increase in urinary NGAL in patients receiving bicarbonate infusion was observed compared with control ($P = 0.011$). The incidence of postoperative RRT was similar but hospital mortality was increased in patients treated with bicarbonate compared with chloride (11/174 (6.3%) vs. 3/176 (1.7%), OR 3.89 (1.07 to 14.2), $P = 0.031$). See Figure 1.
Conclusion On this basis of our findings we do not recommend the use of perioperative infusions of sodium bicarbonate to reduce the incidence or severity of AKI in this patient group.

P414
Tubular damage biomarkers linked to inflammation or iron metabolism predict acute kidney injury

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Introduction Here, we compare the value of acute tubular damage markers measured early in the course after cardiac surgery for prediction of postoperative AKI compared with conventional markers used in clinical routine for risk assessment of acute renal function loss.
Methods One hundred adult patients undergoing cardiac surgery in the control arm of a RCT (NCT00672334) were analyzed. We quantified the following biomarkers in urine: NGAL, hepcidin, midkine, IL-6, α_1-microglobulin; in plasma: NGAL, hepcidin, haptoglobin, CK, CKMB, CRP, leukocytes, lactate, urea and creatinine.
Results Preoperatively, no biomarker predicted AKI. At ICU arrival, four urinary tubular damage markers showed good discriminatory ability (AUC ≥0.80) for subsequent development or absence of AKI (Figure 1). An excellent predictive value was found for uNGAL/uhepcidin ratio (AUC 0.90, Figure 1). This ratio combines an AKI prediction marker (NGAL) and a marker of protection from AKI (hepcidin), potentiating their individual discriminatory values. Contrarily, at ICU admission, none of the plasma biomarkers was a good early AKI predictor with AUC-ROC ≥0.80.
Conclusion Several urinary markers of acute tubular damage predict AKI after cardiac surgery and the biologically plausible combination of NGAL and hepcidin provides excellent AKI prediction.
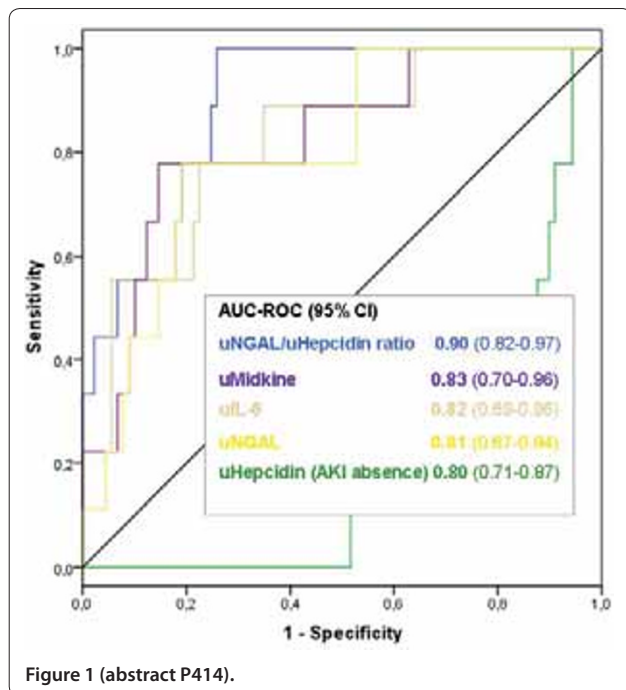

Figure 1 (abstract P414).

P415
Renal response and acid–base balance alterations during furosemide administration

