Haemodynamic optimization: 0822–0826

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0822

EALTY LACTATE-DIRECTED THERAPY IN CRITICALLY ILL PATIENTS ADMITTED TO THE INTENSIVE CARE: A RANDOMIZED CONTROLLED MULTI-CENTRE TRIAL

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INTRODUCTION. Hyperlactatemia in critically ill patients is associated with increased mortality. However, it is unknown whether the use of lactate clearance as a resuscitation endpoint improves survival.

OBJECTIVE. The primary objective of this multi-centre study was to assess the effect of lactate-directed therapy on hospital mortality, in patients admitted to the ICU with a lactate level of ≥3.0 mmol/l.

METHODS. We randomly allocated patients with hyperlactatemia to either lactate-directed therapy or non-lactate-directed therapy during the first 8 h of ICU stay. Hospital mortality (primary outcome), resuscitation endpoints, administered therapy, organ failure and use of health care resources were compared between the two groups.

RESULTS. In the intention-to-treat population of 348 patients, early lactate-directed therapy did not significantly reduce in-hospital mortality as compared with non-lactate-directed therapy (33.6% vs. 43.5%, p = 0.067). However, when restricted for predefined risk factors, hospital mortality was lower in patients assigned to early lactate-directed therapy (hazard ratio 0.61, 95% CI 0.43 to 0.87, p = 0.000). Additionally, early lactate-directed therapy resulted in a lower SOFA score between 9 and 72 h, earlier discharge from the ICU, earlier weaning from mechanical ventilation and earlier cessation of inotropes.

CONCLUSION. The use of lactate-directed therapy in the initial resuscitation of critically ill patients admitted to the ICU with increased blood lactate levels reduces hospital mortality in an intention-to-treat analysis corrected for predefined risk factors. In addition lactate-directed therapy significantly decreases organ failure and the use of health care resources in these patients.

TRIAL REGISTRATION. ClinicalTrials.gov number NCT00270073.

0823

A RANDOMISED CONTROLLED TRIAL OF THE EFFECTS OF POST-OPERATIVE HAEMODYNAMIC OPTIMISATION ON MICROVASCULAR FLOW, TISSUE OXYGENATION AND INFLAMMATORY MARKERS AFTER MAJOR ABDOMINAL SURGERY

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INTRODUCTION. Perioperative Goal Directed Haemodynamic Therapy (GDT) appears to improve outcome [1]. Importantly, the biological mechanisms underlying these beneficial effects have not been investigated.

OBJECTIVES. To assess the effects of three haemodynamic regimens on tissue microvascular flow and oxygenation in patients after major abdominal surgery.

METHODS. Approval from the local ethics committee and the competent authority were obtained. Patients were enrolled in a 3-arm protocol (DO2I, DO2I+NTG, DO2I+DOPEX) for 48 h immediately after major abdominal surgery. The central venous pressure (CVP) group received intravenous (iv) colloid boluses to achieve a sustained rise in SV plus a fixed rate infusion of dopexamine (0.5 μg/kg/min). The map group received dobutamine (5 μg/kg/min). The DO2I+DOPEX group received iv colloid boluses to achieve a sustained rise in SV. In the DO2I+DOPEX group, patients received iv colloid boluses to achieve a sustained rise in SV plus an increasing infusion rates of nitroglycerin (up to a maximum dose of 133 μg/min). In the high dose of nitroglycerin group, patients received iv colloid boluses to achieve a sustained rise in SV plus a nitroglycerin infusion rate of 133 μg/min. In the low dose of nitroglycerin group, patients received iv colloid boluses to achieve a sustained rise in SV plus a nitroglycerin infusion rate of 66 μg/min. In the CVP group, patients received iv colloid boluses to achieve a sustained rise in SV plus a fixed rate infusion of dopexamine (0.5 μg/kg/min). Data collected included oxygen delivery index (DO2I) (lithium dilution & pulse power analysis), mean arterial pressure (MAP), sublingual microvascular flow video (sidestream darkfield imaging), cutaneous microvascular flow (Schulze dye-chloroethylic acid and serum levels of E-1β, E-2, E-3, I-8, TNF-α and ICAM-1. Data are presented as mean (SD) or median (IQR).

RESULTS. 315 patients were recruited. In all groups, cardiovascular fluid therapy with dopexamine was associated with an increase in DO2I, ScvO2, sublingual and cutaneous microvascular flow and tissue PO2 but no difference in serum inflammatory markers. There were no significant differences in morbidity, mortality or hospital stay (Tables 1, 2, 3).

CONCLUSIONS. Nitroglycerin dose-dependently increased tissue perfusion, measured by ECD, with SDF imaging and AVA 3.0 software. Capillaries were defined as micro-vessels with a diameter of <20 μm. We pooled all nitroglycerin-induced changes in delta-T and delta ScvO2.

Fig 1

0824

INVERSE CORRELATION BETWEEN NITROGLYCERIN-INDUCED CHANGES IN CENTRAL-PERIPHERAL TEMPERATURE GRADIENT AND CHANGES IN SUBLINGUAL PERFUSED CAPILLARY DENSITY IN PATIENTS WITH CARDIACogenic SHOCK OR END-STAGE CHRONIC HEART FAILURE

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OBJECTIVE. To investigate the correlation between nitroglycerin-induced changes in central-peripheral temperature gradient and changes in sublingual perfused capillary density in patients with cardiogenic shock or end-stage chronic heart failure.

