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UNDERSTANDING THE DISEASE

Management of oliguria

Marlies Ostermann[1*], Andrew D. Shaw[2] and Michael Joannidis[3]

Oliguria is common in critically ill patients. It may be a physiological response to hypovolemia (representing intact glomerular and tubular function) or could be due to decreased glomerular filtration or tubular injury and be a sign of acute kidney injury (AKI). The skill is to distinguish between both scenarios and to manage patients accordingly.

In health, the maximal urine concentrating ability of the human kidney is about 1200 mOsm/kg H_2O. A standard diet in the Western world generates a daily solute load of approximately 600–650 mOsm. Therefore, the minimum obligatory urine volume is 500–600 ml/day for most people. However, daily solute load depends on dietary intake and catabolic state and may exceed 650 mOsm. Further, elderly and critically ill patients and subjects with chronic kidney disease (CKD) are typically not able to concentrate urine to an osmolality of 1200 mOsm/kg. Thus, the obligatory minimum urine volume required for solute excretion may be higher in some patients.

The definition of oliguria varies in the literature [1]. Reports in the 1950s and 1960s defined oliguria as a total urine output (UO) of < 700 ml/24 h and severe oliguria as urine volumes < 500 ml/24 h. Since the 1970s, 400 ml/24 h is commonly used as the cut-off. Weight-based definitions of oliguria have also appeared in the literature, including < 0.5 ml/kg/h and < 0.3 ml/kg/h for ≥ 6 h [2, 3]. Although the relationship between actual and ideal body weight has not been formally explored, it is recommended to use ideal body weight to avoid over-diagnosing AKI, in particular in obese patients.

*Correspondence: marlies.ostermann@gstt.nhs.uk
[1] Department of Critical Care, King's College London, Guy's & St Thomas' NHS Foundation Trust, London SE1 7EH, UK
Full author information is available at the end of the article

Physiological versus pathological oliguria

Oliguria is a physiological response to the release of anti-diuretic hormone and activation of the sympathetic nervous system and the renin–angiotensin–aldosterone system following intravascular hypovolaemia ("renal success") [4] but also nausea or pain. In contrast, oliguria may also be a feature of reduced glomerular filtration and tubular injury. Other context-specific causes include increased intra-abdominal pressure during laparoscopic surgery, clamping of the renal vasculature and inadvertent injury of the urogenital tract.

Impact of oliguria

Isolated physiological oliguria is typically transient and has a relatively good prognosis whereas oliguria in the context of AKI is associated with worse outcomes and increased mortality. A retrospective multi-center analysis of critically ill patients in the Intensive Care Unit confirmed that episodes of oliguria (defined as UO < 0.5 ml/kg/h) occurred on 37% of study days but only 6.2% of episodes of oliguria of 1 h or more were associated with a creatinine rise [5].

Conflicting data also exist regarding the association between intraoperative oliguria and risk of postoperative AKI in patients undergoing major surgery. A sub-analysis of 2444 patients enrolled in the 'Restrictive versus Liberal Fluid Therapy in Major Abdominal Surgery' (RELIEF) trial showed that 36% patients had intraoperative oliguria (defined as a UO < 0.5 ml/kg/h) [6]. Whilst the risk of a subsequent serum creatinine rise was slightly higher in patients with oliguria versus non-oliguria, most oliguric patients did not have a subsequent creatinine rise consistent with the consensus criteria for AKI. Greater intensity of oliguria (i.e. UO < 0.3 ml/kg/h) did not increase the risk either. In contrast, a retrospective analysis of 3560 patients undergoing major abdominal surgery found an association between intraoperative oliguria and subsequent fulfilment of AKI criteria but only if UO < 0.3 ml/kg/h [3]. Thus, intraoperative oliguria may or may not

be associated with a serum creatinine rise and should be evaluated carefully (Fig. 1).

Management

The challenge of managing oliguria is to distinguish between normal physiology and pathological conditions and to predict which patients will progress to developing a creatinine rise consistent with new or worsening AKI [7]. Concern exists that inappropriate management of oliguria (i.e. inappropriate fluid or diuretic administration) may cause harm and turn "normal physiology" into a pathophysiological scenario.

