formulations should be used if available. Frequently measure aminoglycoside trough concentrations and renal function and adjust the dose as required or prescribe alternative drugs. A continuous decrease in glomerular filtration rate shortly after ACE-I initiation should raise the suspicion of bilateral renal artery stenosis and ACE-I should be avoided in these patients.

- **Frequently monitor renal function**

Frequent renal function monitoring will allow for the early detection of drug-induced renal injury. Beware of relying on the creatinine concentration alone to give an indication of renal function, as a value in the laboratory reference range could be falsely reassuring. Creatinine concentrations should be adjusted for weight, age or gender using formulae to estimate the glomerular filtration rate to give a more accurate indication of the renal function. The Cockroft and Gault formula and the Modification of Diet in Renal Disease (MDRD) are the most widely used formulae to estimate renal function.

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- Up to 25% of acute renal failure is drug induced.
- Drug-induced renal injury can present as acute renal failure, chronic renal failure, nephrotic syndrome or tubulopathy.
- Drug-induced renal injury can be minimised by identifying high-risk patients and evaluating the nephrotoxic risk against the therapeutic benefit of administering the drug.
- Take drug-specific precautions to minimise drug-induced renal injury.
- Frequently calculate the renal function by using a formula to calculate the estimated glomerular filtration rate such as the Cockroft and Gault formula and the Modification of Diet in Renal Disease (MDRD), which is often reported by laboratories as the eGFR.

Case report

Deadly herbicide ingestion

A 27-year-old man committed suicide by drinking 1- 2 gulps of a blue-green herbicide, paraquat (Gramoxone). He presented with signs of corrosive mucosal damage, mild transaminitis and acute renal impairment. A diagnosis of paraquat poisoning was delayed, as he initially told health care workers that he had ingested glyphosate (Roundup). He developed type 1 respiratory failure due to pulmonary oedema. Although he had a mitral valve replacement 16 years ago and defaulted follow-up and treatment, he was not in cardiac failure. Over a period of 2 weeks his renal function and transaminitis resolved, but his respiratory function gradually worsened despite his X-ray infiltrates clearing. He died 22 days after paraquat ingestion owing to probable lung fibrosis.

Paraquat ingestion, even one or two gulps, is potentially deadly. Patients with acute, severe fulminant toxicity (more than 40 mg/kg ingested) present with multi-organ failure and die within hours. Patients typically present initially with corrosive damage followed by renal and hepatocellular toxicity within 2 - 3 days. Paraquat accumulates preferentially in pneumocytes. Several days after ingestion irreversible pulmonary fibrosis follows, causing severe morbidity and ultimately mortality.

Treatment consists of initial aggressive management to prevent absorption: activated charcoal should be administered and gastric lavage can be considered within 30 minutes - 1 hour of ingestion, keeping in mind the high risk of perforation. Ipecac is contraindicated. Experimental data indicate that cyclophosphamide and steroids may minimise pulmonary toxicity if administered within 24 hours. Oxygen may increase lung injury by providing oxygen free radicals. Paraquat ingestion is a notifiable condition.

ERIC DECLOEDT, ***MB ChB, BSc Pharmacology (Honours)***
Registrar, Division of Clinical Pharmacology, Department of Medicine,Groote Schuur Hospital, Cape Town

KAREN COHEN, ***MB ChB, FCFP (SA), MSc (Epid), Dip HIV Man, Dip Obst***
Consultant, Division of Clinical Pharmacology, Department of Medicine, Groote Schuur Hospital, Cape Town

Correspondence to: E Decloedt (eric.decloedt@uct.ac.za)

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