of cardiac arrest the recovery of arterial blood pressure was rapid (<5s). After 1 min or more, the RV dilation and impingement on the LV cavity prolonged recovery to 20s. For more prolonged arrests, recovery to normal aortic pressure was delayed by simulated ischaemia for up to 2 min.

*Discussion*: The model simulates the main features of cardiac arrest physiology in detail. Recovery of aortic pressure after a cardiac arrest is more prolonged with longer duration of arrest, mainly due to the effects of ischaemia but also, to a lesser extent, due to RV dilation.

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## **PROGNOSIS**

## AP-091

Comparison between simultaneously recorded continuous encephalogram and standard encephalogram in post-cardiac arrest patients

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Introduction: During post-anoxic coma, the value of standard electroencephalogram (stEEG) is limited by its inability to provide a continuous assessment. A continuous EEG (cEEG) monitoring system (S5<sup>TM</sup> Datex Ohmeda, Finland) is now available but its efficiency is not established. We assessed the value and limitations of cEEG by comparison with stEEG in post-cardiac arrest patients treated by mild hypothermia.

Methods: Over 6 months, all consecutive patients in post-cardiac arrest were studied. During the first 36 h, 1 cEEG and 3 stEEG per patients were recorded. The cEEG was spliced into 3-h periods which were analyzed off-line by a neurophysiologist and two intensivists blinded for stEEG results. Each analysis was classified as normal voltage, low voltage, epileptic activity, burst suppression and flat EEG. Ability of the cEEG to predict stEEG patterns was evaluated by assessing sensitivity, specificity, positive and negative predictive values (PPV and NPV). We also assessed the influence of hypothermia, sedation and the inter-observer agreement (see Table 1).

Results: 23 patients were studied (mean age  $57\pm17$  years) of whom 27% survived. 62 pairs of cEEG and stEEG were suitable for analysis. The inter-observer agreement was 0.67.

The PPV and NPV of cEEG to predict the existence of a severe abnormal stEEG pattern (epileptic activity or burst suppression or flat EEG) were, respectively, 73% and 89% but improved in normothermia and after sedation termination (PPV 88% and NPV 93%) (see Table 1).

Table 1				
	Sensitivity	Specificity	PPV	NPV
Normal voltage (%)	100	94	80	100
Low voltage (%)	63	88	81	75
Epileptic activity (%)	50	80	8	98
Burst suppression (%)	55	98	86	91
Flat EEG (%)	60	100	100	93