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AKI / ARF / SIS - Miscellanea of mechanical ventilation, AKI and sepsis

001611

The outcome of Acute kidney injury in the intensive care unit of A sub Saharan Tertiary Hospital

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INTRODUCTION. Acute kidney injury is characterized by sustained rise in serum creatinine and reduction in urine output. It may also be accompanied by retention of nitrogen products and electrolyte disturbances. The incidence of AKI varies between 36 and 67% among critically ill patients with a mortality rate of 50 to 70%.
OBJECTIVES. We determined the incidence and outcome of acute kidney injury in critical care patients
METHODS. A total of 177 patients, 18 years and older were studied. Data were collected on admission and daily during hospitalization until discharge or death. AKI was defined as:1-2 1) absolute increase in serum creatinine ≥0.3 mg/dL or ≥1.5 times the baseline level, or 2) requirement for renal replacement therapy, or 3) oliguria defined as urine output <400 ml in 24 hours
RESULTS. AKI was observed in 34.3% of our ICU admission, among of whom 4.7% developed AKI during their ICU stay. The mean duration of onset of AKI was 1 (25th to 75th percentile 1-2) days. The overall ICU 30 days mortality was 42.4%, however the 30 days mortality in patients with AKI was 85.5%. Renal replacement therapy was only possible in 36.6% patients. Inotropic support was administered in 59.1% patients with AKI. Factors mitigating against dialysis included proctated hypotension in 63.6%, lack of fund in 18.1%, delayed screening for HIV and Hepatitis B in 18.3%.
CONCLUSION. Acute kidney injury is a common problem in the critically ill patient and is associated with a high mortality rate at our institution.

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000742

Acute kidney failure in the post operatory of peripheral vascular surgery, a prospective single- center experience

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INTRODUCTION. Acute kidney failure (AKF) is identified in 30-40% of cases in post-operative patients. There is limited literature on the importance and the correlation between peripherical vascular surgeries and the development of AKF.
OBJECTIVES. Analysis of the connection between factors related to AKF or acute-on-chronic-kidney failure (AOCKF) in the ICU during the first 7 days after peripherical vascular surgeries, for example: arterial bypasses, amputations, angioplasties and embolectomies.
METHODS. The definition of AKF is defined by the AKIN: abrupt increase in serum creatinine ≥ 0.3 mg/dL or 50% from the baseline. This study is prospective, observational and non-randomized. Patients on previous dialysis were excluded. Continuous variables were summarized as medians and ranges, and categorical variables as percentages.
RESULTS. A database of 65 patients was evaluated. The data was acquired between April 2018 and March 2019. The median age was 70 (range 22-88 y); 73% were male. 25 patients (38%) had AKF.
The average age in both groups is similar, as well the prevalence of comorbidities (hypertension and DM), distribution of sexes and mortality. 46% of the patients with chronic-kidney disease developed an AOCKF (28% of the AKF group). The frequencies of the following values are bigger in AKF group: Re-surgery, emergency surgery, use of vancomycin, gentamicin or amikacin.
CPK values were different in the two groups (4332 AKF; 2937 no AKF), but our sample was inconclusive to demonstrate a real correlation with AKF. When CPK in the first 24h was divided in three categories (<10.000; 10000-20000 and >20000), it was observed in a Kaplan Meier analyses a correlation between these categories and post-operative hemodialysis.
CONCLUSION. Despite our small sample, CPK when analyzed as a categorical variable, showed a statistical significance in patients submitted to hemodialysis. Despite differences in both groups, as CPK average as well as further factors related to a more serious condition; like patient urgency surgery, re-surgery and the use of antibiotics; our analysis was inconclusive to stablish those factors as predictive

0.857 (p=0.042), and the optimal cutoff point for D-dimer was 19.8 µg/dL because at that level, sensitivity (80%) and specificity (86%) were well balanced.
CONCLUSION. The plasma D-dimer levels may be used to screen for VTE in spinal cord injury patients who require long-term hospitalization and rest. The optimal measurement timing is 13 days after injury, and optimal threshold level is 19.8 µg/dL.

