Occasional essay

"I gave her a litre of saline and then some frusemide to 'kick' the kidneys along"

The night registrar was explaining his response to a urine output of 15 mL/hr for two hours in an 80 year old lady who had had a mitral valve replacement 8 hours previously and who was being managed in the intensive care unit during her immediate post operative period. The result was a diuresis of 160 mL for the first hour, 65 mL for the second hour, and then 12 mL for the third hour. The CVP increased from 12 mmHg to 18 mmHg and the arterial blood gas decreased from 82 mmHg (F_IO_2 40%) to 63 mmHg (F_IO_2 50%).

"The initial mean arterial pressure was 70 mmHg, central venous pressure was 12 mmHg and the patient had a serum creatinine of 0.032 mmol/L before the operation - why did the kidney's need 'kicking along'?" I asked.

"Well the nurse was not happy about the urine output, so I thought that..."

"Hang on" I said - I could feel my heart race. "What does your name badge say?" I didn't wait for an answer. "It says 'Doctor'. What does the nurse's name badge say? It says 'Nurse'". I then returned to the point.

"Oliguria is a number, not a disease"

Why was I getting worked up? I guess it's because over the last 30 years, or so, I had had the same conversation with almost all of our trainees. It seems that medical schools still teach the utility of 'kicking the kidneys' when oliguria occurs.

I gathered my thoughts as I started to use the episode as a teaching exercise to explain, as I understood, the principles involved.

"What determines urine output?" I began.

"Renal perfusion" uttered the registrar.

"Anything else?" I enquired.

"Nephron integrity" he said hopefully.

I knew that he was now getting into the nebulous, the sort of answer that a trainee will hope says everything but says nothing. So rather than prolonging the agony I led by saying "Oliguria is usually defined in an adult as a urinary output of less than 20 mL/hr. This assumes that the catheter drainage system is patent and collects all urine accurately - which it often doesn't, - the patient has a solute excretion of 600 mosmol/day and a maximum antidiuresis - which requires an adequate nephron population, hypertonic medulla and normal distal nephron ADH responsiveness. If a patient has a solute load of 300 mosmol/day - a not unusual figure for a frail octogenarian - then during maximum antidiuresis, this may be excreted in 250 mL of urine, that is, 10 mL/hr, before solute retention occurs. In the immediate postoperative period, cortical influences such as nausea, pain and anxiety often provide a maximum non-osmoreceptor ADH secretion, so one might expect that this patient would have an hour or two with a urine output of 10 mL/hr with normal renal function."

I added "If the patient had a normal blood pressure, normal pulse rate and normal peripheral perfusion, the indication for saline would appear to be minimal. And if one suspects an acute renal injury, frusemide will not reduce the incidence of acute renal failure - no matter what the cause."

"But everybody else uses it" he retorted.

"Terrific" I said, "do you treat patients by ritual or reason? Find me an evidence based reference that states frusemide reduces the incidence of acute renal failure." I then added my standard provocation: "The same challenge has been given to all our registrars but none have yet provided me with a 'landmark' study."

Intensivists live in a measurement obsessed community in an imperfect world. What is valuable gets measured and what gets measured is given value. The clinician often has to deal daily with an enormous array of numbers generated from intravascular devices that derive pressures, flows and volumes as well as values from biochemical and haematological tests of blood and other body fluids. The numbers are often compared with those values that are found in 95% of normal individuals (who are supine, at rest, and have been fasting), and, by implication, values that lie outside this 'normal range' are required to be acted upon. However, values that lie outside the 'normal' range should be reviewed in the context of the underlying disease. Numbers are symbols that should be carefully interpreted. If they represent an abnormality caused by disease, the disease should be treated. Twiddling a number by itself is not necessarily associated with a better outcome, and not understanding the physiological or pathophysiological processes involved is a poor platform upon which to base therapy.

The hourly urine output is a measurement commonly recorded in the critically ill patient and often used as a clinical index of renal function. A low or absent value (i.e. oliguria or anuria) are usually perceived as a poor or deteriorating renal state, and commonly prompt a prescription of intravenous fluid or a diuretic. The attention to urine output as a marker of renal function is understandable as the disorder often encountered in critically ill patients, and caused by shock, sepsis, trauma, pancreatitis and various toxins (e.g. intravenous

radiocontrast, aminoglycosides, amphotericin, myoglobin, haemoglobin), is acute tubular necrosis (ATN), for want of a better term, with the major initial feature being a reduction in urine output.[1-3]

To prevent the development of ATN, loop diuretics (e.g. frusemide) could theoretically improve medullary oxygen balance by inhibiting regional oxygen requirements.[4] However, in clinical practice, these agents offer no protection against, and may even increase the severity of, ATN.[1,5-8] In the management of established ATN, low dose frusemide (0.5 - 1.0 mg/kg) is of no benefit,[9-11] and in four prospective randomised controlled trials, high dose frusemide (i.e. 3 - 10 mg/kg) was not associated with any improvement in outcome.[12-15] In one multicentre, retrospective study of patients with acute renal failure, both loop and thiazide diuretic therapies were associated with a 66% increase in in-hospital mortality, and a 77% increase in the odds of death or non-recovery of renal function compared with patients who did not receive diuretics.[16]

"Well what do you do if the patient stops passing urine then?" is the common refrain from a berated trainee, to which I usually reply:

"I make sure that the urinary catheter is not blocked, that there is no 'post renal' problem (I may request a renal ultrasound to assess the renal size and pelvicalyceal system), ensure circulatory adequacy (I may use arterial and right heart catheters to assess the need for inotropic agents and intravascular volume, as pulmonary oedema caused by inappropriate saline infusions will not improve renal function), cease or monitor carefully all nephrotoxic agents (e.g. aminoglycosides, NSAIDs, COX-2 inhibitors), treat all septic foci (antibiotics, surgical drainage, excision of necrotic tissue) and *wait*. If the renal function continues to deteriorate, I use renal replacement therapy - which has been associated with a reduction in mortality."

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