In general, oliguria should prompt an assessment of haemodynamics and fluid status and exclusion of any obstruction of the urogenital system (Fig. 1). Fluid administration is only indicated for patients with intravascular hypovolaemia and circulatory failure to increase venous return, stroke volume and consequently systemic oxygen delivery. Concomitant vasopressor therapy should be considered in case of vasodilation and hypotension and inotropic support in the setting of heart failure. Diuretics only have a role in oliguria if fluid accumulation has occurred and should only be administered following an assessment of the intravascular fluid status [8].

Intraoperatively, clinicians often use UO as a surrogate for organ perfusion. Whilst fluids are the appropriate treatment for intravascular hypovolaemia and acute circulatory failure, oliguria due to raised intra-abdominal pressure and neurohormonal activation is likely fluid-unresponsive. A randomized controlled trial (RCT) in patients undergoing bariatric laparoscopic surgery comparing high fluid volume (10 ml/kg/h) versus low volume

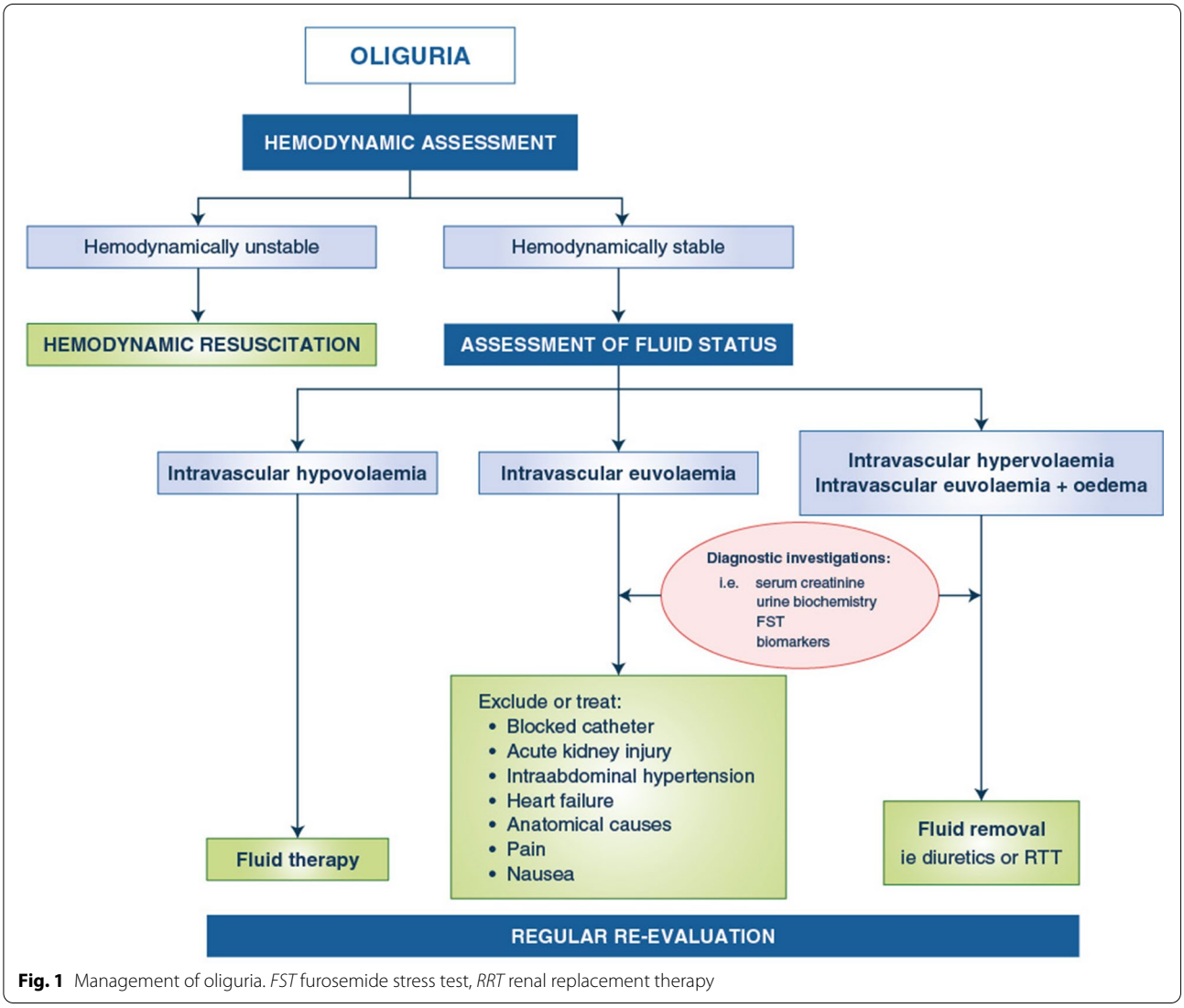

Fig. 1 Management of oliguria. *FST* furosemide stress test, *RRT* renal replacement therapy

therapy (4 ml/kg/h) concluded that UO was low regardless of the volume of fluid administered [9]. Similarly, a RCT in 40 colectomy patients without significant risk of AKI showed that actively targeting an UO of 0.5 ml/kg/h versus a lower target of 0.2 ml/kg/h did not change renal function but resulted in the administration of additional 2.3 l fluid volume within 24 h post-surgery [10]. Meta-analyses have suggested that targeting UO per se did not alter mortality either [11]. Thus, the term "permissive oliguria" has been suggested to avoid harm from over-treating oliguria.

Outside the operating room, the same principles apply (Fig. 1). Historically, urine biochemistry is considered to be useful to differentiate between physiological and pathological oliguria. In health, kidneys respond to transient hypovolaemia or hypoperfusion by increasing urine osmolarity and excreting less sodium and urea. However, this physiological response can be variable and may be confounded by common conditions and co-interventions, including CKD, diuretics, aminoglycosides and cardiopulmonary bypass. A recent meta-analysis evaluated the accuracy of fractional excretion of sodium (FENa) to distinguish intrinsic from prerenal AKI and concluded that FENa may be useful in oliguria provided patients did not suffer from CKD or had received diuretics [12].

