Acute Kidney Injury Associates With Increased Long Term


Background: Perioperative acute kidney injury (AKI) is common and increases risk of morbidity and mortality. The study objectives were to determine the impact of preoperative risk factors and intraoperative events, with special focus on hypotension, on the risk of perioperative AKI. Methods: In this observational cohort study, patients undergoing major elective noncardiac surgery who were scheduled for an overnight admission to the postoperative unit at the Karolinska University Hospital, Stockholm, Sweden, from October 2012 to June 2013 and January 2015 to April 2016, were included. Preoperative risk factors (comorbidities), intraoperative events (hypotension defined as a percentage decrease in systolic blood pressure relative each patient’s baseline lasting > 5 min) and postoperative data were collected from medical records. Plasma creatinine were measured before, on the first, second and third day after surgery; AKI was determined according to the KDIGO criteria. Data were analysed with STATA version 14.2; the Mannu0392 Whitney u-Test or the chi-square test were used for continuous and categorical variables in the bivariate analyses, multivariable logistic regression, sensitivity analysis and tests of interaction were also performed. Results: Of the final cohort of 470 patients, 127 (27%) developed AKI in the perioperative period. The AKI patients were more often men, had a higher ASA class, a higher frequency of treated hypertension and higher preoperative creatinine. During anaesthesia and surgery, the AKI subgroup had more hypotensive events and blood loss. In the postoperative phase, positive fluid balance was more common among AKI patients, as was myocardial damage and 30-day mortality. Intraoperative hypotension was frequent; 286 patients (61%) had a 40%, and 68 patients (14%) a 50%, reduction from preoperative baseline. Multivariate analyses demonstrated that an intraoperative hypotensive event >50% was associated with a more than doubled risk of AKI (adjusted OR 2.46, CI 1.31-4.62). Conclusions: In patients undergoing noncardiac surgery, there was a high incidence of perioperative AKI. Intraoperative avoidance of hypotension may decrease the risk of AKI substantially.

A acute kidney injury (AKI), which manifests as an abrupt decline in renal function, occurs in ~1% of all hospitalization. Ischemia reperfusion injury (IRI), a common cause of AKI, can occur in any situation where blood flow to the kidney is significantly reduced such as hypertensive crisis, cardiovascular surgery, and inevitably during renal transplantation. Mortality from AKI is up to 80% due to incomplete knowledge of the pathogenesis of IRI and the lack of an effective therapy. It is thought that cellular damage as a result of hypoxia signals the release of proinflammatory cytokines that lead to a systemic inflammatory response. Characteristically during inflammation there is an increase in the blood level of C-reactive protein (CRP), an acute phase reactant whose rise associates with worsened outcomes of AKI. Despite this association, no studies have previously investigated whether CRP has an active role in the AKI process. Using transgenic mice that express human CRP, wild type mice, and CRP-deficient mice (CRLP-/-) we show here that CRP can exacerbate renal damage as early as 24 hours after injury. While many types of immune cells have been implicated in the damaging inflammatory response in AKI, we provide evidence that myeloid derived suppressor cells are likely the CRP responsive leukocyte of importance in AKI. Our data showing that CRP modifies pathways of importance regulating the inflammatory cell response could lead to more effective therapeutic options for AKI.

Introduction: Acute kidney injury (AKI) is commonly observed during the treatment of acute decompensated heart failure. During aggressive diuresis in hospitalized patients, neurohormonal antagonists...
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are often reduced or withheld when a decrement in renal function as indicated by rising creatinine is observed. Neutrophil gelatinase-associated lipocalin (NGAL) is released by tubular epithelial cells of the nephron upon injury and has been detected in the serum much earlier than creatinine. We sought to reproduce previous data supporting the fact that NGAL is an early biomarker of AKI hypothesizing that it would be predictive of the onset or worsening of acute kidney injury. Additionally, we identified specific patient scenarios in which the idea of NGAL-guided changes in neurohormonal blockade may be instituted. Methods: Serial serum and urine NGAL levels were measured from 53 patients with acute decompensated heart failure admitted through the emergency department at the Veterans Affairs San Diego Healthcare System (VASDHS). Serum and urine NGAL levels were obtained on admission and daily up to five days of hospitalization. Patient demographics, laboratory values, medication regimens, physical exam information, and other tests or imaging results were recorded on case report forms. Primary and secondary diagnoses given during hospitalization were also recorded. Study protocols did not influence clinical care decision-making. Results: Of the 53 patients in our study cohort, 24 developed AKI as defined by the AKIN criteria. Serum NGAL was significantly elevated on time point 2 in those who developed AKI versus those who did not (201.9 ng/ml, IQR 122.6-287.8 versus 141.2 ng/ml, IQR 98.55-168.9, p=0.039). Additionally, the maximum serum NGAL across all five time points for each patient was significantly elevated in the AKI group (507.5 ng/ml) versus the non-AKI group (242.1 ng/ml), p=0.007. Per logistic regression the log-odds of having AKI increases by 0.00399 (p=0.024) for each unit of increase in maximum NGAL. Increasing maximum NGAL was significantly associated with having AKI (OR = 1.004, 95% CI: 1.005-1.0075). In patients who developed AKI, serum NGAL levels reached a maximum level 0.25 days earlier than creatinine. Mean time to maximum serum NGAL was 2.42 days compared to 2.67 days for creatinine in the AKI group (p=0.58). Conclusions: Serum NGAL is a potential biomarker of AKI in patients who have acute decompensated heart failure. Importantly NGAL may be a more sensitive biomarker of AKI capable of detecting renal tubular injury earlier than serum creatinine. Further studies are required to elucidate NGAL’s role in guiding neurohormonal antagonist dose adjustments in the hospitalized patient.