METHODS. A nitroglycerin dose-response study was performed in fifteen patients with cardiogenic shock (n = 9) or end-stage chronic heart failure (n = 6) admitted to Erasmus University Medical Center. We did hemodynamic measurements at baseline and during increasing infusion rates of nitroglycerin (up to a maximum dose of 133 μg/min). As parameters of tissue perfusion, we measured central-peripheral temperature gradient (delta-T) and sublingual perfused capillary density (PCD). Sublingual PCD was measured with SDF imaging and AVA 3.0 software. Capillaries were defined as micro-vessels with a diameter of <20 μm.

RESULTS. Nitroglycerin dose-dependently decreased mean arterial pressure (p = 0.01), cardiac filling pressures (central venous pressure: p < 0.001; pulmonary capillary wedge pressure: p = 0.001), and mixed-venous oxygen saturation (p = 0.02). Nitroglycerin decreased delta-T (p < 0.001) and improved sublingual PCD (p < 0.001). Macro-hemodynamic and microcirculatory responses to nitroglycerin infusion were consistent in patients with either cardiogenic shock or end-stage chronic heart failure. A significant correlation was found between pooled changes in both parameters of tissue perfusion (Spearman r = −0.48, p < 0.001; Fig 1).

CONCLUSIONS. The study is continued according to an interim analysis stopping rule of adjusted p < 0.001.

0825

VASOPRESSIN, EPINEPHRINE, AND CORTICOSTEROIDS FOR INHOMOGENEOUS CARDIAC ARREST [NCT00729794]

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INTRODUCTION. The addition of vasopressin during cardiopulmonary resuscitation (CPR) and of steroids during and after CPR may increase the rates of return of spontaneous circulation (ROSC) and improve post-arrest survival [1]. We seek to provide definitive evidence supporting this hypothesis and its generalizability by adequately increasing the size of the originally studied population [1] in the context of a three-center, randomized, controlled trial. Herein, we report the results of the second interim analysis.

METHODS. Adult in-patients with cardiac arrest were randomized to receive either (1) vasopressin (20 IU/CPR cycle for 5 cycles) plus epinephrine (1 mg/CPR cycle) plus methylprednisolone single dose (40 mg on the first CPR cycle) (study group); or (2) placebo plus epinephrine plus placebo (control group). Following return of spontaneous circulation (ROSC), study group patients with postresuscitation shock also received stressed dose hydrocortisone (300 mg/day for 3–7 days and then gradual taper), whereas controls received placebo. Primary endpoints were ROSC for at least 15 min, and survival to discharge either to home or to a rehabilitation facility.

RESULTS. Data from 180 patients were analyzed. Patient clinical profiles were similar; Study group patients vs. controls had higher mean arterial pressure during and after CPR (mean (SD), 75.1 (21.9) vs. 50.4 (13.0) mm Hg and 94.3 (37.2) vs. 70.7 (22.2) mm Hg, p < 0.001), and higher rates of ROSC (71/85 vs. 55/95, p < 0.001) and discharge to either home or a rehabilitation facility (75/85 vs. 80/95, p = 0.009).

CONCLUSION. These results indicate improved study group survival. The study is continued.

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GRANT ACKNOWLEDGEMENT. Supported in part by the Thorax Foundation.

REFERENCE. 1. Mentzelopoulos SD et al. (2009) Arch Intern Med 169:15–24
0826

COMBINATION OF STATIC AND FUNCTIONAL PRELOAD PARAMETERS ENABLES EARLY DETECTION OF RIGHT VENTRICULAR FAILURE

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AIMS. Diagnosis of right ventricular dysfunction (RVD) or failure is often delayed or missed. In case of RVD, optimization of cardiac preload is essential for providing hemo-
dynamic stability. So far, static right ventricular (RV) preload parameters such as central venous pressure (CVP) or RV end-diastolic volume (RVEDV) are used in clinical practice for preload assessment in RVD. As the interdependence of RV and left ventricular (LV) function is supposed to be reflected in the relationship of RV and LV preload parameters, we now investigated in an experimental model whether the combination static RV and functional LV preload parameters can be used for early detection of RVD and for guidance of volume therapy.

METHODS. After approval by the local governmental commission, fifteen anesthetized pigs (29 ± 0.8 kg) were examined. CVP, RV end-diastolic pressure (RVEDP) and mean arterial pressure (MAP) were recorded using micro-tip catheters. Mean pulmonary artery pressure (MPAP) and RVEDP were measured by a thermodilution pulmonary artery catheter (VOLEN, Pulson, Germany). Pulse pressure variation (PPV) and stroke volume variation (SVV) (derived from an ultrasonic flowprobe on the ascending aorta) were calculated over 5 respiratory cycles. Cardiac output (CO) was derived from a pulmonary artery flowprobe. After a baseline measurement, RVD was induced by increasing MAP by 50% by continuous infusion of the thromboxane-analagon U46619. Then, 300 ml blood were extracted (vol extraction), followed by a volume challenge (600 ml vol challenge).

RESULTS. The increase in RV afterload resulted in a reduction of MAP and CO. CVP, SVV and PPV were unchanged, while heart rate (HR), RVEDP and RVEDV remained unchanged. Volume extraction decreased MAP, MPAP and RVEDP without changes in SVV or PPV. During the volume challenge, MAP, MPAP, CPPD and RVEDV increased again, without significant changes in CO, SVV or PPV.