Another diagnostic tool that may be used to interrogate tubular function is the furosemide stress test (FST). The methodology is based on furosemide gaining access to the tubular lumen by active secretion via the human organic anion transporters (OAT) 1 and OAT3 in the proximal convoluted tubule. The FST consists of a single dose of intravenous furosemide (1.0 mg/kg for loop diuretic naïve patients and 1.5 mg/kg for those with prior loop diuretic exposure) and replacement of the UO ml for ml each hour with an isotonic solution for 6 h to minimize the risk of hypovolaemia [13]. An UO of > 200 ml in 2 h after furosemide administration is considered an indicator of preserved renal tubular function.

Novel blood or urine kidney biomarkers have potential to increase the diagnostic yield of oliguria and identify patients with increased and low risk of developing AKI [14]. The fact that some biomarkers show higher negative predictive values than positive predictive values for AKI suggests that they might be especially useful at identifying patients with low AKI risk so that unnecessary treatments and investigations can be avoided [15]. Importantly, oliguria may increase the risk of false positive signals of urinary biomarkers when not corrected for urinary creatinine [15].

Oliguria is relatively frequent in patients in the ICU and in the perioperative setting and most episodes are not followed by a serum creatinine rise consistent with AKI. While clinically important, isolated oliguria should be interpreted with caution and within the clinical context. The FST and new AKI biomarkers have potential to help differentiating between physiological oliguria and pathophysiological conditions.

Author details
[1] Department of Critical Care, King's College London, Guy's & St Thomas' NHS Foundation Trust, London SE1 7EH, UK. [2] Department of Intensive Care and Resuscitation, Cleveland Clinic, Cleveland, OH, USA. [3] Division of Intensive Care and Emergency Medicine, Department of Internal Medicine, Medical University Innsbruck, Innsbruck, Austria.

Declarations

Conflicts of interest
MO, MJ and AS report no conflict of interest related to the content of the manuscript.

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