Acute kidney injury (AKI) is a major kidney disease associated with high mortality and morbidity. Long-term AKI may lead to chronic kidney disease and end-stage renal disease. Several clinical trials failed due to lack of efficacy and undesired side effects. Studies showed that macromolecular delivery systems would be a promising method to target kidney, however, little is known about how physicochemical properties affects the polymers deposition in ischemia-reperfusion (I/R) AKI. Gene therapy has been well studied as a promising therapeutic agent for several diseases, including cancer and AKI. A though small interfering RNA (siRNA) has been commonly used to treat AKI through hydrodynamic injection, this method has some disadvantages. The expression of CXCR4 increases in response to AKI. Emerging evidence shows that CXCR4/SDF-1 axis is implicated in regulating trafficking and invasion of inflammatory cells in the injured kidneys. The inhibition of the axis appears to exert beneficial therapeutic effect in AKI. First, to understand how physicochemical properties affects renal accumulation in AKI, we synthesized a panel of 9 fluorescently labeled polymers with a range of size and different net charge. By testing biodistribution in unilateral I/R animal model, we found negatively charged pMAA-5 and neutral pHPMA-36 had greatest potential for accumulating in I/R kidneys as compared with sham-operated kidneys. The polymers passed through glomerulus and sustained in proximal tubular cells for up to 24 hours after injection. We also confirmed the consistency of biodistribution in bilateral I/R animal model by confirming biodistribution of pAPMA-30 and pHPMA-16 in bilateral I/R animal model. This study demonstrated for the first time that polymers with specific physical characteristics exhibit promising enhanced ability to accumulate in AKI. We, second, to explore the potential of CXCR4/SDF-1 axis in the treatment of AKI, we formulated polyplexes with our previously synthesized polymeric CXCR4 antagonist (PCX). Biodistribution study indicated that the majority of injected polyplexes can accumulated in injured renal rubule cells. Transfection effect of the polyplexes in unilateral I/R injury mouse model showed a better silencing effect compared with hydrodynamic injection, which provides a novel dual-functional pharmacological method for treatment of AKI.

“This informative book has been put together with the support and input of many clinical renal experts, who have been willing to share their knowledge and years of experience and I’m sure it will be an excellent resource for those caring for kidney patients for the first time – as well as for those with some years of experience!” - From the Foreword by Rosemary Macri, Chief Executive of the British Kidney Patient Association. Kidney Disease Management has been written to help optimise the care of people with chronic kidney disease (CKD) across the healthcare spectrum. It is aimed at a range of professionals, including nurses, junior doctors, general practitioners, pharmacists and dietitians. Specialists in training may also find it useful. It highlights the practical considerations necessary to care for people with kidney problems situations where a specialist practitioner is not always required or immediately available. This book explores: policy context and CKD overview of CKD and management managing CKD in primary care treatment modalities in CKD psychosocial aspects of living with CKD A cute kidney injury in hospitalised patients surgery and kidney injury medication management in CKD nutrition and CKD support and palliative care for people with CKD Key features: Based on best practice and written in an accessible format aimed at busy practitioners. Emphasises the centrality of the patient and family and theneed to share information and expertise across traditional boundaries Written by professionals renowned in their field, bothgeneralists and specialists, who have extensive experience of the practicalities of managing complex patients

The lack of a standard definition for acute kidney injury has resulted in a large variation in the reported incidence and associated mortality. RIFLE, a newly developed international consensus classification for acute kidney injury, defines three grades of severity - risk (class R), injury (class I) and failure (class F) - but has not yet been evaluated in a clinical series. In this general intensive care unit population, acute kidney ‘risk, injury, failure’, as defined by the newly developed RIFLE classification, is associated with increased hospital mortality and resource use. Patients with RIFLE class R are at higher risk of progression to class I or class F. Patients with RIFLE class I or class F incur a significantly increased length of stay and an increased risk of inhospital mortality compared with those who do not progress past class R or those who never develop acute kidney injury, even after adjusting for baseline severity of illness, case mix, race, gender and age.
Background The global rise in antibiotic resistance has led to an increased need for effective antimicrobial treatments. Polymyxins have re-emerged in recent years due to their strong antimicrobial activity against resistant gram-negative pathogens. However, there is concern of renal toxicity associated with the use of polymyxins. This study aims to assess the frequency of occurrence of acute kidney injury (AKI) in the context of incident use of intravenous (IV) polymyxin E (sodium colistimethate; CMS) or IV polymyxin B (PMB), and subsequent mortality, healthcare resource utilization and total hospitalization costs associated with AKI, separately for patients receiving CMS or PMB. Methods A retrospective cross-sectional database analysis using Premier was conducted from January 1, 2012 and September 30, 2015. Patients were included if they were 18 years of age with incident treatment of CMS or PMB for 73 consecutive days (defined as the index admission). Patients with polymyxin use in the previous six months were excluded. Patients with cystic fibrosis or who received both CMS and PMB during the same admission were excluded. The last admission included was on August 31, 2015, allowing for a 30-day follow-up period. Outcome variables included frequency of AKI occurrence, mortality, healthcare utilization and total hospitalization costs during the index admission, and hospital re-admissions occurring within a 30-day period following discharge for the index admission. Descriptive statistics were used to summarize patient characteristics. Bivariate statistics were used to compare healthcare utilization, costs and readmissions in patients who did and did not experience AKI during the index admission. A multivariable logistic regression was conducted to determine the association between AKI during the index admission and mortality. All analyses were stratified by type of polymyxin (CMS or PMB). Results A total of 4,886 patients with incident use of a polymyxin were included; 4,103 patients received CMS and 783 received PMB. The frequency of occurrence of AKI was 31% in the CMS cohort and 27% in the PMB cohort. In the multivariable analysis, the presence of AKI during the index admission was associated with significantly higher mortality in both the CMS cohort (OR 2.26; 95% Confidence Interval (CI) 1.92 to 2.66; p < 0.001). 

