were identical. The half inactivation potential \( V_{1/2} \) was significantly different.

\[ \text{Materials and Methods:} \] The study was realised on two groups of rats: group S rats underwent caecal ligation and puncture and C rats were the control group. Using the patch-clamp technique in isolated rat flexor digito- 

brevis myofibers, we investigated the electrophysiological effects on \( Na(\pm) \) 10 days after the start of sepsis.

Results and Discussions: 63 and 36 measurements were realised in group S and C respectively. Voltage dependence of peak \( Na(\pm) \) current \( (gNa) \) was low 10 days after the start of sepsis. We conclude that it is possible to calculate from conductance the number of open gate channels. \( (n B S: 279 \text{ vs } C 498) \). The magnitude of \( Na(\pm) \) current amplitude \( (I m) \) was diminished. However the activation \( m \) and inactivation \( 

\( V_{m} \) constants of gate channels were identical. The half inactivation potential \( V_{1/2} \) was significantly different.

Results: Sepsis attenuates sodium channel activity, decreases the peak \( Na(\pm) \) current amplitude and the conductance without modifying the opening properties of channel gating.

Sepsis causes modification of conductance in Na channels at NMJ. This can be due to modification of the number of channels, or variation of their subunits. Further studies must be done to confirm this hypothesis.

Voltage-gated sodium channel changes in NMJ during sepsis

A-678

Euglycemic hyperinsulinemia in an ewe septic shock model


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Introduction: Euglycemic hyperinsulinemia (EH) has been shown to reduce plasma concentrations of tumor necrosis factor alpha in lipopolysaccharide-induced systemic inflammation in pigs (1). The aim of our study was to investigate whether EH could improve survival in a seepic shock model.

Methods: Fourteen anesthetized, mechanically ventilated, hemodynamically monitored sheep received 1.5-g/kg body weight feces intra- 

toanally to induce sepsis. After two hours, animals were randomized to: a) control group (n = 7) – insulin 0.25U/kg/hour, 20% glucose (to maintain blood glucose 40–90mg/dl), and potassium (to maintain potassium level 4.0–5.5mM/L); control group (n = 7) – no insulin. Ringer’s lactate (RL) + hydroxyethyl starch (Voluvien) (volume ratio = 1:1) was titrated to maintain pulmonary artery occlusion pressure (PAOP) at baseline level throughout the experimental period without administration of any antibiotics or vasoactive drugs. The animals were followed until spontaneous death or for a maximum of 30 hours.

A-679

Can liver perfusion improve by adding dobutamine to norepinephrine in septic shock?

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Background and Goal of Study: To investigate the effect of dobutamine when added to norepinephrine on liver perfusion as measured by the plasma disappearance rate (PDR) of indocyanine green (ICG) in septic shock.

Materials and Methods: Ten patients fulfilling the criteria of septic shock were recruited in this auto-control clinical trial. Mean arterial pressure (MAP) was kept above 70mmHg with norepinephrine and invasive haemodynamic monitoring by PICCO was commenced. Hypovolaemia as indicated by intrathoracic blood volume index (ITBVI) <850ml/m², was corrected and then the PDR ICG was measured by the LIMON, by giving 0.25mg/kg ICG. If PDR <18% dobutamine was administered at a rate of 5µg/kg/min. After 60 minutes of treatment haemodynamic and PDR measurements were repeated. Blood samples were taken when haemodynamic stability by nor- epinephrine was achieved (t0) and after one hour of isotropic support (t1).

Results and Discussions: There was no difference in cardiac output (4.0 ± 0.9 vs. 4.0 ± 0.9L/min/m², p = 0.91), heart rate (102 ± 15 vs. 108 ± 25/min, p = 0.95), central venous haemoglobin saturation (76 ± 9 vs. 77 ± 10%, p = 0.37), and serum lactate levels (1.75 ± 1.45 vs. 2.15 ± 1.40mmol/L, p = 0.36) between the two measurement points. PDR increased slightly but it did not achieve statistical significance (14.7 ± 6.7 vs. 16.1 ± 8.7%, p = 0.22).