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1. None

001023

Development of an ovine model of haemorrhagic shock: Characterisation of systemic and local oxygen supply/demand imbalance

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INTRODUCTION. The global burden of haemorrhagic shock (HS) is substantial and mostly caused by severe physical trauma. Essential pathophysiologic features of HS are imbalance between oxygen delivery and demand, hyper or hypocoagulatory states and compensatory anti-inflammatory response syndrome, ultimately resulting in multi-organ failure (1). Animal models of HS are essential to study underlying pathobiology and develop new treatments.
OBJECTIVES. To develop an ovine model of severe HS caused by blood loss > 30% of the estimated blood volume (EBV) and study dynamics of oxygen supply/demand imbalance systemically and at end-organ tissue level.
METHODS. Six adult female Leicester cross breed sheep (46±5 Kg) were anaesthetised, intubated and on mechanical ventilation. We cannulated the femoral artery for blood sampling and mean arterial pressure (MAP) monitoring. Swan-Ganz catheter was inserted through the right jugular vein. Animals were haemorrhaged through consecutive collections of 300 mL of blood over 90 min, up to approximately 30% of the EBV. HS was halted in case of MAP < 50 mmHg, heart rate (HR) >200 beats/min, or venous oxygen saturation (SvO2) <50%. At baseline, and every 15-min thereafter, haemodynamic parameters were recorded, and arterial blood gas analysis performed, oxygen delivery (D02) computed. Microdialysis probes were positioned into various organs to measure, at baseline and end of bleeding period, interstitial lactate and lactate/pyruvate ratio.
RESULTS. All sheep survived HS. On average, sheep were haemorrhaged 1055±193 mL of blood (33.6±5.2% of EBV). At baseline, and at the end of HS period, Hb varied from 10.3±2.1 to 7.6±10.8 g/dL (p<0.01), arterial lactate from 2.0±1.5 to 4.3±1.2 mmol/L (p<0.01), HR from 102±14 to 154±30 bpm (p=0.06), MAP from 95.6±8.8 to 51.3±15.2 mmHg (p<0.01), cardiac output from 4.4±1.4 to 2.1±0.8 L/min (p<0.01), SvO2 from 76.3±9.8 to 54.0± 24.3% (p=0.192) and D02 from 552±218 to 241±114 ml/min (p< 0.01). Table below depicts development of organ-specific oxygen debt, measured by microdialysis.
CONCLUSION. We described an ovine model of HS, characterized by significant impairment in cardiac output and oxygen delivery, resulting in multi-organ oxygen supply/demand imbalance. The model will be first used to compare the efficacy of different resuscitation fluids.

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Table 1 (abstract 001023). See text for description

	Brain	Renal cortex	Liver	Skeletal muscle
Lactate (mmol)				
Baseline	3.3±1.3	0.9±0.6	2.4±1.4	3.4±0.9
End of haemorrhage	5.0±1.7	11.8±16.9	5.4±1.3	7.6±2.5
(P-value)	(0.25)	(0.05)	(0.06)	(0.04)
Lactate/Piruvate Ratio (%)				
Baseline	50.6±62.1	24.9±12.1	87.0±49.4	43.2±14.1
End of haemorrhage	60.8±52.2	53.6±30.6	76.7±22.9	145±116
(P-value)	(1.00)	(0.05)	(0.31)	(0.18)

001119

Inhaled Argon improves neurological outcome in experimental traumatic brain injury

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INTRODUCTION. While supportive treatment in the management of Traumatic Brain Injury (TBI) has progressed over the past 20 years, specific drug treatments are lacking [Maas *et al*]. New strategies are needed. Altering how patients are ventilated, could be an easily modifiable variable in TBI management. Data in *in vitro* and *in vivo* models of ischemic heart [Ristagno *et al*] and brain injury [Loetscher *et al*] show that the gaseous agent Argon is endowed with neuroprotective potential. Whether Inhaled Argon (iAr) is protective in experimental TBI is presently unknown.
OBJECTIVES. To test the effects of inhaled Argon administered after experimental TBI in mice on neurological functions and structural outcome by longitudinal behavioural assessments and magnetic resonance imaging (MRI) including T2W and DWI sequences.
METHODS. Severe TBI was performed in anesthetized mice (C57BL/6J, 8 weeks old, male) over the left parietotemporal cortex by controlled cortical impact as previously described [Zanier *et al*]. Immediately after TBI, mice were randomized to 24h treatment by iAr 70%-O2 30% (n=10) or air (n=10) from 10 minutes after TBI. Sensorimotor deficits were evaluated at the end of treatment (24h post TBI) and at 1 week by neuroscore and simple neuroassessment of asymmetric impairment (SNAP) tests. MRI (7 Tesla, Bruker) was performed at 3 days post TBI to evaluate contusion volume by T2W. The effect of iAr on acute brain edema, was analysed in a subset of mice (n=3 per group) by DWI-MRI. Maps of the apparent diffusion coefficient generated by DWI-MRI were used to evaluate iper/ipo intense regions as a proxy of vasogenic and cytotoxic edema, respectively. A simple random allocation was applied to assign a subject to an experimental group. Data acquisition and analysis were done blindly. Data were

5.75), lower PaCO2 (OR, 0.90; 95% CI 0.83–0.98) and higher PR (OR, 1.03; 95% CI 1.01–1.05) are associated with progression of respiratory acidosis after NMBA infusion.
CONCLUSION. Emphysema, higher pH, lower PaCO2 and higher PR are risk factors for post-NMBA respiratory acidosis. It suggested patients with respiratory failure who showed active cardiopulmonary compensation especially in those with emphysema might have a risk for a progression of respiratory acidosis after muscle paralysis.