Acute kidney injury (AKI) is a frequent clinical syndrome among hospitalized patients, independently associated with both short- and long-term mortality. Previous investigations attempted to identify effective interventions to prevent AKI or promote kidney function recovery in patients with AKI. Most were unsuccessful. Hence, additional studies are required in the field of AKI research. In this Special issue, we are making a call to action to stimulate researchers and clinicians to submit their studies on AKI conducted in nephrology, internal medicine, critical care, and other disciplines that will provide additional knowledge and skills in the field of AKI research, ultimately to improve patient outcomes.

This book presents up-to-date information on the clinical-pathophysiological features of acute renal injury and discusses the KDIGO diagnostic criteria, as well as novel experimental findings, including in the area of regenerative medicine. It also highlights the clinical-pathophysiological importance of AKI in clinical settings, including differential diagnoses and management of AKI. In the past, the pathology associated with sudden renal impairment was characterized as acute renal failure (ARF). However, in the 2000s, the joint efforts of specialists in fields including nephrology, intensive care medicine, and cardiovascular medicine led to the introduction of a novel concept known as acute kidney injury (AKI). As medical care progressed, patients such as high-risk elderly subjects who were not deemed to be candidates for invasive therapy came to be treated in intensive care units (ICUs). As a result, kidney injury as a subset of multiple organ failure was re-considered as AKI, especially in intensive care medicine. AKI was then proposed as a novel disease concept to emphasize the importance of early diagnosis and early intervention to improve prognosis. Presenting novel features, such as the definition of AKI, risk factors and management; biomarkers, such as neutrophil gelatinase-associated lipocalin (NGAL) and L-type fatty acid-binding protein (L-FABP); long-term outcomes of AKI; as well as renal regeneration using iPS cell, manipulation of embryonic genes, and xenotransplanted embryonic kidney, this book is of interest to all physicians and researchers in this field around the globe.

The first section of the book covers the basics of nephrology and second section focuses on acute kidney injury. This easy to reference text examines the physiological and biochemical aspects of renal diseases - all in one convenient resource. Experts in the field discuss topics of increasing concern in nephrology including newer methods of assessing renal function. The field of acute kidney injury in nephrology is a rapidly evolving one with research translating into clinical guidelines and standards. This text brings together experts to provide an authoritative reference for management of AKI in various clinical settings. Pregnancy related AKI is an important entity which has also been discussed in detail. The recent advances in the field of critical care AKI have been incorporated as well and help the reader to update their knowledge.