| TABLE 1 | Baseline | RV Vol extraction | Vol challenge |
|---------|----------|------------------|---------------|
| HR (beats/min) | 96 (15) | 98 (16) | 100 (19) | 100 (15) |
| MAP (mmHg) | 25.1 (3.3) | 37.4 (9.4)** | 34.1 (6.4)** | 41.0 (7.8)# |
| CVP (mmHg) | 72.3 (2.8) | 60.1 (11.3#) | 53.2 (10.6#) | 63.1 (12.9#) |
| CO (l/min) | 2.8 (0.6) | 2.3 (0.7)** | 2.1 ± 0.7 | 2.3 ± 0.6 (06) |
| CPPD (mmHg) | 11.3 (4.6) | 12.6 (7.9)* | 10.3 (4.7)* | 14.8 (6.8)* |
| RVEDP (mmHg) | 13.7 (2.2) | 14.9 (6.6) | 11.4 (4.4) | 16.6 (3.0) |
| RVEDV (ml) | 112 (27) | 110 (19) | 108 (23) | 124 (19)# |
| SVV (%) | 11 (3) | 14 (4)* | 14 (5) | 13 (4) |
| PPV (%) | 13 (4) | 17 (6)* | 16 (7)# | 15 (5) |

Mean values (standard deviation) are presented; * p < 0.05 vs. baseline; ** p < 0.001 vs. baseline; ± p < 0.01 vs. RVD; # p < 0.05 vs. vol extraction.

CONCLUSIONS. A simultaneous increase in both static RV (CVP) and functional LV preload parameters suggests RVD. Further diagnosis is warranted. Continuous registration of CVP and SVV or PPV may provide an early indicator of RVD and help to guide volume therapy in RVD.

Acute lung injury: Pathophysiology: 0827–0831

0827

THE SELECTIVE 7nACHR AGONIST GTS-21 ATTENUATES VENTILATOR-INDUCED INFLAMMATION AND LUNG INJURY

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Infectious and inflammatory processes may initiate or perpetuate acute lung injury (ALI). It has been suggested that a pro-inflamatory cytokine milieu, such as the one created by ventilator-induced lung injury (VILI), may be responsible for activating the cholinergic anti-inflammatory pathway (CAP), which is supposed to prevent excessive inflammation caused by ventilator-induced lung injury. To study the effect of a selective 7nACHR agonist on acute lung injury induced by ventilator-induced lung injury.

METHODS. GTS-21 is a selective 7nACHR agonist which is supposed to prevent excessive inflammation caused by ventilator-induced lung injury. The authors compared the effects of GTS-21 on lung inflammation caused by ventilator-induced lung injury. GTS-21 was selected for this study because it is selective for the 7nACHR. The authors used an animal model of ventilator-induced lung injury to study the effects of GTS-21 on lung inflammation caused by ventilator-induced lung injury.

RESULTS. GTS-21 significantly reduced lung inflammation and collagen deposition in an animal model of ventilator-induced lung injury. These effects were dose-dependent and were observed at a dose of 1 mg/kg. The authors also observed that GTS-21 reduced the expression of pro-inflammatory cytokines, such as TNF-α and IL-1β, and reduced the production of reactive oxygen species (ROS) in lung tissue.

CONCLUSIONS. GTS-21 is a selective 7nACHR agonist that reduces lung inflammation and collagen deposition in an animal model of ventilator-induced lung injury. These effects are dose-dependent and are observed at a dose of 1 mg/kg. GTS-21 reduces the expression of pro-inflammatory cytokines and reduces the production of ROS in lung tissue. These findings suggest that GTS-21 may be a potential therapeutic target for the treatment of ventilator-induced lung injury.

0828

PHOSPHOINOSTIDE 3 KINASE BETA ATTENUATES INFLAMMATION AND FIBROSIS IN A BLEOMYCIN MODEL OF ACUTE LUNG INJURY

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Our group has previously demonstrated that phosphoinositide 3 kinase (PI3K) plays a key role in the development of acute lung injury. However, the specific contribution of PI3K isoforms in this process is still not fully understood. In this study, we investigated the role of PI3Kβ in a bleomycin model of acute lung injury.

METHODS. Male C57BL/6 mice were randomly assigned to three groups: Control, Bleomycin, and Bleomycin + GSK-J551. The Bleomycin group received a single intratracheal injection of bleomycin (5 U/kg), while the GSK-J551 group received intratracheal saline injection. Animals were sacrificed at day 14, and lung inflammation and fibrosis were assessed.

RESULTS. GSK-J551 significantly reduced lung inflammation and fibrosis in the bleomycin group. In particular, GSK-J551 reduced the expression of pro-inflammatory cytokines, such as TNF-α and IL-1β, and reduced the production of reactive oxygen species (ROS) in lung tissue. These findings suggest that GSK-J551 may be a potential therapeutic target for the treatment of acute lung injury.

CONCLUSIONS. GSK-J551 is a selective PI3Kβ inhibitor that reduces lung inflammation and fibrosis in a bleomycin model of acute lung injury. These effects are dose-dependent and are observed at a dose of 1 mg/kg. GSK-J551 reduces the expression of pro-inflammatory cytokines and reduces the production of ROS in lung tissue. These findings suggest that GSK-J551 may be a potential therapeutic target for the treatment of acute lung injury.
0830
ROLE OF PTX3 IN LEUKOCYTE RECRUITMENT IN ACUTE PHASE IN A MURINE MODEL OF ACID ASPIRATION LUNG INJURY
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INTRODUCTION. Pentraxins are a superfamily of acute phase proteins. The prototypic long pentraxin 3 (PTX3) is rapidly produced and released by various cell types in response to inflammatory signals. A recent prospective study showed that PTX3 is elevated in ALI and ARDS and that its levels correlate with parameters of lung injury and systemic involvement.

OBJECTIVES. Our aim was to determine the role of PTX3 in inflammation in a murine model of ALI in the acute phase.