Conclusion(s): The results of this pilot study suggest that routine adminis- 

tration of dobutamine to norepinephrine in order to improve liver perfusion in septic shock cannot be supported, as indicated by the non-significant change in ICG PDR. These results are in contrast to recent animal data (1), however, the completion of the study is required to come to firm conclusions.

References:

A-681

Terlipressin or norepinephrine in hyperdynamic septic shock: a prospective, randomized study

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Background and Goals: To compare, in patients with hyperdynamic septic shock, the effects of norepinephrine (NE) or terlipressin (TP) on haemody- 

namic parameters and renal function.

Material and Method: Twenty patients with hyperdynamic septic shock were prospectively randomized to receive NE or TP after fluid resuscitation. Global haemodynamic parameters, oxygen consumption, urine flow, creati- 

nine clearance, and arterial blood lactate levels were measured.

Results: Mean arterial pressure, systemic vascular resistance, pulmonary vascular resistance, and left and right ventricular stroke work were signifi- 

cantly increased with both drugs. With TP, but not with NE, a significant decrease in heart rate (from 113 ± 17 to 104 ± 11b/min, P < 0.01) and carbon dioxide (from 5.1 ± 1.7 to 4.2 ± 1.6L·min⁻¹·m⁻², P < 0.05) was observed, with no change in stroke volume. Oxygen delivery (from 784 ± 131 to 701 ± 92mL·min⁻¹·m⁻²) and consumption (from 244 ± 69 to 210 ± 54mL·min⁻¹·m⁻²) were significantly decreased with TP, but not

References:
with NE. Blood lactate concentrations were significantly decreased with both drugs. Renal function assessed by urine flow, creatinine clearance and sodium extraction fraction was improved with both drugs.

**Conclusions:** In patients with hyperdynamic septic shock, both NE and TP were effective to raise mean arterial blood pressure. With TP, but not NE, the improvement in blood pressure was achieved at the expense of cardiac index and oxygen consumption which were significantly decreased. Renal function was improved with both drugs. Larger studies should be carried out to further evaluate the impact of these findings on long-term organ dysfunction or survival of septic shock patients.

**A-682 Increasing mean arterial pressure in patients with septic shock: effects on oxygen variables and renal function**
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**Background and Goals:** The effects of increasing mean arterial pressure (MAP) were measured on oxygen variables, and renal function in septic shock.

**Material and Methods:** Twenty-eight patients with septic shock who required fluid resuscitation and pressor agents to increase and maintain MAP > 60 mmHg were included in a prospective, open-label, randomized, controlled study. Patients were treated with fluid and norepinephrine to achieve and maintain a MAP of 65 mmHg. Then, they were randomized in two groups: in the first group (n = 14), MAP was maintained at 65 mmHg, in the second group (n = 14), MAP was increased to 85 mmHg by increasing the dose of norepinephrine. Haemodynamic parameters (MAP, heart rate, mean pulmonary artery pressure, pulmonary artery occlusion pressure, cardiac index, systemic vascular resistance index, pulmonary vascular resistance index, left and right ventricular stroke index), metabolic parameters (oxygen delivery, oxygen consumption, blood lactate, arterial lactate), and renal function parameters (urine flow, serum creatinine, creatinine clearance) were measured.

**Results:** After introduction of norepinephrine, similar values of haemodynamic, metabolic, and renal function parameters were obtained in both groups. No changes were observed in group 1 during the study period. Increasing MAP from 65 to 85 mmHg with norepinephrine in group 2 resulted in a significant increase in cardiac index from 4.8 ± 1.8 to 6.0 ± 1.6 L min⁻¹ m⁻² (P < 0.01), arterial lactate (3.1 ± 1.4 and 2.6 ± 1.6 mmol L⁻¹) and oxygen consumption (247 ± 32 and 228 ± 52 mL min⁻¹ m⁻²) did not change. No changes were observed in renal function parameters: urine flow 61 ± 38 and 73 ± 46 mL, serum creatinine 163 ± 182 and 150 ± 143 µmol L⁻¹, and creatinine clearance 48 ± 39 and 70 ± 68 mL min⁻¹ 1.73 m².

**Conclusions:** Increasing MAP from 65 to 85 mmHg with norepinephrine neither affects metabolic parameters nor improves renal function.