000931

Residual alveolar overdistension and collapse at electrical impedance tomography-guided positive end-expiratory pressure in patients with and without ARDS

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INTRODUCTION. Electrical impedance tomography (EIT) can assess alveolar overdistension and collapse during a decremental positive end-expiratory pressure (PEEP) trial in mechanically ventilated patients [1]. Using this approach, it is possible to identify the "best compromise" PEEP at which overdistension and collapse are minimized as shown in patients with acute respiratory distress syndrome (ARDS) [2] and in patients during general surgery [3]. However, even with the "best compromise" PEEP, a variable amount of simultaneous overdistension and collapse remains. We hypothesized that the sum of overdistension and collapse detected by EIT at the "best compromise" PEEP is higher in patients with ARDS than in lung-healthy control patients during general surgery and might be a marker of disease severity.
OBJECTIVES. To investigate whether the sum of residual overdistension and collapse at the "best compromise" PEEP is higher in patients with ARDS than in patients without pulmonary pathology.
METHODS. We compared EIT data sets of 12 patients with ARDS (5 female, age: 67±11 years, height: 174±9 cm, weight: 79±22 kg, PaO2/FiO2 at baseline: 151±38 mmHg) to 8 patients without pulmonary pathology (all female, age: 38±19 years, height: 169±5 cm, weight: 77±21 kg) undergoing general anesthesia for laparoscopic gynecological surgery. Decremental PEEP trials were carried out in all patients during pressure-controlled ventilation with constant driving pressure. In control patients, one PEEP trial was performed after induction of general anesthesia in the supine position and another one during capnoperitoneum in the Trendelenburg position. All PEEP trials were analyzed as described in [1] and "best compromise" PEEP was defined as the value associated with equally balanced overdistension and alveolar collapse. Residual overdistension and collapse was calculated as the sum of overdistension and collapse at the "best compromise PEEP". Data sets were compared by one-way analysis of variance with Bonferroni post test. Written informed consent was obtained from control patients and from the legal representatives of ARDS patients. Numerical values are presented as mean±SD.
RESULTS. In ARDS patients, we identified 14.5±7.1% of residual overdistension and collapse at the "best compromise" PEEP of 13.3±2.4 mbar. In control patients, the sum of residual overdistension and collapse was 6.3±3.8% at the "best compromise" PEEP of 7.3±2.4 mbar in supine position and 3.4±3.5% at 18.0±2.1 mbar in the Trendelenburg position with capnoperitoneum. The difference between ARDS and lung-healthy patients was statistically significant both for supine ($p<0.01$) and Trendelenburg positions ($p<0.001$).
CONCLUSION. Our preliminary results imply that the sum of residual alveolar overdistension and collapse at the "best compromise" PEEP level is higher in patients with ARDS than in control patients both during ventilation in the supine and in the Trendelenburg position with capnoperitoneum. This supports the hypothesis that this measure might serve as a marker of disease severity in mechanically ventilated patients.

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000933

Development and characterisation of novel animal models of Acute Respiratory Distress Syndrome (ARDS) endotypes

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INTRODUCTION. Acute Respiratory Distress Syndrome (ARDS) still remains a serious pulmonary condition with substantial mortality and without specific therapeutic options. Retrospective analysis of randomized controlled trials have identified the presence of two distinct phenotypes, based on clinical and biological variables (1–3), namely a hypo-inflammatory (P1) and a hyper-inflammatory (P2) endotype. The development of clinically relevant models to replicate these endotypes, will likely enable better understanding of these endotypes.
OBJECTIVES. We aimed to develop an ovine model of P1 and P2 ARDS endotypes and to characterize clinical and biological features, opening the pathway for further prospective evaluation of endotype-specific treatment options.
METHODS. We studied 20 anesthetized sheep on mechanical ventilation up to 6 hours, and randomized into three distinct groups: 1) the oleic acid (OA) only group received an intravenous infusion of OA to achieve a PaO2/FiO2 ratio <100 mmHg (n=8); 2) the OA-IV-LPS group received an OA infusion and intravenous infusion of lipopolysaccharide (n=5) and; 3) the OA-IP-LPS group received an OA infusion and intrapulmonary infusion of LPS (n=7). Pulmonary, hemodynamic and laboratory parameters were assessed hourly. In addition, interleukin (IL) -6, -8 and -10 were quantified in serum and bronchoalveolar lavage (BAL).
RESULTS. Severe impairment in PaO2/FiO2 ratio was found in all study groups (Figure 1). We found a) more hemodynamic and pulmonary impairment b) decreased albumin and platelets levels and c) upregulation of inflammatory (IL-6 and IL-8) and anti-inflammatory cytokines (IL-10) in serum and BAL only in OA-IV-LPS in comparison with the other two groups.
Therefore, the OA-IV-LPS model may mimic a pattern closest to the human P2 endotype and the OA only model the P1 endotype.
CONCLUSION. We developed innovative models of ARDS that mimic clinical and inflammatory status of human P1-P2 endotypes, while characterized by similar severity of pulmonary dysfunction. These animal models could be applied to explore in detail mechanical and biological differences between ARDS subphenotypes and identify endotype-specific treatment options.