*AKI* has a high incidence and mortality in critically ill septic patients. We hypothesized that current recommendations for sepsis management do not prevent SA-AKI incidence. Furthermore, the need for CRRT in septic shock patients with SA-AKI is very high. We hypothesized that CRRT timing in these patients should be based on urine output (UO). Finally, when CRRT is indicated, we hypothesized that CVVHD is superior to CVVH in terms of extracorporeal circuit patency and absence of dialytrauma. In our first study the worsening of SA-AKI stage or the appearance of SA-AKI during the following 7 days (from sepsis onset) was clearly associated with a worst outcome in terms of survival (90-day survival). A high percentage of septic patients presented hypotension and this was clearly associated with SA-AKI incidence as well as the presence of an abdominal etiology which is a well-known risk factor for SA-AKI development. Although the accomplishment of the SSC tasks in our study population was globally low, contrary to other studies we did not observe a decrease of SA-AKI incidence in those patients who had high levels of accomplishment. When SSC tasks were separately...
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analyzed, early antibiotic administration was not related with a lower incidence of SA-AKI either. In those patients who were hypotensive, EGDT measures achievement did not decrease SA-AKI incidence. In our second international observational study, we analyzed all those patients with SA-AKI whom required CRRT due to a septic shock condition within the first 24 h from CRRT initiation. A higher age, severity of illness, medical as opposed to surgical admission, a higher BUN at CRRT initiation, a decreased UO and SCr at CRRT initiation, and more days from hospital admission to CRRT initiation were all associated with worse survival. No association between SA-AKI stage at CRRT initiation and 90-day mortality was observed, the same as the majority of previous studies reported. Whether earlier strategies of CRRT initiation (known as "timing") could have an impact in the outcome of patients with SA-AKI was evaluated in a subgroup of homogeneous patients with septic shock all of them presenting advanced SA-AKI stage 3 at ICU admission and initiated on CRRT within the first 5 days from ICU admission. UO and time from ICU to CRRT were compared as timing criteria and UO proved to be more useful when deciding CRRT initiation than a standard "clock time" from ICU admission variable. To demonstrate a higher EC patency with the use of CVVHD in SA-AKI patients we performed a third study comparing the use of CVVHD associated to an adsorption capacity membrane with the use of CVVH associated to the same membrane. Filters were changed at 24 h and 48 h in order to ensure de adsorption capacity. We observed a trend to a longer EC patency with the use of CVVHD although this was not translated in a decrease in the number of dialytrauma events. Cytokines concentrations were measured during the first 72 h and no differences were observed between both groups with the use of an adsorption capacity membrane. Based on all these previous findings we can conclude that SA-AKI incidence and mortality are high in critically ill patients with sepsis especially in those who present hypotension or septic shock. These last patients due to their severe condition often require CRRT which should be initiated only in advanced AKI stages with immediate initiation criteria together with the help of UO. Finally, CRRT in SA-AKI patients when necessary should be encouraged to a preferential use of CVVHD associated to adsorption capacity membranes which seem to improve EC patency with no clinical outcome differences when compared to CVVH. — TDX.

Sepsis is a life-threatening condition caused by a dysregulated immune response to infection. Interestingly, sepsis mortality increases with acute kidney injury (AKI) and patients with AKI worsen with sepsis. It is interesting to note that most of the clinical trials on sepsis treatment that derived from the results of translational researches are a failure. This is, in part, because of the complexity of human sepsis in comparison with animal models. A other reason for the failure-translation might be the improper matching of the animal models to the individual patient. It is possible that the main mechanism of sepsis induction in each patient with the variety causes of sepsis might be different. Indeed, immune response to sepsis depends on genetic background, route of immune activation, and organisms. Thus, sepsis treatment classified by "mechanistic approach" to individual patient might be more proper than the classification with "sepsis severity". Specific treatment of sepsis in individual patient according to the specific immune response characteristic might be a more proper translational strategy. Indeed, the understanding in immune response pattern of sepsis and sepsis pathophysiology is necessary for "sepsis mechanistic approach". Then, we conclude most of the topics and our hypothesis regarding SA-AKI in this review.

A cute kidney injury (AKI) is still associated with high morbidity and mortality incidence rates, and also bears an elevated risk of subsequent chronic kidney disease. A though the kidney has a remarkable capacity for regeneration after injury and may recover completely depending on the type of renal lesions, the options for clinical intervention are restricted to fluid management and extracorporeal kidney support. The development of novel therapies to prevent AKI, to improve renal regeneration capacity after AKI, and to preserve renal function is urgently needed. The Special Issue covers research articles that investigated the molecular mechanisms of inflammation and injury during different renal pathologies, renal regeneration, diagnostics using new biomarkers, and the effects of different stimuli like medication or bacterial components on isolated renal cells or in vivo models. The Special Issue contains important reviews that consider the current knowledge of cell death and regeneration, inflammation, and the molecular mechanisms of kidney diseases. In addition, the potential of cell-based therapy approaches that use mesenchymal stromal/stem cells or their derivates is summarized. This edition is complemented by reviews that deal with the current data situation on other specific topics like diabetes and diabetic nephropathy or new therapeutic targets.

A cute Kidney Injury: New Insights for the Healthcare Professional / 2012 Edition is a ScholarlyPaper™ that delivers timely, authoritative, and intensively focused information about A cute Kidney Injury in a compact format. The editors have built A cute Kidney Injury: New Insights for the Healthcare Professional / 2012 Edition on the vast information databases of ScholarlyNews™. Y ou can expect the information about A cute Kidney Injury in this eBook to be deeper than what you can access anywhere else, as well as consistently reliable, authoritative, informed, and relevant. The content of A cute Kidney Injury: New Insights for the Healthcare Professional / 2012 Edition has been produced by the world’s leading scientists, engineers, analysts, research institutions, and companies. All of the content is from peer-reviewed sources, and all of it is written, assembled, and edited by the editors at ScholarlyEditions™ and available exclusively from us. Y ou now have a source you can cite with authority, confidence, and credibility. M ore information is available at http://www.ScholarlyEditions.com/.

