FIVE CAUSES OF ANURIA

DETECTING DETERIORATION, EVALUATION, TREATMENT, ESCALATION, AND COMMUNICATING IN TEAMS

2ND EDITION 2009

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NHI NGUYEN

AIM

The aim of this chapter is to give you the knowledge to:

- understand the first four causes of anuria, i.e. blocked catheter, kinked catheter, no catheter and catheter in the wrong place;
- understand other causes of no or low urine output and become familiar with the various non-mechanical causes of oliguria and anuria;
- learn and understand some simple assessment and treatment options for oliguria and anuria and the relevant warning signs of *When to Worry*.

Early Warning Signs related to poor urine output 	
Poor peripheral circulation	pH 7.2–7.3
Note of decreased urine output	Base deficit –5 to –8 mmol/L
Urine output <200 mL/8hrs	

Table 11

Late Warning Signs related to poor urine output 	
Urine output <200 mL/24 hours Anuric	pH <7.2
	Base Deficit < –8 mmol/L

Table 12

An early warning sign (see Table 11) – urine output <200 mL in 8hrs is associated with increased risk of death. This is also true when there is no catheter or no strict fluid balance or when decreased urine output or failure to void is noted by the nursing staff.

Further down the slippery slope, urine output <200 mL in 24 hours (see Table 12), death is much more likely and for anuria there is a very high risk of death. Early attention to the urine output is very important when looking for deterioration in the patient's condition.

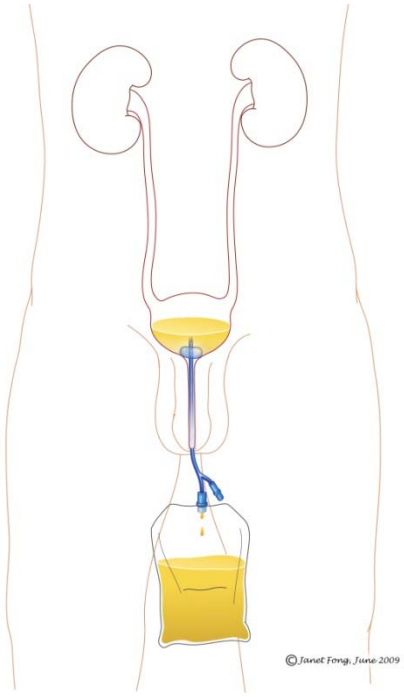
FIVE CAUSES OF OLIGURIA OR ANURIA

1	•Blocked catheter
2	•Kinked catheter
3	•No catheter
4	•Catheter in the wrong place (vagina or prostatic bed).
5	•Others – hypovolaemia, hypoperfusion, obstruction, drugs

Table 13

URINE OUTPUT – WHAT IS NORMAL?

Normal urine production is usually > 1 mL/kg/hr. This is, however, very variable and dependent on the amount of solute (substances dissolved in water including electrolytes, products of metabolism, drugs and glucose) that need to be excreted.



The preconditions for adequate urine production consists of three components:

1. Pre-renal – the blood supply to the kidneys must be adequately maintained.
2. Renal – the kidney units (glomerulae and tubules) must be functioning properly.
3. Post-renal – there cannot be obstruction to urine flow.

WHAT IS OLIGURIA?

Oliguria: 100–400 mL/day

Anuria: <100 mL/day

Absolute anuria: nil **(remember to exclude the first 4 causes of anuria!)**

PRE-RENAL CAUSES OF OLIGURIA

CARDIOVASCULAR CAUSES OF OLIGURIA

1. **Hypovolaemia**
    a. Haemorrhage
    b. Vomiting or diarrhoea
    c. High nasogastric or other drain losses
    d. Diuretic therapy
    e. Pulmonary oedema
2. **Decreased Systemic Vascular Resistance**
    a. Sepsis
    b. Antihypertensive medications
    c. Side effects of other drugs, e.g. ACE inhibitors
3. **Cardiac Pump Failure**
    a. Acute myocardial infarction
    b. Arrhythmias
    c. Cardiomyopathy
    d. Cardiac tamponade.

PATHOPHYSIOLOGY OF POOR URINE OUTPUT

Any abnormal cardiovascular state listed above can result in reduced renal blood flow to the very vascular layer (the medulla) which is exquisitely sensitive to reduced oxygen supply because it requires and extracts maximal oxygen supply. Like the canary sent down the coal mine, historically used to detect inadequate oxygen for the miners, the kidney is a great 'canary' for the body, warning of inadequate cardiovascular status and tissue oxygenation even when the obvious cardiovascular signs such as heart rate and blood pressure appear normal. Via autoregulatory mechanisms and neurohumoral pathways a reduction in renal blood flow or mean systemic arterial pressure will lead to a reduced urine output. If oliguria is due to poor renal perfusion secondary to a fall in mean arterial pressure then this is

potentially reversible. Early recognition of a problem with urine output and blood volume status with timely, simple intervention is important.

Simple **treatment measures** include fluid challenges of 250–500 mL aliquots repeated as necessary with an aim to increase circulating volume, blood pressure and renal perfusion.