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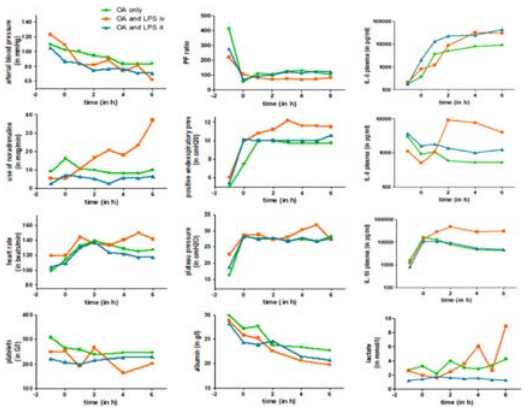

Fig. 1 (abstract 000933). See text for description

admission might suggest a specific role for injurious spontaneous breathing. Early identification of patients at risk for late intubation might prompt prophylactic interventions.

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Table 1 (abstract 000946). See text for description

	Sepsis/septic shock at the admission	No intubation	Early intubation	Late intubation	P value
Organ failure at admission, N (%)					
0	53 (3%)	29 (13%)	19 (2%)	5 (1%)	<0.0001
1	235 (16%)	77 (37%)	110 (12%)	48 (14%)	
2	314 (22%)	51 (24%)	199 (22%)	64 (18%)	
>2	823 (57%)	51 (24%)	550 (62%)	222 (65%)	
GCS, median [IQR]	15 [10-15]	15 [15-15]	14 [9-15]*	14 [10-15]*	<0.0001
SAPS II, median [IQR]	48 [36-63]	35 [25-47]	50 [37-65]*	52,5 [40-67]*	<0.0001
SOFA, median [IQR]	8 [5-12]	6 [3-8]	9 [6-12]*	10 [6-13]*	<0.0001

*p<0,05 vs No intubation group

000946

Early factors associated with increased risk for intubation in spontaneously breathing patients with sepsis and septic shock

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INTRODUCTION. Systemic inflammation and metabolic acidosis, which characterize patients with sepsis and septic shock, can increase the spontaneous respiratory drive and effort and the risk of patient self-inflicted lung injury, with potentially detrimental effects on outcome[1]. A retrospective study reported that septic shock patients intubated during their intensive care unit (ICU) stay had fewer 28-day alive without organ support, as compared to patients already intubated upon admission[2].

OBJECTIVES. To compare ICU mortality in a large cohort of patients with sepsis/septic shock classified as: already intubated upon admission (early intubation) vs. intubated during ICU stay (late intubation) vs. never intubated (no intubation). To identify early factors associated with risk for late intubation.

METHODS. We included 1425 patients with sepsis (824) and septic shock (601), consecutively admitted to three tertiary level general ICUs in Italy between 2014-2018. Patients data and outcomes were prospectively collected in a centralized clinical registry (PROSAFE Giviti electronic case report form). Categorical variables are expressed as counts (%) and compared with Chi-square or Fisher's exact test. Median (IQR) was used for continuous variables and differences in distributions were assessed by ANOVA on ranks with Dunn's post-hoc test.

RESULTS. Patients with late intubation were 339 (24%), early intubation 878 (61%), no intubation 208 (15%). Patients in the late intubation group had significantly higher hospital mortality than patients in the other two groups (47% late vs. 31% early vs. 10% no, p<0.0001). Table 1 shows differences between groups at ICU admission: patients with late intubation presented more organ failures, lower GCS, higher SOFA and SAPS II scores in comparison to no intubation

In patients with late intubation, upon admission, we also observed significantly lower oxygenation, more severe acute kidney injury, higher bilirubinemia, lower systolic pressure, higher imbalance of acid-base status vs. no intubation (p<0.01 for all). Interestingly, early clinical and physiologic derangements were similar between late vs. early intubation groups.