Background: A cute Kidney Injury (AKI) is common after Hematopoietic Stem Cell Transplantation (HSCT) and is associated with increased morbidity and mortality. Risk factors for AKI after HSCT are not fully understood, and there are limited pediatric studies that describe post HSCT AKI. Objective: To assess unique risk factors for AKI in the HSCT population. Design/M et hod: We conducted a retrospective cohort study of patients 20 years age who received an HSCT at Seattle Children's Hospital from 10/1/2008 to 7/31/2017 (n = 484). We defined AKI using Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Severe AKI was KDIGO Stage 2. W e collected information on demographics, baseline transplant characteristics (including conditioning regimen, donor type, cell source, indication for HSCT), post-HSCT complications (inotrope use, sepsis, ventilator use), and mortality at 1 year. Multinomial logistic regression was used to estimate the association between AKI and potential risk factors. W e used adjusted cox proportional hazard ratios to evaluate differences in mortality between groups. Results: 186 patients (38%) developed AKI. Of those with AKI, 79 (42%) had
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severe AKI and 27 (15%) required renal replacement therapy (RRT). There were no significant differences in demographics and indication for HSCT between the groups. Fluid overload was common in all groups with 53 (67%) of those with severe AKI with a fluid overload >10%. Nephrology consult was obtained in fewer than 50% of those with severe AKI. Children who received a cord blood transplant or mismatched transplant had a higher relative risk of severe AKI on univariable analysis only. In both univariable and multivariable analysis, the risk of severe AKI was lower in those who had myeloablative conditioning (RR 0.65 (95% CI 0.3-0.99)) and those who received Tacrolimus (RR 0.42 (95% CI 0.11-0.73)). Risk of mortality was 4.61 (2.61-8.15) times higher in severe AKI compared to no AKI. Conclusions: AKI and fluid overload are common in pediatric patients post HSCT. In our study, severe AKI was less frequent in those with received a myeloablative conditioning regimen or Tacrolimus and was associated with higher mortality. These risk factors could represent unique causes of AKI in this population that warrant further evaluation for use in predicting AKI after HSCT. Nephrology consultation was underutilized and often delayed.

A cute Kidney Injury (AKI) is a complex syndrome that is prevalent among hospitalized patients. In recent years, occurrence of AKI events has risen due to a growing susceptibility of fragile and elderly subjects and an increase in the use of complex procedures such as cardiovascular surgery and imaging techniques. Exposure to potentially nephrotoxic drugs, such as new chemotherapeutic agents, is also proving to be a cause of AKI. This book summarizes recent advances in various settings. A reappraisal of current definitions and staging classifications for AKI in the literature is followed by a description of new criteria for identifying patients at risk and characterizing early kidney damage by using biomarkers. Other important topics include the sequelae of AKI and AKI in special populations such as children, the elderly, and those with cancer. The effects of AKI and its consequences on healthcare expenditures are also addressed from several perspectives. AKI management requires the cooperation of different specialists to optimize outcomes. This book is thus a perfect tool not only for nephrologists, but for every specialist involved in the complicated endeavor of improving patient care.

A cute kidney injury (AKI) is a common condition with significant associated morbidity and mortality. Although impressive progress has been made in the understanding of the molecular and biochemical mechanisms of kidney injury, as well as in the clinical care of patients with AKI, outcomes have remained disturbingly static over the last 40-50 years. This new book presents topical research data in the study of the causes, diagnosis and treatment of acute kidney injury. Topics discussed include classification of AKI; acute renal failure in the newborn; kidney ischemia and reperfusion injury; pandemic H1N1 influenza A infection and AKI; the role of oxidative stress in renal ischemia; biomarkers in acute kidney injury and B2 adrenoceptor therapy in AKI.

This book describes the techniques, strategies, and drugs that have been demonstrated by at least one paper published in a peer-reviewed journal to significantly influence survival in patients with or at risk for acute kidney injury. Each chapter focuses on a specific intervention. The scope is accordingly wide, with coverage of topics as diverse as the type, timing, and dose of renal replacement therapy (RRT), anticoagulation and specific indications for renal replacement therapy, perioperative hemodynamic optimization, fluid balance, diuretics, colloid, fenoldopam, terlipressin, N-acetylcysteine, and vasopressin. A variety of settings are considered, including critically ill patients, cardiac surgery, and hepatic and hematologic disorders. The topic selection was made using a democracy-based approach in which hundreds of specialists from dozens of countries expressed, via the web, whether they agreed with these topics and whether they used the techniques in their daily clinical practice. The clear text is supported by “how to” sections and “key point” boxes that provide easily accessible practical information. The book will be of interest for a wide variety of specialists, including intensivists, nephrologists, emergency doctors, and anesthesiologists.

A cute kidney injury is defined as an abrupt change in serum creatinine and/or urine output, and a majority of patients admitted to the ICU have some evidence of the disorder. Unfortunately, treatment for this complex syndrome is as yet lacking and understanding is limited. An interdisciplinary panel of experts has contributed to this volume, illuminating some of the fundamental and complex aspects of the disorder ranging from pathophysiology to treatment, from emerging biomarkers to genetic polymorphisms. Other contributions focus on immunological issues or the many complications of acute kidney injury and co-morbid conditions encountered, covering the fundamentals as well as the latest developments. Moreover, important technical aspects of extracorporeal therapies including vascular access, anticoagulation or fluid composition are introduced, and different approaches to renal support from intermittent dialysis to continuous therapies and hybrid techniques are discussed. A description of advanced extracorporeal techniques of organ support and their role in the management of sepsis and acute kidney injury in the context of an overall strategy of multi-organ failure management concludes the discussions. This volume not only provides a practical and up-to-date summary of current knowledge and technology, but also imparts a fundamental understanding of the pathogenesis and likely future developments in this field. It also serves to challenge and re-examine the fundamental underlying assumptions we hold regarding critical illness in general and acute kidney injury in particular.