METHODS. Mice were injected with 100 µg of human PTX3 i.v. (hPTX3+) or sterile saline (hPTX3−) and ventilated (Vi: 8–10 ml/kg, RR 140 min−1, FiO2: 0.21). In order to induce lung injury, 2 ml/kg of HCl (pH = 1.5) was intratracheally instilled. Mice were ventilated for 10 min, then kept in an oxygenated chamber until full awakening (FiO2: 0.5). To evaluate the severity of injury, animals were sacrificed after 3 h and Broncho-Alveolar Lavage (BAL) was performed and lungs were removed and stored.

RESULTS. 3 h after acid instillation, pre-treatment with hPTX3 allowed a significant reduction of total cell recruitment in the alveolar space; in particular, neutrophilic influx in BAL was significantly reduced compared to mice treated with saline. We observed the same results also in pulmonary tissue.

CONCLUSIONS. Our results indicate that PTX3 plays a role in the acute phase of lung injury induced by acid aspiration.

0831
CRITICAL ROLE OF ADENOSINE RECEPTOR A1 FOR LPS-INDUCED TRAFFICKING OF PMNS IN THE LUNG
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INTRODUCTION. Excessive transmigration of PMNs play a major role in the early development of acute lung injury (ALI). Extracellular adenosine is involved in various inflammatory pathways by signalling through four subtypes of adenosine receptors, A1, A2A, A2B and A3, all members of the family of G protein-coupled receptors. A1 has particularly been implicated in leukocyte recruitment to activated tissues. However, its role in acute pulmonary inflammation remains elusive. We therefore sought to characterize the role of A1 in a model of LPS-induced migration of PMNs into the lung.

MATERIALS AND METHODS. In a murine model of acute lung injury, C57Bl6 and for A1 deficient mice (A1−/−) were exposed to aerosolized LPS. We used a flow cytometry-based method to quantify the accumulation of PMNs in all compartments of the lung. LPS-induced microvascular permeability was determined by the extravasation of Evans blue, and the release of inflammatory cytokines into the bronchoalveolar lavage fluid (BAL) was assessed by ELISA. In addition, we tested the effects of the selective A1 agonist 2′,Me-CCPA in vitro and in vivo. To determine specific effects of A1 on hematopoietic and non-hematopoietic A1-expressing mice were generated by transfer of bone marrow between wildtype and A1−/− mice.

RESULTS. LPS inhalation resulted in significant accumulation of PMNs in all compartments of the lung. In A1−/− mice, PMN recruitment into interstitial and alveolar airspace of A1 gene deficient mice was significantly higher than in wildtype mice. Pharmacological activation of A1 significantly reduced PMN migration to interstitial and alveolar airspace of wildtype but not of A1−/− mice. In A1−/− mice PMN migration into the interstitial was significantly higher than in chimeric mice that expressed A1 only on non-hematopoietic cells. Pharmacological activation of A1 in chimeric mice did only reduce PMN migration when A1 was expressed on hematopoietic cells. LPS-induced increase in Evans blue leakage was significantly higher in A1−/− mice compared with wild-type mice. Consistently, this pharmacological activation of A1 resulted in a significant decrease of microvascular permeability in wildtype mice. Pretreatment with the specific A1 agonist (1 mg/kgG, i.p.) decreased the release of the chemokines MIP-2 and TNFα into the alveolar space of wildtype mice.

CONCLUSION. A1 plays a critical role in LPS-induced PMN transmigration and microvascular permeability in a murine model of ALI. These protective effects appear to be mediated by A1 on hematopoietic cells. Activation of A1 reduces PMN trafficking during pulmonary inflammation and maybe a promising approach to develop innovative therapeutic strategies for the treatment of ALI.
CONCLUSION.

and negative LH, 0.52).

12) into a 66% sensitivity and 66% specificity to detect an intrinsic AKI (positive LH, 1.94; negative LH, 0.52).

INTRODUCTION.

Serum creatinine is a late marker of acute kidney injury (AKI), hindering timely intervention. Plasma and urine NGAL have been shown to be useful early markers for AKI when the timing of the renal insult is known, such as post-cardiac surgery and radiocontrast nephropathy [1]. In children, NGAL both in urine and plasma is an excellent early marker of AKI with an area under the receiver operator characteristic curve (AUCROC) in the range of 0.79–0.9 [2]. However, its performance in a general adult medical-surgical ICU setting has not yet been well described.

OBJECTIVES.

The study aims at evaluating the usefulness of plasma NGAL as an early marker of AKI in an adult general ICU.

METHOD.

This was a prospective cohort study of 307 consecutive incident patients to an adult ICU, enrolled within 24 h of ICU admission. We excluded 5 patients with ESRD, leaving 302 patients for analysis. Clinical data, including urine output and serum creatinine, were collected daily up to ICU discharge. Blood samples for NGAL were collected daily from ICU admission for up to 4 days. Plasma NGAL (pNGAL) was measured in 873 blood samples (median 3 samples/patient), using a point-of-care device (Triage NGAL, Biosite Inc, San Diego, CA).

AKI was defined using the RIFLE (Risk-Injury-Failure-Loss-Endstage renal disease) classification. RIFLEinitial refers to the patient’s RIFLE class on the 1st day of AKI. Diagnostic characteristics of pNGAL were evaluated with receiver operating characteristic (ROC) curves. We defined an event as AKI occurring within 48 h of the first pNGAL measurement.

RESULTS.

Of 302 patients, 133 (44%) developed AKI during their ICU stay. In the AKI group, 90 patients (29.8%) had AKI within 24 h of ICU admission while 43 (14.2%) developed AKI up to 4 days after ICU stay (range, 2nd to 45th ICU day). RIFLEinitial class was Risk in 92 patients (30.5%), Injury in 17 (5.6%) and Failure in 24 (7.9%). Fifteen patients (5%) were treated with renal replacement therapy for AKI in the ICU.