CONCLUSION. Mortality of ICU patients with sepsis and septic shock is significantly higher when intubation occurs after ICU admission. Comparable baseline severity with patients already intubated at

000952

Bronchoscopy bronchoalveolar lavage in mechanically ventilated patients in the prone position with Acute Respiratory Distress Syndrome

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INTRODUCTION. Mechanical ventilation in prone position has demonstrated to decrease mortality in patients with severe Acute Respiratory Distress Syndrome (ARDS). It is scarcely documented in the literature clinical experience about use of fiberoptic bronchoscopy (FOB) in prone position , only 9 patients, (1,2). Bronchoalveolar lavage in these conditions are exceptional.

OBJECTIVES. The aim of the present study are: to describe the clinical experience in the realization of FOB in critically ill patients ventilated in prone position and to analyze the complications related to the procedure.

METHODS. Retrospective study performed in an medical-surgical ICU. Time of study was five years. Inclusion criteria were patients with ARDS and indication of FOB. Informed consent was requested for the performance of the procedure. The following variables were collected: Demographic data, heart rate and arterial pressure during the FOB, ICU lenght of stay, level of positive end-expiratory pressure (PEEP) and categories of ARDS according Berlin classification criteria, complications and mortality. The statistical analysis was performed using SPSS program, the quantitative variables are shown in mean ± standard deviation.

RESULTS.

11 FOB were performed during the time of study. Most of patients (81.8%) were indicated for microbiological study performing bronchoalveolar lavage. In one case it was indicated due to complete atelectasis and another case due to pulmonary hemorrhage. The procedure was performed with control pressure ventilator mode with FiO2 of 100% without modifying the previous PEEP. Table 1 shows clinical characteristics. Mean PaO2/FiO2 was 185,1 ± 52,05, resulting between 200 and 300 in 5 patients, between 100 and 200 in 5 cases and < of 100 in one. In most patients (72.7%) no complications related to FOB were observed. 18.2% developed self-limiting supraventricular tachycardia without hemodynamic impact nor needing to increase vasoactives and we observed in 9% transient hypoxemia that was corrected by removing the FOB.

CONCLUSION. In our experience the realization of FOB in patients with ARDS ventilated in prone position is a safe procedure and does not need to decrease or to withdraw the previous PEEP.

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001753

High Flow Oxygen Therapy application outside the ICU in Do Not Intubate patients with hypoxemic respiratory failure

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INTRODUCTION. Since the definition of "subjective experience of breathing discomfort" provided in 1999 by the ATS consensus statement, the concept of dyspnea has been widened covering not only a sensory-perceptual domain (i.e., dyspnea due to increased work of breathing) but also a behavioral-emotional area. Hence, the relief of dyspnea may not be strictly linked to the improvement of gas exchange, but also to the reduction of psychological suffering. Do-not-intubate (DNI) patients with acute hypoxemic respiratory failure (AHRF) are usually treated with noninvasive respiratory supports, mainly aimed at improving patients' comfort. High flow oxygen therapy (HFOT) seems to have a significant impact on dyspnea Limited literature data are available about the treatment of AHRF in DNI patients, included the relief of dyspnea
OBJECTIVES. To assess the effects of HFOT in DNI patients affected by AHRF, hospitalized in general wards
METHODS. We selected a subset of patients included in a larger trial evaluating the use of HFOT outside the ICU under the constant supervision of an Intensivist. 9 wards of Ospedale Policlinico (Milan, Italy) were equipped with HFOT devices; in each ward, educational meetings were given by an ICU physician and an ICU nurse. HFOT was started in patients with AHRF (PaO2/FiO2 ≥150 and <300). The ICU Outreach team followed the patients daily. Arterial blood gases, respiratory rate (RR), dyspnea (assessed by Borg scale) and comfort (assessed through a visual analogic scale) were collected just before the beginning of HFOT, after 2 hours and then every 24. Data are presented as median and interquartile range.
RESULTS. From November 2017 to April 2019, the ICU Outreach Team followed 118 patients. 50 patients (42%) were classified as DNI; 18 (36%) were terminally ill (End-Of-Life). Median age was 76 [66.75-85] years. Hospital mortality was 68%. Twenty-four patients (48%) had oncologic comorbidities and 16 (32%) had COPD. Median PaO2/FiO2 was 156 [116-191]. The duration of HFOT was 10 [4-15] days. After 2 hours from the beginning of HFOT, we observed a significant reduction of RR from 28 [24-31] to 23 [20-28] breaths/min ($p < 0.05$), a reduction of Borg scale value from 3 [2-5] to 2 [1-3] points ($p < 0.05$) and an increase in comfort scale from 3 [2-3] to 3.5 [3-4] points ($p < 0.05$). Gas exchange did not improve
CONCLUSION. HFOT is effective inalleviating dyspnea and improving comfort in DNI patients with AHRF hospitalized in general wards. The effect on dyspnea is not explained by an improvement of PaO2/FiO2 ratio