Advances in long-term improvement and outcomes of patients with kidney disease will require the use of novel biomarkers to identify patients at high risk for kidney disease and to diagnose kidney disease early for effective treatment. A biomarker is a substance found in the blood, body fluids or tissues that provides a measure of normal biological or pathological processes or response to pharmacological compounds or drugs. There are a wide variety of biomarkers including but not limited to mRNAs, proteins and peptides, and lipid molecules. In AKI, important pathophysiological processes such as inflammation, apoptotic and necrotic cell death and, tubule regeneration may be reflected in blood or urine. An array of candidate markers along with clinical information in long-term clinical studies with appropriate analytical methodologies will likely provide prognostic information. Despite well-known limitations, currently the most widely used biomarkers for the early diagnosis of CKD and AKI are proteinuria, serum creatinine and blood urea nitrogen. Most clinicians are aware that serum creatinine and blood urea nitrogen are poor biomarkers due to inherent characteristics of these molecules and
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handling by the kidney. Creatinine is secreted and urea nitrogen is reabsorbed by the renal tubules. Many endogenous substances interfere in the assay for creatinine. Serum creatinine and urea appear late after acute kidney injury and the serum levels in part depend on the generation (large or small body mass). A cute kidney injury is a non steady state condition thus serum creatinine and urea nitrogen will lag behind kidney injury. For these reasons new biomarkers are imperative. With knowledge of these limitations in use of current biomarkers and the lack of progress in reducing the mortality and morbidity from kidney disease, there has been a great surge of interest in identifying novel biomarkers with a particular emphasis on the early diagnosis of kidney disease. A variety of methods have been employed including transcriptomics, proteomics, gene arrays and lipidomics. Currently, candidate biomarkers have been found in different disorders and have been tested in humans and many candidate biomarkers have yet to be identified. Most studies to date are preliminary and require validation in large multicentre studies followed by commercial assay development validation and testing. This new book outlines the rapid advances made in the field of biomarker development for kidney disease in which a variety of novel molecules have been identified and studied in humans. A cute Kidney Injury: New Insights for the Healthcare Professional: 2013 Edition is a ScholarlyBrief™ that delivers timely, authoritative, comprehensive, and specialized information about Diagnosis and Screening in a concise format. The editors have built A cute Kidney Injury: New Insights for the Healthcare Professional: 2013 Edition on the vast information databases of ScholarlyNews™. Y ou can expect the information about Diagnosis and Screening in this book to be deeper than what you can access anywhere else, as well as consistently reliable, authoritative, informed, and relevant. T he content of A cute Kidney Injury: New Insights for the Healthcare Professional: 2013 Edition has been produced by the world’s leading scientists, engineers, analysts, research institutions, and companies. A ll of the content is from peer-reviewed sources, and all of it is written, assembled, and edited by the editors at ScholarlyEditions™ available exclusively from us. Y ou now have a source you can cite with authority, confidence, and credibility. More information is available at http://www.ScholarlyEditions.com/.

A cute kidney injury (A K I) is a serious disorder in which sudden impairment of kidney function occurs secondary to one or more of a variety of underlying conditions and exposures. It is very common in (elderly) ICU patients and associated with very high mortality. Many of those who survive suffer from permanent kidney failure and other long-term morbidities. Renowned experts from around the world have contributed to this new publication, creating a succinct yet complete review of the most controversial aspects of A K I. The topics range from epidemiology and basic science to pathophysiology and clinical issues. It is intended as a concise reference work for physicians and nurses who deal with A K I in clinical nephrology and intensive care wards on a daily basis.

Background and Goal of Study: Several studies showed that acute kidney injury (A K I) is associated with chronic kidney disease (C K D) after cardiac surgery1). However, there are no studies investigating the association between A K I and C K D after major abdominal surgery. In addition, recent investigations suggested that patients with cancer develop C K D after A K I2). The aim of this study was to determine the association between C K D and A K I in cancer patients after major abdominal surgery. To this end, we retrospectively studied non-CKD patients who underwent major abdominal surgery and developed A K I after surgery Materials and Methods: A total of 478 patients who underwent major abdominal surgery (excluding cancer and urological surgery) between 2013 and 2015 were followed for 2 years. A K I was defined as KDIGO classification and C K D was defined using guidelines of Japan Society of Nephrology. Cox regression analysis and multivariate logistic analysis were performed for abdominal surgery with cancer and non-cancer patients by matching propensity scores. The propensity score for abdominal surgery for cancer was estimated using age, preoperative eGFR and A K I covariates. Results and Discussion: A total of 101 patients developed C K D (21%). A K I was associated 26 patients (36%, p=0.002) and cancer surgery was included 70 patients (69%, p=0.003). M atching was possible for 110 cases. During the 2-years follow-up, 11 cases (20%) developed C K D after cancer surgery as opposed to 2 cases (3.6%) in non-cancer surgery ( hazard ratio: 4.9; 95% CI: 1.3 to 31.6; p=0.016). On the other hand, 6 cases (46%) developed A K I as opposed to 13 cases (8.1%) in the non-A K I group (hazard ratio: 4.7; 95% CI: 1.5 to 14.2, p=0.009). M ultivariate logistic analysis showed that A K I and cancer surgery are risk factors for C K D (A K I: odds ratio: 6.6, 95% CI 1.3 to 39.4, p=0.025; cancer surgery: odds ratio: 8.8, 95% CI: 1.6 to 81.5, p=0.009). These results indicate that the risk of C K D in cancer patients who undergo major abdominal surgery and have A K I during the perioperative period is higher than in non-cancer patients without A K I. Conclusion(s): This study shows that cancer and A K I are independent risk factors for C K D after major abdominal surgery.