Plasma NGAL was a good diagnostic marker for AKI development within the next 48 h (Fig. 1, area under ROC curve was 0.78, 95% CI 0.65–0.90). Using a cut-off of 150 ng/ml for pNGAL, the sensitivity was 73% and specificity was 81%.

CONCLUSIONS.

Plasma NGAL appears to be a useful early marker for the development of AKI in a heterogenous adult ICU population, in which the timing of the renal insult is largely unknown. Plasma NGAL allows the diagnosis of AKI up to 48 h prior to a rise in serum creatinine. It is worth noting, however, that the area under ROC is slightly lower than that reported in several pediatric studies.

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### Table 1: Functional Properties of Platelets and Leukocytes

| Variable | Group | T0 | T1 | T6 | T24 |
|----------|-------|----|----|----|-----|
| AUC (AU) | COOL 18.5 (17; 22) 18 (15.5; 19) 14 (9.5; 17) 12 (6.5; 18) |
| HEPARIN 11.5 (19; 8.5) 14 (9.5; 18) 13 (9; 16.5; 20.5) 14 (10.5; 16.5) |
| VEL 5.9 (5.4; 6.1) 5.3 (4.8; 5.5) 5.3 (4.5; 5.3) 4.3 (4; 5.2) |
| DNA (%) | COOL 26.6 (21.5; 34) 29.3 (23; 35) 29.3 (23; 35) 18.5 (14; 22) |
| HEPARIN 61.6 (48.8; 64) 44 (4; 51) 42 (3.7; 71.5) 5 (41; 5.6) |
| MA (mm) | COOL 5.6 (15; 25) 5.6 (15; 25) 5.6 (15; 25) 5.6 (15; 25) |
| HEPARIN 67.3 (20.3; 32.3) 39.2 (19; 33.4) 39.2 (19; 33.4) 54 (15; 29) |
| r (min) | COOL 4.9 (1.9) 4.9 (1.9) 4.9 (1.9) 4.9 (1.9) |
| HEPARIN 65.7 (61; 61) 65.7 (61; 61) 65.7 (61; 61) 65.7 (61; 61) |

#### Reference

Krouzecky et al (2009) Intensive Care Med 35:364–370

**Conclusion.** The method of selective in-circuit blood seems to be effective in maintaining
ECC patency for several hours without adversely affecting functional properties of
platelets and leukocytes.

**Grant Acknowledgement.** MSM 020260819 Replacement of and support to
some vital organs.

**Reference.** Krouzecky et al (2009) Intensive Care Med 35:364–370

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### Table 2: TEG Parameters and Platelet and Leukocyte Counts

| TEG Parameters | Platelet and Leukocyte Counts |
|----------------|------------------------------|
| T0 | T1 | T6 | T24 |
| Group F | Group H | Group F | Group H |
| t (min) | 4.9 (1.9) | 4.9 (1.9) | 4.9 (1.9) | 4.9 (1.9) |
| M (min) | 61 (15) | 61 (15) | 61 (15) | 61 (15) |
| Ly (%) | 3.6 (1.2) | 4.4 (1.2) | 4.1 (1.4) | 4.3 (0.9) |
| PIL | 161 (41) | 153 (1) | 141 (0) | 159 (53) | 151 (148) | 134 (158) |

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Severe infections 2: 0842–0846

0842
IMPACT OF ORAL DECONTAMINATION WITH CHLORHEXIDINE 1% GEL ON COLONISATION OF THE RESPIRATORY TRACT IN VENTILATED PATIENTS IN A GENERAL ICU
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INTRODUCTION. Ventilator associated pneumonia (VAP) is common in critically ill patients, and is associated with increased mortality. Aspiration of bacteria from the oropharynx is thought to be important in the pathogenesis of the condition. Consequently, selective digestive tract decontamination has been used to reduce the oral bacterial load, in an attempt to reduce the incidence of VAP, although the efficacy of this remains unclear [1,2]. Concerns regarding increased selection of antibiotic-resistant bacteria have resulted in a search for alternative strategies to reduce airway contamination. Topical application of the antiseptic chlorhexidine gluconate (CHX) has achieved varying results, however, the concentration and formulation has varied between studies. In cardiac surgical patients at low risk of VAP, CHX 0.12% was effective at reducing VAP [3]. In medical and mixed ICU populations, CHX 0.12% was found to be ineffective in most trials although CHX 2% did show a reduction in VAP [4]. CHX 2% is not available in the UK. CHX 1% gel is used by our oral surgery department, so we introduced this to our ICU and audited the results.

OBJECTIVES. To assess the impact of routine oral CHX 1% gel application on culture rates from bronchoalveolar lavage (BAL) and protected catheter (PC) screening performed on ventilated patients in a 14-bed medical and surgical ICU in a University Hospital.

METHODS. Permission was obtained from our clinical audit department. Data were collected retrospectively for all BAL and PC samples for two consecutive 6-month periods: before and after the implementation of routine CHX oral decontamination (in January 2008). PC samples were collected routinely for screening on a twice weekly basis, and BAL was performed when clinically indicated. PC samples were considered positive if >10⁴ colony forming units (CFU) were present; BAL if >10⁴ CFU were present. Where a sample grew multiple organisms, this was considered as a single positive result.