Table 1 (abstract 001753). See text for description

	Before HFOT	After 2 h HFOT	After 24 h HFOT	p-value
pH	7.45 [7.41-7.51]	7.48 [7.42-7.52]*	7.47 [7.41-7.50]	**<0.01**
pO2 (mmHg)	69 [59-89]	76.5 [63-99.5]	75 [62.3-85.5]	0.39
pCO2 (mmHg)	41 [35-52]	41.5 [37-54.2]	43 [37.2-57.5]	0.26
PaO2/FiO2	156 [116-191]	147 [123-208]	156 [120-195]	0.59
Respiratory Rate (breaths/minute)	28 [24-31]	23 [20-28]*	24 [20-30]*	**< 0.0001**
Borg	3 [2-5]	2 [1-3]*	2 [1-3.7]*	**< 0.0001**
Comfort	3 [2-3]	3.5 [3-4]*	3 [3-4]*	**< 0.0001**
FiO2 (%)	50 [39-54]	50 [40-60]*	50 [35-60]*	**<0.01**
HFOT Flow (L/min)	-	60 [50-60]	60 [50-60]	0.23
HFOT Temperature (°C)	-	34 [31-36.5]	34 [31-36]	0.78

* p<0.05 vs Before HFNC

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SIS - Sepsis treatment and biomarkers

000347

Quantification of plasma exosome as potential diagnostic marker for septic shock

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INTRODUCTION. Exosomes have been studied extensively in several diseases including cancers and cardiovascular diseases as an inflammatory marker, however, little is known about their role and behavior in sepsis. Sepsis is a dysregulated inflammatory response syndrome that leads to multiple organ failure. Current sepsis biomarkers may be helpful in determining organ failure and evaluating the patients' clinical course, but there are no direct molecular biomarkers to predict subsequent organ failure. Exosome play an important role in the inflammatory response, coagulation process, and cardiac dysfunction in sepsis.
OBJECTIVES. The objective of this study was to evaluate the association of plasma exosome with severity of sepsis and mortality in critically ill patients with sepsis.
METHODS. Plasma exosome levels were measured in 80 patients prospectively enrolled in ongoing ICU cohort in Korea between April 2014 and January 2019, in comparison to healthy controls (n = 4). The levels of plasma exosomes expressing CD9 among groups of healthy donor, sepsis and septic shock were compared. To detect and quantify exosomes in plasma, we used a CD9 expression based enzyme-linked immunosorbent assay (ELISA) kit according to the manufacturer's protocol (Novusbio, Littleton, CO, USA).
RESULTS. A total of 80 patients admitted to the medical ICU and diagnosed sepsis or septic shock at our institution were recruited, among which 36.5% presented with septic shock. The median age of patients was 68 (IQR; 61–75) years and 73% were male. Patient with septic shock had higher exosome levels in the plasma compared with healthy controls and patients without shock (median(IQR);

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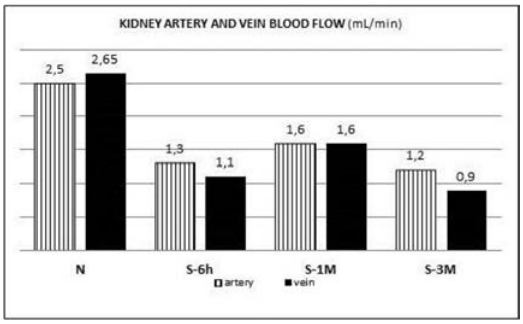

Fig. 1 (abstract 000984). Renal artery and vein blood flow following sepsis induction. S=sepsis, h=hours, M=month, N=name

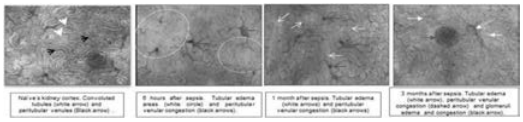

Fig. 2 (abstract 000984). SDF images of the kidney cortex following the sepsis showing microcirculatory injuries up 3 months post-sepssis.