The kidneys participate in all vital processes of the body to maintain overall homeostasis and health. When kidneys are injured during surgical interventions, metabolic and hemodynamic control is disrupted, leading to dysfunction associated with greater mortality, length of hospital stay and cost. Peri-operative Kidney Injury presents the epidemiology, risk factors, diagnosis, treatment and outcomes associated with kidney injury during the peri-operative period. Concepts and principles of care to prevent kidney complications during surgical procedures are provided to equip health care professionals along with strategies to manage acute kidney injury and associated challenges when they occur. Chapters detail diverse surgical settings, ranging from the more common, such as abdominal, cardiac and vascular surgeries, to the intricately complex, including the use of the left ventricular assist device and organ transplants. This practical and comprehensive text blends the evidence-based standards of care with cutting edge advances in the field, while also providing the reader with a peek into innovations on the horizon. A cute kidney failure is an important clinical area in the intensive care unit setting. An estimated 5-20% of critically ill patients experience an episode of acute kidney failure during the course of their illness, and about 5% of patients admitted to an ICU will eventually require renal replacement therapy. In these patients, in-hospital mortality is extremely high, exceeding 50%. Thus, the early detection and causal treatment of acute kidney problems is vitally important for a successful outcome. Written by internationally renowned experts, this clinical reference offers helpful advice with the most recent
information on the definition, epidemiology, pathophysiology, and clinical causes of acute kidney failure as a fundamental prerequisite for prevention of this disorder. Moreover, it also covers differential diagnostic approaches for patients with acute renal failure and provides a detailed outline of important measures for their clinical management. Finally, separate chapters are dedicated to various key aspects related to the adequate delivery of acute renal replacement therapy. It is intended as a helpful guide for all clinicians involved in the care of patients at risk of developing acute kidney problems.

Background: Sepsis is the most common cause of acute kidney injury (AKI). AKI is associated with poor outcomes including progression to chronic kidney disease, increased intensive care unit and hospital length of stay, and mortality. Recent evidence suggests that trajectory of AKI (duration and resolution/persistence), rather than KDIGO stage, is associated with these poor outcomes. Fluid management decisions made during early resuscitation have the potential to improve AKI trajectory in patients with septic shock. Objective: To examine the association between emergency department (ED) fluid management patterns and AKI resolution in a cohort of ED patients with septic shock. Methods: Retrospective cohort study of 292 patients with septic shock identified in the ED at an academic county hospital in Seattle, WA from 2009-2015. Multivariable relative risk regression was used to examine the association between two exposures (1) total resuscitation fluid volume administered in the ED and (2) total volume of lactated Ringer's solution administered in the ED, and the outcome of unresolved AKI at the earliest of death, discharge, or hospital day 5. Measurements and Main Results: Two-hundred six patients (71%) had sepsis-associated AKI and 83 (28%) had unresolved AKI. Patients with unresolved AKI were older, had more comorbidities, and were more severely ill. Mortality during the first 5 hospital days was more common among patients with unresolved AKI (34%) than those with resolved AKI (10%).

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The lack of a standard definition for acute kidney injury has resulted in a large variation in the reported incidence and associated mortality. RIFLE, a newly developed international consensus classification for acute kidney injury, defines three grades of severity - risk (class R), injury (class I) and failure (class F) - but has not yet been evaluated in a clinical series.

A cute kidney injury (AKI) in the pediatric intensive care unit (PICU) is associated with poor hospital outcomes. In adults, AKI is associated with the development of chronic kidney disease (CKD) and hypertension in the long-term, both of which are cardiovascular risk factors. However, the late renal effects of child AKI are unclear. Objectives: 1. Use patient-oriented research to determine if PICU-AKI is associated with long-term hypertension and microalbuminuria. 2. Develop novel pediatric CKD and hypertension definitions using provincial administrative healthcare data to evaluate AKI-long-term renal outcomes associations. Methods: Two observational cohort studies: 1) Retrospective Database cohort: n=2499 from two Montreal PICUs; 2) Patient-oriented cohort: 101 patients in an ongoing longitudinal study, 7 years post-PICU admission. Database cohort: medical chart data was collected and merged with 5 years post-PICU provincial administrative healthcare data. Administrative health data CKD and hypertension definitions were derived iteratively (diagnostic, procedural, medication codes). Patient-oriented cohort: 24-hour ambulatory blood pressure monitoring (ABPM) and first morning urine collection were performed to ascertain hypertension and microalbuminuria. Blood samples were not collected at this study visit and therefore glomerular filtration rate was unavailable. Univariate analyses were performed to compare long-term outcomes in patients with vs. without AKI. Results: Database cohort: n=2404; 24.2% (477/1973 with data available) developed AKI. Patients with AKI were younger, had a higher Pediatric Risk of Mortality (PRISM) I score, were more likely to require mechanical ventilation and had a longer length of PICU stay. Within 5 years post-PICU, 8.6% had a CKD diagnosis (AKI: 15.7%, non-AKI: 6%), p<0.001.