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Introduction Furosemide is one of the most employed diuretics in the ICU for its ability to induce negative water balance. However, one common side effect is metabolic alkalosis [1]. We aimed to describe the time course of urinary excretion and changes in plasmatic acid–base balance in response to the administration of furosemide.
Methods We connected the urinary catheter of 39 ICU patients to a quasi-continuous urine analyzer (Kidney INstant monitorinG®), allowing measurement of pH (pHU), sodium, chloride, potassium and ammonium concentrations (Na+U, Cl–U, K+U, NH4+U) every 10 minutes. The study period lasted 3 hours after a single intravenous bolus of furosemide (time 0). In 13 patients receiving two or more administrations over a longer period (46 (26 to 49) hours), according to clinical needs, we reviewed data on fluid therapy, hemodynamics and acid–base balance from the beginning to the end of the observation.
Results Ten minutes after furosemide administration, Na+U and Cl–U rose from 65 ± 6 to 140 ± 5 and from 109 ± 6 to 150 ± 5 mEq/l respectively, while K+U fell from 60 ± 5 to 39 ± 4 mEq/l ($P <0.001$ for all electrolytes vs. time 0) with a consequent increase in urinary anion gap (AGU = Na+U + Cl–U – K+U). Urinary output increased from 10 (5 to 19) to 53 (29 to 71) ml/10 minutes ($P <0.05$). After the first hour Cl–U remained higher than Na+U, which progressively decreased, leading to a reduction in AGU and pHU over time. In parallel, a progressive increment in NH4+U was observed. In patients receiving more than one administration we observed an increase in arterial base excess (1.8 ± 0.8 vs. 5.0 ± 0.6 mmol/l, $P <0.001$) and plasmatic strong ion difference (SIDpl) (31 (30 to 33) vs. 35 (34 to 36) mEq/l, $P = 0.01$) during the study period. These changes were due to a decrease in plasmatic Cl⁻ concentration (109.0 ± 1.1 vs. 106.6 ± 0.9 mEq/l, $P = 0.009$). Plasmatic sodium and potassium concentrations did not change. In these patients, considering the total amount of administered fluids and urine, a negative water and chloride balance was observed (–460 ± 403 ml and –48 ± 48 mEq, respectively).
Conclusion Furosemide acts immediately after administration, causing a rise in urinary output, Na+U and Cl–U concentrations. Loop-diuretic-induced metabolic alkalosis may be due to an increased urinary chloride loss and the associated increase in SIDpl.
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P416
Red cell distribution width is not a predictor of mortality in acute kidney injury

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Introduction Given the significant morbidity and mortality associated with acute kidney injury (AKI), there is a need to find factors to help aid decision-making regarding levels of therapeutic support. As a prognostic biomarker, the red cell distribution width (RDW) has attracted interest in the setting of critical care when added to existing scoring systems [1]. By examining RDW in a previously studied AKI cohort, we aimed to evaluate the utility of this routine blood test.
Methods A cohort of 209 mixed critical care patients who received renal replacement therapy for AKI had their demographic and biochemical data retrieved from electronic databases. Outcomes were gathered for ICU and hospital mortality. Incomplete datasets were discarded, leading to 153 complete sets. RDW data were taken from the first sample after admission to the ICU, as were all other biochemical values apart from pre-RRT creatinine and potassium. Overall cohort characteristics were gathered, and two groups were created: those with a RDW value within normal range (≤14.5%) and those with a greater than normal value

(>14.5%). We then further subgrouped RDW to assess the correlation between rising levels and ICU mortality.

Results A total 77.1% of our cohort had a RDW greater than the normal laboratory range at time of ICU admission. Key baseline characteristics (age, APACHE II score, length of stay, ICU mortality) did not differ significantly between patients with normal and abnormal RDW. When subgroup analysis was performed, no statistically significant correlation between rising RDW and ICU mortality was found (Spearman correlation = 0.426, *P* = 0.233).

Conclusion In this cohort of critically ill patients with AKI, RDW was not found to be a predictor of mortality. Our results contradict those of recent studies [1,2]. However, both groups of RDW patients in our study suffered a higher ICU mortality than in other studies. To further explain these findings, we intend to perform multivariate logistic regression analysis and assess the effect of social deprivation on RDW.

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P417

Value of Tc-99m DMSA renal scan in assessing the prognosis and outcome of acute renal failure due to sepsis

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Introduction Acute renal failure (ARF) is a common complication in patients admitted to the ICU. Sepsis is also a well-known risk factor for the development of ARF. The combination of ARF and severe sepsis was reported to carry a mortality up to 70% whereas the mortality of ARF alone is 40 to 45%. The aim of the study is to evaluate the role of renal perfusion scanning in detecting the prognosis and outcome of patients with acute renal failure due to sepsis.

Methods Forty patients with acute renal failure due to sepsis, aged between 15 and 74 years, were enrolled in the study. They were monitored for their ICU prognosis and outcome after doing renal perfusion scanning. All patients were subjected to routine ICU and laboratory investigations including APACHE II and SOFA score.

Results Thirty patients had normal renal scan and 10 patients had abnormal renal scan. The mortality percentage was higher among abnormal renal scan cases (three out of 10, 30%) compared with cases with normal renal scan (seven out of 30, 23.3%) with nonsignificant *P* value: 0.6. The median length of stay/day in ICU was longer among nonsurvivors than survivors 15.5 ± 10, 11.5 ± 8, *P* value: 0.058 (approaching significance). APACHE II score was higher in nonsurvivors than survivors 23.9± 3.2, 19.6± 4.2, *P* value: 0.0001. The percentage of mortality among cases that needed mechanical ventilation was higher (nine out of 16, 56.3%) compared with mortality cases that did not need mechanical ventilation (one out of 24, 4.2% with *P* value: 0.0001).