RESULTS. Prior to CHX use, 154 samples were collected. Of these 24 (15.6%, 3 BAL, 21 PC) were positive. Following commencement of routine CHX use, 225 samples (71 BAL, 154 PC) were collected. Of these, 19 (8.4%, 4 BAL, 15 PC) were positive. The reduction in positive results was statistically significant (2-sided Fisher’s exact test, p = 0.047). CHX appeared to exhibit greatest impact on gram-negative organisms (23 positive cultures pre-CHX, 13 post-CHX), whilst having the opposite effect upon fungi (3 positive cultures pre-CHX, 6 post-CHX).

CONCLUSIONS. In this small retrospective study, a reduction in the rate of microbiological contamination of the lower respiratory tract was observed following the introduction of routine oral decontamination with CHX 1% gel in invasively ventilated patients. No adverse incidents involving CHX were reported during the study period.

REFERENCES. 1. de Smet NEJM Jan 09 2. Chan BMJ 2007 3. Deriso AJ Chest 1996 4. Koeman Am J Respir Crit Care Med 2006

GRANTS. None.

0843
ANTIBIOTIC PRESCRIBING HABITS IN A TERTIARY REFERRAL INTENSIVE CARE UNIT
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INTRODUCTION. Antibiotic prescribing habits in 30 consecutive patients admitted to a tertiary referral mixed medical-surgical adult ICU were assessed. The aim of the study was to assess the impact of routine oral CHX 1% gel application on culture rates from bronchoalveolar lavage (BAL) and protected catheter (PC) screening performed on ventilated patients in a 14-bed medical and surgical ICU in a University Hospital.

METHODS. Permission was obtained from our clinical audit department. Data were collected retrospectively for all BAL and PC samples for two consecutive 6-month periods: before and after the implementation of routine CHX oral decontamination (in January 2008). PC samples were collected routinely for screening on a twice weekly basis, and BAL was performed when clinically indicated. PC samples were considered positive if >10⁴ colony forming units (CFU) were present; BAL if >10⁴ CFU were present. Where a sample grew multiple organisms, this was considered as a single positive result.

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CONCLUSIONS. In this small retrospective study, a reduction in the rate of microbiological contamination of the lower respiratory tract was observed following the introduction of routine oral decontamination with CHX 1% gel in invasively ventilated patients. No adverse incidents involving CHX were reported during the study period.

REFERENCES. 1. de Smet NEJM Jan 09 2. Chan BMJ 2007 3. Deriso AJ Chest 1996 4. Koeman Am J Respir Crit Care Med 2006

GRANTS. None.

0844
REDUCING METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS (MRSA) TRANSMISSION AND BACTERIAEMIA IN INTENSIVE CARE: IMPACT OF UNSELECTIVE DECOLONISATION IN ADDITION TO STANDARD MEASURES
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INTRODUCTION. Methicillin-resistant Staphylococcus aureus (MRSA) is a worldwide cause of morbidity and mortality in the intensive care unit (ICU) [1]. Current recommendations include active surveillance, improved antibiotic stewardship and the general principles of infection control [2]. It has also been suggested that unselective decontamination may be effective for the control of MRSA propagation when other measures have failed [3]. We present our experience of reducing MRSA bacteriaemia in the ICU by a series of measures, including unselective decontamination of all patients admitted to the unit.

OBJECTIVES. To examine the effectiveness of a series of interventions to reduce MRSA transmission and bacteriaemia in the ICU.

METHODS. In April 2005, detailed data collection began for all cases of MRSA in our institution, a mixed medical and surgical ICU. Between April and December 2005, MRSA control measures included: active surveillance with isolation of carriers; reduced cephalosporin and fluoroquinolone usage; improved environmental cleaning; hand hygiene and infection control training for staff; chlorhexidine 4% wash for carers and contacts. In February 2006, unselective decolonisation of all patients was begun. All ICU admissions received chlorhexidine 4% wash, once daily for 5 days and mupirocin 2% nasal ointment, three times daily for 5 days.

RESULTS. During the first 12 months studied (April 2005-March 2006 inclusive), 51 patients were admitted to ICU colonised with MRSA. 31 patients became MRSA colonised in ICU and there were 15 cases of MRSA bacteriaemia in ICU. During the last 12 months studied (January 2006 to December 2006 inclusive), 48 patients were admitted to ICU colonised with MRSA (an increase of 45%), 3 patients became MRSA colonised in ICU (a reduction of 90%) and there was 1 case of MRSA bacteriaemia (a reduction of 93%). Mupirocin resistance was not detected in any samples from ICU during the period studied.

CONCLUSIONS. The measures taken were effective in reducing MRSA transmission by 90% and in reducing MRSA bacteriaemia. This becomes evident by a reduction in the number of patients admitted to the ICU colonised with MRSA. Unselective decontamination with 4% chlorhexidine washes and nasal mupirocin appears to have contributed significantly to this effect.

REFERENCES. 1. Eur J Clin Microbiol Infect Dis (2008) 27:409–413 2. J Hosp Infect (2006) 63:51–54 3. J Infect Chemother (2005) 11:231–233

GRANT ACKNOWLEDGEMENT. None.

0845
IDENTIFYING SEPSIS IN TRIAGE: ASSESSMENT OF A NURSE TRIAGE TOOL
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BACKGROUND. International guidelines highlight the necessity of early identification of severe sepsis. Triage procedures in the emergency department (ED) represent the first step in the sepsis chain of survival.

OBJECTIVES. This study assessed a tool intended for the triage nurse to correctly identify sepsis or impending severe sepsis.

METHODS. A prospective study was done in three ED, during 2 months. A specific sepsis triage aid was used for the identification of a possible sepsis syndrome and for risk factors of impending or obvious severity. The sepsis syndromes were ultimately confirmed or classified by the emergency physician. Final diagnosis was compared with the initial triage.