Some inflammatory conditions such as pancreatitis and anaphylaxis result in a mixture of hypovolaemia, hypotension and decreased systemic resistance.

Raised intra-abdominal pressure can result in oliguria by a number of mechanisms including direct renal vein compression and inferior vena cava compression leading to reduced venous return and hence decreased cardiac output.

RENAL CAUSES OF OLIGURIA

GLOMERULAR AND TUBULAR DISEASE

These are diseases which affect the kidney parenchyma. They include a variety of auto-immune diseases (uncommon) as well as systemic diseases such as diabetes. The kidney units can also be affected by drugs such as aminoglycosides, non steroidal anti-inflammatory drugs, diuretics, penicillins, cephalosporins, immunosuppressives such as cyclosporine and tacrolimus, cisplatinum, angiotensin converting enzyme inhibitors (ACE1) and radiological contrast.

Always review the drug chart looking for potential nephrotoxins.

Adequate hydration is important to minimise drug nephrotoxicity.

Beware of inadequate fluid intake and dehydration with continued medication ingestion particularly in the elderly.

POST-RENAL CAUSES OF OLIGURIA

OBSTRUCTION TO FLOW OF URINE

This can occur as an extrinsic compression of the urinary outflow tract, for example from prostatic hypertrophy, large calculi, retroperitoneal fibrosis or a mass causing bladder neck obstruction. Check to see if the catheter is draining. If there are no obvious kinks in the catheter and it appears to be in the right place, change the catheter and check the tip to see if there is a blockage. If unable to encourage oral intake, the catheter should be flushed regularly with a three way catheter.

APPROACH TO MANAGEMENT

ASSESSMENT

- Use the ABCDEFG algorithm and DETECT assessment
- Check history
- Assess fluid status
- Look at the input/output charts noting ALL the inputs and losses
- Examine the patient including the abdomen and tubes using look, listen and feel approach. Assess the colour of the urine. Is it dark, clear or blood stained? Don't forget to palpate the bladder or do a bladder scan.

Look in particular for the potential pre-renal, renal and post-renal causes outlined above.

It is often a combination of a few factors. In addition there are some conditions that increase the risk of developing acute renal problems. These include pre-existing renal failure, chronic hypertension (a higher blood pressure may be required to defend the kidneys), diabetes mellitus, vascular disease, trauma (particularly crush injury), chronic heart failure, diuretic dependence, conditions resulting in raised intra-abdominal pressure and radio-contrast.

INVESTIGATIONS

Elevation in serum urea and creatinine levels are late markers of renal problems as they only rise after there has been a >50% drop in glomerular filtration rate. Routine urgent tests for the oliguric patient with potential renal impairment include serum urea, creatinine, sodium, potassium, calcium and creatinine kinase and arterial blood gases and a simple urine dipstick test for blood, protein and sugar. Send a urine specimen to the laboratory for biochemistry, culture and microscopy including casts. Bladder and renal ultrasounds may be useful adjuncts but nothing should supersede clinical assessment and simple treatment measures.

MANAGEMENT

- **See goals of therapy (Table 14 p 56)**
- **Cardiovascular resuscitation** involves fluid resuscitation to haemodynamic targets such as blood pressure, heart rate and urine output all assessed with repeated clinical examination. If appropriate expertise exists, central venous pressure can be measured via a central venous catheter and maintained at around 8–12 mmHg and blood pressure can be measured invasively via an arterial line. Inotropes may be indicated if fluid challenges fail.
- **Prevent further damage** by stopping nephrotoxins, reducing intra-abdominal pressure, treating infection and relieving obstruction.
- **Recognise and treat complications such as hyperkalaemia. (See 'Treatment of Hyperkalaemia' p 56).**

GOALS OF THERAPY

1	• **Recognition** of problem
2	• **Fluid resuscitation** (250 - 500 mL aliquots repeated as necessary)
3	• **Stop** potential **nephrotoxins**
4	• **Do not give diuretics** unless the patient has overt clinical signs of fluid overload
5	• If fluid resuscitation does not work **inotropes** may be required to defend renal perfusion
6	• **Aim for a urine output of 0.5 mL per kilogram per hour**

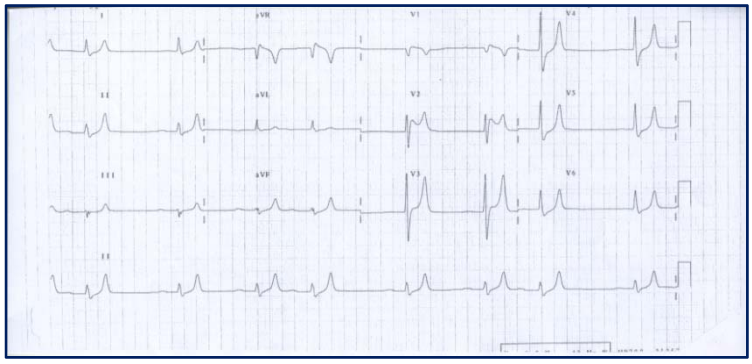
Table 14

Our focus had been on oliguria. Remember polyuria (>200 mL for 2 or more hours) in the absence of recent diuretic therapy can be a harbinger of something serious. Be aware of possible diabetes mellitus or diabetes insipidus. If allowed to continue unchecked polyuria can lead to serious fluid and electrolyte disturbances, e.g. hyponatraemia, hypokalaemia and dehydration. If you are unsure what to do, call for help.