The book offers novel insights on topics such as congenital obstructive nephropathy, cerebral-renal salt wasting, and the role of hemoglobin variability in clinical outcomes of CKD which are not very often discussed in the literature. With comprehensive and insightful reviews by eminent clinicians and scientists in the field, this book is a valuable tool for nephrologists.

Associated with both acute kidney injury (AKI) and cardio-renal syndromes (CRS), new biomarkers represent both a popular area of investigation and a new opportunity for advancement of therapy. This book contains the resolutions of the most recent ADQI conferences on biomarkers in AKI (Dublin) and on cardio-renal syndromes (Venice). The first part answers specific questions about new biomarkers and their use and utility in AKI: What are the most suitable candidate molecules and physiologic measures, how solid and evidence based is the discovery phase? How can we incorporate the new biomarkers in the AKI conceptual model describing the evolution from susceptibility to insult, decreased GFR and organ death? Even if we have a positive biomarker pattern and we can identify patients...
at risk or patients with early or even subclinical AKI, how is this information affecting our clinical behavior and practice? The second part is dedicated to the appraisal of the current knowledge about the pathophysiological mechanisms involved in different forms of CRS: it contains contributions on the state-of-the-art knowledge and practice of CRS, particularly focusing on the pathophysiology of the five subtypes. A acute and chronic mechanisms of damage are explored in depth, with particular attention to the primacy of organ involvement and the subsequent pathways of organ crosstalk. Presenting the most recent research in the field of biomarkers, AKI and CRS, this publication is an important educational tool for advanced investigators and clinical experts, but also for students and fellows.

A timely update A acute kidney injury (AKI) is a serious and as yet incompletely understood disorder in which sudden impairment of kidney function occurs secondary to one or more of a variety of underlying conditions. This disorder is very common in (elderly) ICU patients and is associated with very high mortality. Many of those who survive suffer from permanent kidney failure and other long-term morbidities, which may include cardiovascular disease and immune dysfunction. Epidemiologic evidence suggests that AKI is not a single disease, but a syndrome comprised of multiple, often coexisting, etiologies. Being usually part of multorgan failure syndrome, it calls for multiple organ support therapy. The publication at hand contains sections on prerenal azotemia syndromes, dying of or with AKI, pathophysiology of sepsis-induced acute kidney injury, developments in prevention / treatment / rehabilitation, and renal support. Reporting the latest recommendations from experts, it provides valuable information for those that are interested in understanding the disorder and its treatment options.

This comprehensive guide covers the causes, characteristics, and presentations of acute kidney injury (AKI), as well as prevention and treatment. The first part of the book features chapters on the epidemiology and diagnosis of AKI. This is followed by sections on pathophysiology, clinical syndromes and patient management. Authored by leading clinicians, epidemiologists, basic scientists, and clinical trialists, this book captures the latest evidence and best practices for treating patients with AKI.

This book is the first title that focuses exclusively on kidney disease and its impact in the cardiac catheterization laboratory. The increasing prevalence of vascular risk factors such as diabetes, obesity and hypertension coupled with increased longevity has resulted in a worldwide epidemic of cardiovascular and chronic kidney disease (CKD). Never has the impact of one organ system on the other been so profound, as in the current context of cardio-renal interactions. The cross talk between the heart and kidneys is highly relevant in the field of interventional cardiology, given the increasing number of trans-catheter procedures being performed in patients with underlying kidney disease. These procedures also have a significant impact on kidney function and require thoughtful interdisciplinary planning by a cardirenal team, to achieve optimal outcomes. This book assembles the collective expertise of several international leaders in the field of interventional cardiology and nephrology to summarize this complex interface. The book is divided into seven sections to comprehensively cover the topic, including sections on best practices with reduction of contrast associated acute kidney injury, cutting edge techniques to minimize kidney risk with complex interventions, impact of transcatheter valvular procedures on kidney function and the utility of cardio-nephrology teams. Less recognized complications with high morbidity such as atherosclerotic renal disease are featured prominently, to increase awareness in the interventional cardiology and nephrology communities. This book is a valuable resource for interventional and structural cardiologists, general cardiologists and nephrologists dealing with the significant overlap areas between these two specialties. It is also relevant to medical students, trainee physicians in nephrology and cardiology, advanced care practitioners and nursing personnel in both specialties. Given the major impact of kidney function on outcomes in patients undergoing cardiac procedures, this textbook serves as a focal point to integrate relevant clinical data from both specialties and help interventional cardiologists achieve optimal outcomes, especially in patients with (or at risk for) kidney disease.