001096

Cardiac and renal effects of remote ischaemic preconditioning on diabetic patients and the relationships with antioxidant and inflammatory markers

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INTRODUCTION. Remote ischemic preconditioning (RIPC) has demonstrated beneficial effects on acute kidney injury (AKI) and/or cardiac function after coronary angiography (CA). These protective effects have been partly explained by enhanced of the antioxidant or anti-inflammatory mechanisms. However, the effects by RIPC might be attenuated in diabetic patients, a population with enhanced of pro-inflammatory and pro-oxidant pathways.
OBJECTIVES. To evaluate the effects of RIPC on the myocardial and renal function, as measured by plasma Troponin I, N-terminal prohormone of brain natriuretic peptide (NT-proBNP), creatinine (Cre) and cystatin C (Cysc C) levels (an early marker of glomerular function), as well as plasma levels of the antioxidant enzyme Heme oxygenase-1 (HO-1), the acute phase reactants increasing during inflammation (ferritin) and protein C reactive (PCR), and the ratio of neutrofile/linfocites (NLR).
METHODS. A prospective, randomized, single-center trial was conducted at the St. Lucía University Hospital (Cartagena, Spain). Diabetic patients admitted to medical intensive care unit, with an acute coronary syndrome requiring standard CA, were enrolled and randomly assigned to an RIPC group (n = 36) or a control group without RIPC (n = 35). In the RIPC group, four cycles of 5-min of ischemia and 5-min of reperfusion were applied to the right upper limb by a cuff inflated to 50 mmHg over the systolic blood pressure. We recorded demographical, clinical and analytical data before and after 24, 48 and 72 hours of CA procedure. We analyzed qualitative variables with percentages by categories, and square Chi; quantitative variable with median ± standard deviation and T student; or median, interquartile rank and U- Mann Whitney, depending on statistical distribution
RESULTS. Patients receiving RIPC showed lower levels of NT-proBNP (pg/ml) at 72 hours (4010±6600) compared to those in the control group (6373±9600), p<0.05, without any differences in the troponin I peak. Regarding to the renal function, the increases in Cre (mg/dl) and Cysc C (mg/L) before and after 72 hours of CA were similar between groups (Cre RIPC; 1.05±0.4 vs 1.37±1 and control; 1.10±0.5 vs 1.17±0.6 and Cysc C RIPC; 1.16±0.5 vs 1.35±0.8 and control; 1.28±0.6 vs 1.38±0.6). Importantly, changes before and after 48 hours in ferritin (ng/ml) (RIPC; 175±176 vs 218±161 and control; 115±98 vs 145±92); HO-1 (ng/mL) (RIPC; 0.72±1.5 vs 1.36±1.5 and control; 0.77±1.5 vs 1.56±2.2); PCR (mg/dL) (RIPC; 4.25±4.3 vs 5.27±4.2 and control; 4.90±6.1 vs 3.96±3.7); and NLR (RIPC; 0.70±0.1 vs 0.70±0.1, control; 0.72±0.1 vs 0.70±0.1); were similar in both groups.
CONCLUSION. The RIPC maneuver before CA in diabetic patients demonstrated favorable effects on cardiac function without improving the renal parameters. These effects could not be attributed to increase in the antioxidant or inflammatory mechanisms analyzed. Alternative pathways underlying these effects merit further investigation.

001129

A novel technique to increase extracorporeal citrate clearance to achieve regional anticoagulation of high blood flows

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INTRODUCTION. Regional anticoagulation allows to perform extracorporeal treatments avoiding the complications of systemic anticoagulation. Citrate-based regional anticoagulation is the most widespread technique but, due to limited citrate clearance, may be applied only to low extracorporeal blood flows (i.e. < 200 ml/min). We developed a novel technique to highly increase extracorporeal citrate clearance, thus achieving regional anticoagulation of higher blood flows.
METHODS. Five healthy swine (42±3 Kg) were sedated, mechanically ventilated and connected to a prototype extracorporeal circuit for continuous renal replacement therapy featuring a citrate-removal stage based on absorbent materials and replacement fluids. Blood flow was 500 ml/min. Sodium citrate was continuously infused at the circuit inlet (5 mmol/L). Heparin was continuously infused. Citrate concentration and Heparinase Kaolin thromboelastography (HK-TEG) were measured on arterial blood, extracorporeal blood downstream the citrate infusion port and downstream the citrate-removal stage.

Samples were collected at baseline, 2, 8, 15, 30, 45, 60, 90, and 120 minutes for citrate and every 30 min for HK-TEG. Calcium chloride was infused to maintain systemic ionized calcium within physiological range. The experiment lasted 2 hours.
RESULTS. Citrate concentrations in blood samples were stable within the first 30 minutes than started to slowly increase (Figure-left). The efficacy of the citrate-removal stage in removing citrate was 88% at 30 minutes and decreased down to 48% at the end of the study (Figure-right) due to loss of efficiency of the absorbent materials. During the whole experiment, HK-TEG in artery showed normal coagulation: Reaction time (R) was 8.6 ±1.3 minutes, with Maximum Amplitude (MA) of 72.2±6.2 mm. In the extracorporeal circuit, HK-TEG showed no sign of clot formation R>60 min, MA=0 mm.
CONCLUSION. The tested prototype effectively removed the infused citrate required to anticoagulate 500 ml/min of extracorporeal blood flow.