RESULTS. Among the 253 patients eventually diagnosed with sepsis, 205 were correctly identified by the triage nurse. The calculated triage sensitivity was 81% [95% CI: 76–85] for identification of sepsis and 75% [95% CI: 64–85] for correct prioritization of severe sepsis.

CONCLUSIONS. Some patients did not present initially with clinical criteria of sepsis and deteriorated during their stay in the ED. Most of non-suspected patients with sepsis had not been correctly screened. Furthermore time necessary to apply the screening procedure often seemed too long in the setting of triage.

CONCLUSION. A simple triage aid for sepsis seems promising. Such a tool must use simple but relevant criteria and consider the rapid change in clinical state in potentially septic patients.
**FLUID LEAKAGE PAST TRACHEAL TUBE CUFFS: EFFECT OF SUCTIONING MANOEUVRE AND TYPE OF CUFF IN A BENCHTOP MODEL**

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INTRODUCTION. The leakage of oropharyngeal secretions around tracheal tube cuffs is used as a major risk factor for bacterial tracheal colonization. Tracheal suctioning enhances leakage, by decreasing tracheal pressure. This study aimed to evaluate the effect of a suctioning manoeuvre without disconnection of the ventilator on leakage, according to the modalities of suctioning and the type of cuff.

METHODS. A benchtop model of trachea was created using a plastic tubing whose diameter was in the range of an adult trachea (antero-posterior ID 24 mm, transversal ID 20 mm). The trachea was connected with a suction device that the adrenocortical end was connected to a mechanical ventilator—Servo i (Siemens); the distal end of the trachea was connected to a test lung (Siemens). The ventilator settings were as follows: volume-controlled mode, respiratory rate 12 bpm, peak inspiratory pressure at 25 cm H2O, PEEP at 5 cm H2O. The cuff pressure was set at 30 cm H2O. Then, 0.5 ml blue dye followed by 3 ml saline was instilled just above the cuff. Three types of tubes were tested: Hi-Lo Evac (Mallinckrodt) with polyvinyl chloride cuff, Microcuff (Kimberly-Clark) and Sealgard (Mallinckrodt) with polyurethane cuffs. For each type of tube, leakage of dye was evaluated with each of three sizes of suction catheter (12-14-16 French) and three levels of suction pressure (−100, −200, −400 cm H2O). Suctioning was performed hourly along 8 h. Each experiment was performed five times.

RESULTS. Once the cuffs were inflated to a pressure of 30 cm H2O in the model of trachea, longitudinal folds were observed within the cuff wall of Hi-Lo Evac and Sealgard tubes, but not for Microcuff. During baseline ventilation with PEEP without suctioning, no leakage was observed along 8 h with the three types of tube. The incidence of leakage during suctioning is detailed in Table 1.

### Table 1

| Tracheal tube | Negative pressure (cm H2O) | Suction catheter size (French) | Leakage |
|---------------|---------------------------|-------------------------------|---------|
| Hi-Lo Evac    | 100                        | 5/5/5-0/5                    | −1/5−0/5−0/5 |
|               | 200                        | 5/5/5-0/5                    | 0/5−0/5−0/5 |
|               | 400                        | 5/5/5-0/5                    | 0/5−0/5−0/5 |
| Sealgard      | 100                        | 1/5/0-0/5                    | −1/5−0/5−0/5 |
|               | 200                        | 1/5/0-0/5                    | 0/5−0/5−0/5 |
| Microcuff     | 100                        | 1/5/0-0/5                    | −1/5−0/5−0/5 |
|               | 200                        | 1/5/0-0/5                    | 0/5−0/5−0/5 |
|               | 400                        | 1/5/0-0/5                    | 0/5−0/5−0/5 |

CONCLUSION. In this model, no leakage of fluid was observed past the three types of tube tested during 8 h of volume-controlled ventilation with a PEEP level at 5 cm H2O. During suctioning, when using low levels of negative pressure, no leakage was observed past the three tubes tested. When using high levels of negative pressure, the Microcuff was the sole tube allowing the use of large size catheters without leakage.

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**FLUID LEAKAGE PAST TRACHEAL TUBE CUFFS: EFFECT OF SUCTIONING MANOEUVRE AND TYPE OF CUFF IN A BENCHTOP MODEL**

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METHODS. A benchtop model of trachea was created using a plastic tubing whose diameter was in the range of an adult trachea (antero-posterior ID 24 mm, transversal ID 20 mm). The trachea was connected with a suction device that the adrenocortical end was connected to a mechanical ventilator—Servo i (Siemens); the distal end of the trachea was connected to a test lung (Siemens). The ventilator settings were as follows: volume-controlled mode, respiratory rate 12 bpm, peak inspiratory pressure at 25 cm H2O, PEEP at 5 cm H2O. The cuff pressure was set at 30 cm H2O. Then, 0.5 ml blue dye followed by 3 ml saline was instilled just above the cuff. Three types of tubes were tested: Hi-Lo Evac (Mallinckrodt) with polyvinyl chloride cuff, Microcuff (Kimberly-Clark) and Sealgard (Mallinckrodt) with polyurethane cuffs. For each type of tube, leakage of dye was evaluated with each of three sizes of suction catheter (12-14-16 French) and three levels of suction pressure (−100, −200, −400 cm H2O). Suctioning was performed hourly along 8 h. Each experiment was performed five times.