**A-683 A possible role of white adipose tissue in sepsis**
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**Background and Goal of Study:** It has recently been established that white adipose tissue, besides its metabolic role, storing and releasing fatty acids, has a major role in secreting a variety of hormones and mediators which are referred to as adipokines.(1)

In particular, it has been suggested that adipose tissue releases pro-inflammatory cytokines, e.g. IL-6 in response to hypoxia. Thus we felt tempted to investigate how adipokine expression relates to sepsis in an animal model.

**Methods:** After obtaining a Home Office licence under the Animal Rights Act, we investigated sixteen healthy, non-obese male mice (body weight 25–28 g). Eight animals served as controls, in another eight animals, sepsis was induced by intra-peritoneal LPS injection. Twenty-four hours after induction of sepsis, the experimental animals were killed and RNA extracted from epididymal fat was investigated by real time PCR, in order to quantify expression of IL-6, IL-18, nerve growth factor (NGF), hypoxia-induced factor (HIF), adipin and adiponectin in comparison to controls.

**Results:** Twenty-four hours after induction of sepsis, real time PCR revealed significant increases in the expression of IL-6 (500 fold), NGF (10 fold), TNFα (5 fold) and HIF (3 fold), while there were significant decreases in the expression of IL-18 (0.4 fold), adipin (0.15 fold) and adiponectin (0.2 fold).

**Conclusions:** These results show that white adipose tissue is an organ system which actually suffers from hypoxia during sepsis. It responds by a marked increase in the expression of pro-inflammatory cytokines. At the same time, the expression of anti-inflammatory cytokines as well as the expression of adipin and adiponectin is reduced. Thus, we propose the hypothesis that white adipose tissue may be an important contributor to the pathophysiology of sepsis.

Reference:

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**Resuscitation and Emergency Medicine**

**A-684 Austria Nationwide Early Defibrillation Program**
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**Background and Goal of Study:** Significant improvement of survival and neurological outcome after out of hospital cardiac arrest (OHCA) has been reported thorough public access defibrillation (PAD). Therefore a nation wide non-governmental initiative run by the Austrian Red Cross was started in November 2001 to provide public accessible Automated External Defibrillators (AEDs). Supported through a multimedia campaign 1785 AEDs were placed all over Austria until October 2004. Austrian data showed hospital discharge rate after OHCA at 10.6% so far.

**Materials and Methods:** Data about all OHCA incidences in which AEDs were used by bystanders regardless of their profession were recorded according to Utstein Style since December 2002.

**Results and Discussion:** 52 incidences where reported over a period of 30 months. 5 cases were excluded due to the use of the AED by professional first responders. OHCA was confirmed in 44 cases. Shock was indicated in 23 out of these 44 incidences (52.3%) and advised correctly in 22 arrests (95.7%). Correct analysis was impaired by ECG artefacts in one case and lead to false negative decision. In presence of non shockable ECG-rhythms in 18 cases no shock was indicated correctly every time (100%). Six times no cardiac rhythm was recorded. Mean time from emergency call to first AED shock was 4.78 ± 3.08 minutes vs. 9.91 ± 3.65 (p = 0.0002) minutes until EMS arrival, as first time point of possible AED shock by professional helpers, 15 individuals were admitted to an intensive care unit (65.2%). 11 out of the 15 (47.8 % of all patients found with shockable rhythms) primary survivors already left the hospital in good neurological condition.

**Conclusion(s):** A significant reduction of “Call-to-shock-time” was observed resulting in improved survival and neurological outcome. However the number OHCA incidences were an AED was used was low. Further research is necessary to clarify cost-effectiveness.

Reference:

**A-685 Vasopressin + Milrinone for cardiopulmonary resuscitation (CPR) in pigs with myocardial infarction – effect on cardiac index**
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**Background and Goal of Study:** Vasopressin (VP) in contrast to epinephrine (EPI) increases coronary perfusion pressure during and after CPR [1]. Milrinone (MIL) compared to placebo improves cardiac index (CI) after CPR in pigs [2]. Could the combination of VP and MIL, with its absence of beta-adrenaline stimulation, for CPR in pigs with myocardial infarction [3] lead to an increase of CI?

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