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1. This study was funded by the Italian Ministry of Health: Project Code: GR-2013-02356711; Project Title: Optimization of extracorporeal carbon dioxide removal through blood acidification: development of new technologies; Project Type: Young Researcher.

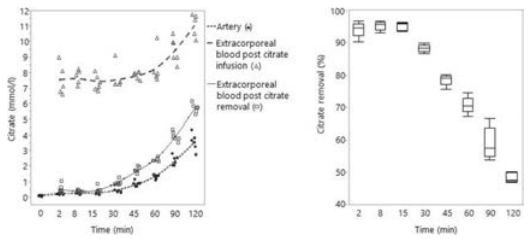

Fig. 1 (abstract 001129). See text for description

001138

Use of cardiac biomarkers as predictors of acute renal injury in cardiac surgery: Prospective Cohort Study

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INTRODUCTION. Acute kidney injury is common after cardiac surgery and is associated with postoperative mortality. Cardiac biomarkers are more accessible and can help to predict this complication.
OBJECTIVES. To investigate relationship between cardiac biomarkers and postoperatively acute kidney injury (AKI) in adults undergoing cardiac surgery1.
METHODS. Prospective cohort study in post-operated cardiac surgery adults. We measured the following cardiac biomarkers in the first 6 hours of the postoperative period: B-type natriuretic peptide (BNP), ultrasensitive troponin I (usTI), creatine phosphokinase (CPK), creatine phosphokinase MB (CPK-MB) and myoglobin.
RESULTS. 63 patients evaluated, 31 (49.2%) had AKI. Patients with AKI had significantly higher levels of BNP, usTI, CPK-MB, and myoglobin. Myoglobin had a good area under the curve (AUC 0.81, 95% CI 0.66-0.95), followed by usTI (AUC 0.71, 95% CI 0.58-0.92) and BNP (AUC 0.71 IC 95% 0.53-0.88) to detect AKI. Myoglobin levels ≥731 ng/mL had a positive likelihood ratio (7.73, 95% CI 1.04-57.24). the time of extracorporeal circulation (Adjusted Odds-ratio 6.11, 95% CI 1.55 to 9.55) and myoglobin elevation (Adjusted Odds-ratio 1.00, 95% CI 1.00 to 1.01) were independent predictors of AKI.
CONCLUSION. The postoperatory levels of cardiac biomarkers are significantly higher in patients with AKI. Extracorporeal circulation (CCE) time and myoglobin elevation were shown to be predictors of AKI. These biomarkers may be suitable for identifying patients at high risk for AKI after cardiac surgery.

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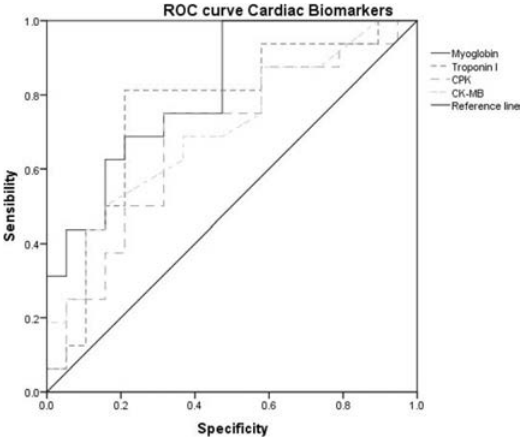

Fig. 1 (abstract 001138). See text for description

001145

Carbon dioxide production derived resting energy expenditure in pediatric continuous renal replacement therapy (CRRT)

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INTRODUCTION. Indirect calorimetry (IC) is the gold standard for estimating energy needs in critically ill children. There is no reported IC data in children receiving CRRT. The modified-Weir equation using real-time carbon dioxide production (VCO2 REE) collected via standard clinical ventilators might more accurately define energy needs without the expense of IC or the inaccuracy of predictive equations.
OBJECTIVES. This pilot feasibility study investigates whether VCO2 REE could be used in pediatric CRRT patients.
METHODS. We investigated if a detectable change in VCO2 occurs after CRRT circuit start and compared daily VCO2 REE to calculated REE. Patients were classified as hypo or hyper metabolic based on the VCO2 REE/calculated REE ratio (<0.9, >1.1, respectively). Continuous VCO2 data were obtained from the Sickbay database (Medical Informatics Corp, Houston TX). Data were sampled prior to initiation of CRRT (baseline) and then every 24 hours for a total of five days or until ventilator discontinuation.
RESULTS. There was no difference in VCO2 between baseline and first hour of CRRT. Median VCO2 REE was 903 cal/d (IQR 554-1600) and calculated REE was 874 cal/d (IQR 606-1427) (n=9). There was a moderate correlation between baseline VCO2 REE and