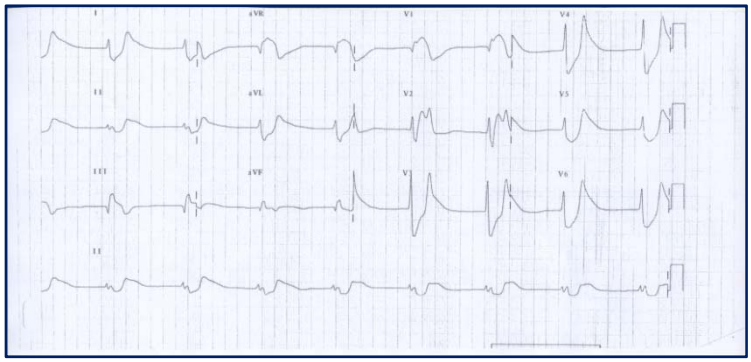
TREATMENT OF HYPERKALAEMIA

1. Treatment strategy depends on severity and rate of rise.

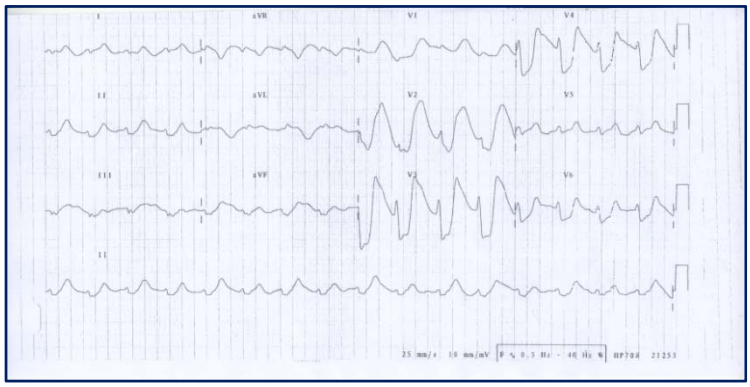
2. Hyperkalaemia >6.5 mmol/L or with significant ECG changes (peaked T waves, wide QRS, ventricular arrhythmias, even asystole and VF) is a MEDICAL EMERGENCY. Therefore invoke your local emergency response system or call for help.



Early signs of hyperkalaemia



Progressing hyperkalaemia



Late signs of hyperkalaemia

3. The step wise approach for hyperkalaemia as a medical emergency in order of priority is:
    a. Institute BLS/ALS as indicated
    b. Stop potassium-containing infusions
    c. Calcium gluconate 10% 10 mL (slow IV push)
    d. Glucose (50 mL of 50% IV bolus) and insulin (10 units of actrapid IV)
    e. Monitor BSL closely
    f. Sodium bicarbonate 8.4% 100 mmols IV over 30 minutes
    g. Inhaled Beta2 -agonist (note 25–40% patients may not respond)
    h. Arrange renal replacement therapy.
4. For modest hyperkalaemia (5.5–6.5 mmol/L); restrict potassium, give resonium 15–30 g PO or via nasogastric or PR. This can be repeated 2 hourly for more severe cases
5. Remember insulin/dextrose and bicarbonate shift K+ into cells and are temporary measures. You need to promote K+ excretion/elimination and reverse the underlying cause.

KEY KNOWLEDGE AND SKILLS

- List causes of anuria.
- Describe the causes and management of oliguria.
- Recognise significant polyuria and the possible adverse consequences.
- Know how to treat hyperkalaemia.

CASE STUDIES

CASE 1

A 75 year old man is Day 1 post-op from an emergency laparotomy for diverticular abscess. He has a background history of hypertension, type II diabetes and osteoarthritis. His usual medications include metformin, aspirin and ACE-inhibitor. He also occasionally takes diclofenac for his arthritis.

As the bedside nurse doing your observations, what sorts of things are you going to look for that may warn of potential deterioration related to renal function? How will you present your concerns to the resident medical officer to convince them to review the patient?

As the medical officer on duty you are asked to review the patient following 4 hours of low urine output. What questions will you ask over the phone? When you see the patient, what is your assessment and early management using the ABCDEFG algorithm and DETECT assessment?

CASE 2

You are asked to review a 70 year old man who has been admitted to hospital with fevers, diarrhoea and vomiting. He has a history of known ischaemic heart disease, diabetes and congestive cardiac failure (last ejection fraction on echocardiography 35%). He has been unable to take his usual medications because of nausea however they include irbesartan-hydrochlorothiazide, aspirin, atenolol and omeprazole. Of note he continues to be nauseated, not tolerating any oral intake and he has not been reported to have passed any urine for the last 8 hours.

As the ward nurse or resident you are asked to assess his fluid status and outline his management.