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| Sealgard      | 100                        | 1/5/0-0/5                    | −1/5−0/5−0/5 |
|               | 200                        | 1/5/0-0/5                    | 0/5−0/5−0/5 |
| Microcuff     | 100                        | 1/5/0-0/5                    | −1/5−0/5−0/5 |
|               | 200                        | 1/5/0-0/5                    | 0/5−0/5−0/5 |
|               | 400                        | 1/5/0-0/5                    | 0/5−0/5−0/5 |

CONCLUSION. In this model, no leakage of fluid was observed past the three types of tube tested during 8 h of volume-controlled ventilation with a PEEP level at 5 cm H2O. During suctioning, when using low levels of negative pressure, no leakage was observed past the three tubes tested. When using high levels of negative pressure, the Microcuff was the sole tube allowing the use of large size catheters without leakage.
OBJECTIVES. Diabetes of injury with increased serum glucose levels has been associated with higher mortality and poor outcome in severely injured children. Peak blood glucose (BG), duration and intensity of hyperglycemia are independently associated with mortality in PICU (Srinivasan 2004). Strict glucose control by intensive insulin therapy is beneficial. However, higher frequency of severe hypoglycemia and positive metabolic effects of glucose have to be mentioned. The aim of this study is to compare applicability of glucose parameters and glucose variability in pediatric trauma patients and establish target BG in severely injured children.

METHODS. prospective clinical study in 2008. 51 children, non diabetic, with Injury Severity Score more than 15 and Pediatric Trauma Score less than 9 points were included. Average age was 9.8 years (6.9–11.9, CI 95%). The mortality showed 13.7%. BG was measured every 3 h by standard biochemical laboratory tests and compared with glucose levels by daily calibrated glucometer. Pediatric scoring systems, predict death rate (PDR) and parameters of hospitalization were collected: Peak BG and glucose intensity were used glucose parameters. The hyperglycemic index, hypo/hyperglycemia ratio and hyperglycemic parameters of hospitalization were collected. Peak BG and glucose intensity were used glucose parameters. The hyperglycemic index, hypo/hyperglycemia ratio and hyperglycemic difference described glucose variability. The nonparametric statistic methods (Spearman correlation and Mann Whitney U test) were used for analysis.

RESULTS. Hyperglycemia over 110 mg/dl [6.1 mmol/l] was in 87.3% patients. Both calculated glucose parameter did not show significant difference between survivals and nonsurvivals. All parameters of glucose variability were significantly higher in nonsurvivals (p < 0.01). Positive statistical significant correlation between peak BG and PDR3 (Spearman coefficient r = 0.661, p < 0.01). Target BG in pediatric trauma patients was established from peak BG and PDR3 correlation by using a linear regression analysis. 137 mg/l [7.6 mmol/l] was calculated as optimal BG for risk of mortality lower than 15%.

CONCLUSION. Hyperglycemia is very common in injured children. Glucose variability better reflects the outcome and prognosis in pediatric trauma patients. Higher difference of glucose variability has been associated with higher mortality and severe organ failure. Calculated target BG is higher than normal glucose level thus could reflect positive metabolic effect of glucose in injured children and minimize severe hypoglycemic episodes.

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OBJECTIVE. Respiratory syncytial virus (RSV) has been found to be the most common viral cause of death in children less than 1 year old. The purpose of this study was to determine the mortality rate and the risk factors for death in children with severe RSV infection.

METHODS. Retrospective review of the medical records of all children managed in a tertiary pediatric intensive care unit (PICU) for severe RSV infection over a period of 9 years (2000-2008). Need for mechanical ventilation (MV) or non invasive ventilatory support was used as a marker of severity. To determine risk factors for death, a univariate then a multivariate analysis using a logistic regression model were performed.

RESULTS. During the study period, 142 patients (median age: 1, 4 months; interquartile range (IQR): 0, 85–2, 4 months) had a severe RSV infection accounting for 3, 8% of total admissions in the PICU and 84% of children managed for RSV infection. Of them 89, 5% required MV and 10, 5% required non invasive respiratory support. Twelve children died (median age: 2 months [IQR 0, 9–5, 1], median length of PICU stay: 17 days [IQR 8–34, 7]) and 130 survived (median age: 1, 4 months [IQR 0, 8–2, 4], median length of PICU stay: 9 days [IQR 5–16]). The overall PICU RSV mortality was 8, 4%. Death was caused by refractory hypoxemia in 4 cases and refractory septic shock in 8 cases. All of the RSV deaths had pre-existing or underlying medical conditions (prematurity: 5 cases, antecedent of neonatal respiratory distress: 5 cases requiring MV in 4 cases, chronic lung disease: 3 cases, cardiac lesions: 3 cases, neuromuscular disease: 1 case) and 7 of them (58, 3%) were hypotrophic. Risk factors for death in univariate analysis were: antecedent of neonatal respiratory distress (41, 7% vs 12, 3%; p = 0, 017), antecedent of MV during the neonatal period (33, 3% vs 7, 6%; p = 0, 001), occurrence of pulmonary air leak (33, 3% vs 3, 8%; p = 0, 003), occurrence of nosocomial infection (66, 7% vs 20%; p = 0, 001), prolonged MV (19, 9 ± 14, 6 days vs 8, 6 ± 6, 9 days; p = 0, 000) and prolonged length of stay (21 ± 15, 8 vs 11, 6 ± 11, 5 days; p = 0, 009). Independent risk factors for death were: antecedent of MV during the neonatal period (OR = 16, 9; 95% CI [2, 7–104]); prolonged MV in the PICU (OR = 12, 1; 95% CI [1, 6–87]) and the occurrence of pulmonary air leak (OR = 9, 9; 95% CI [1, 5–52]).

CONCLUSIONS. The mortality rate of severe RSV infection was 8, 4% in our PICU. Antecedent of MV during the neonatal period; prolonged MV in the PICU and the occurrence of pulmonary air leak are associated with a significantly higher risk of death from severe RSV infection.