Conclusion ARF may exert an independent adverse effect on outcome in septic and septic shock patients. It is also a risk factor for mortality. Tc-99m DMSA scanning is useful for detecting renal dysfunction and help to predict the outcome and prognosis.

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P418

Estimated glomerular filtration rate based on hospital discharge creatinine may significantly overestimate renal function and underestimate chronic kidney disease in survivors of critical illness

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Introduction Acute kidney injury (AKI) complicates over 50% of ICU admissions. Episodes of AKI are a major risk factor for development or progression of chronic kidney disease (CKD); however, methods of estimated glomerular filtration rate (eGFR) may be poorly calibrated
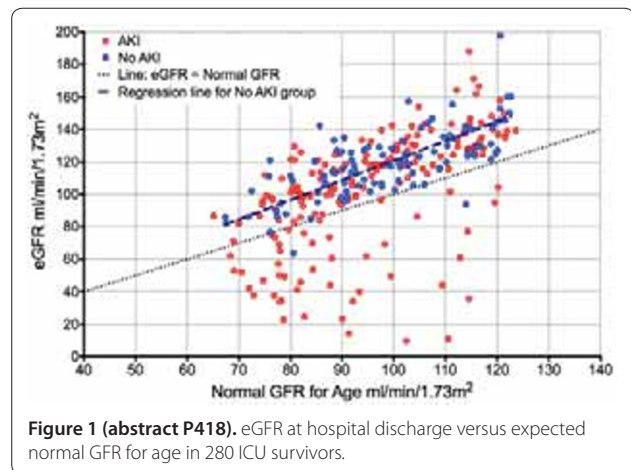

Figure 1 (abstract P418). eGFR at hospital discharge versus expected normal GFR for age in 280 ICU survivors.

to survivors of critical illness who may have reduced muscle mass. We hypothesized that eGFR may underestimate rates and severity of CKD in ICU survivors.

Methods A retrospective observational study of renal function in all patients admitted to a London teaching hospital ICU for ≥5 days and surviving to hospital discharge in 2011. We excluded cases with current or new diagnosis of end-stage renal disease or renal transplant. We assessed AKI in ICU by KDIGO 1 criteria and hospital discharge eGFR by the CKD-EPI equation. For comparison we assumed a normal GFR in a healthy individual as 120 ml/minute/1.73 m^2 at age 20 decreasing by 0.8 per year over age 20.

Results We identified 282 patients, 180 of whom had AKI. Median age was 50 and 68% were male. Median hospital discharge serum creatinine was 57 μmol/l (range 16 to 654), median eGFR was significantly higher than predicted normal GFR for age at 115 versus predicted 95 (*P* <0.001, median difference 16). In patients who had not had AKI discharge the eGFR was 119 versus normal predicted 98 (*P* <0.001, median difference 19), suggesting that eGFR could be overestimating true GFR in our population by at least a factor of 1.23 (Figure 1). Applying this correction factor to eGFRs of patients who had recovered from AKI resulted in 44% more diagnoses of CKD (eGFR <60) at hospital discharge (36 vs. 25).

Conclusion eGFR may overestimate renal function in survivors of critical illness confounding identification of CKD in this at-risk population. Prospective studies with measurement of actual GFR are required to assess the burden of CKD in survivors of critical illness.

P419

Varying models of intra-abdominal hypertension and their effect on renal function in a porcine model

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Introduction Intra-abdominal hypertension (IAH) is an independent predictor of renal impairment and mortality [1]. Organ dysfunction caused by the pressure effect of IAH is well understood, but how this is modified in the presence of bowel obstruction is unclear. The aim of this study was to determine how different IAH models cause renal dysfunction in a pig model.

Methods Twenty-four pigs were divided into three groups; a control group (*n* = 5), a pneumoperitoneum (Pn) (*n* = 10), and an intestinal occlusion (Oc) model (*n* = 10). IAP was maintained for 3 hours at 20 mmHg during which time creatinine, urea, urine output, potassium, and glomerular filtration pressure (GFP) were measured. Statistical analysis was performed using repeated-measures ANOVA.

Results Over the first 3 hours there was a statistically significant difference between the control group and both IAH models for