Al-Rasheed University College
Department of Dentistry
2st Stage

PRACTICAL BIOCHEMISTRY

lab 10
Kidney Function Tests & Blood Urea

Kidney Function Tests

The kidneys are a pair of organs located in the back of the abdomen, all the blood in our bodies passes through the kidneys several times a day.

Each kidney contains around a million units called ***nephrons***, each of which is a microscopic filter for blood. It's possible to lose as much as 90% of kidney function without experiencing any symptoms or problems.

The kidneys perform a wide range of vital functions in the healthy body, such as:

- Removing wastes and water from the blood.
- Balancing chemicals in your body.
- Releasing hormones.
- Helping control blood pressure.
- Helping to produce red blood cells.
- Producing vitamin D, which keeps the bones strong and healthy.

Kidney diseases are silent killers; kidney disease often goes undetected because symptoms may not appear until the organs are actually failing. Early detection and prevention are the best option to manage the rapidly rising chronic kidney disease. It is important to identify kidney disease early because in most cases the damage in the kidneys can't be cured once it has occurred.

Changes in urinary function

- ✓ Feeling cold all the time
- ✓ Skin rashes and itching
- ✓ Ammonia breath and metallic taste
- ✓ Nausea and vomiting
- ✓ Shortness of breath
- ✓ Pain in the back or sides.
- ✓ Difficulty or pain during voiding
- ✓ Blood in the urine
- ✓ swelling in the hands, feet, ankles and/or face
- ✓ Extreme fatigue and generalised weakness
- ✓ Dizziness & Inability to concentrate

Medications may be used to help control some of the issues associated with kidney failure. Once the kidneys fail completely, the treatment options are limited to dialysis or kidney replacement by transplantation.

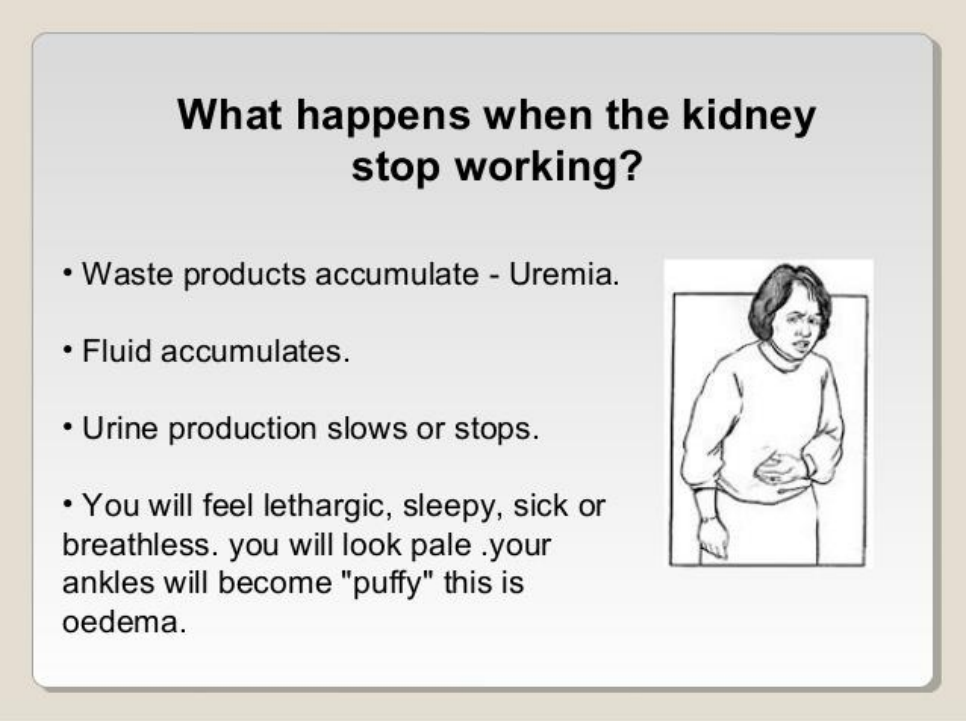
Kidney failure

Acute renal failure (ARF)	A sudden loss of kidney function caused by an illness, an injury, or a toxin that stresses the kidneys (kidney function may recover)
Chronic kidney disease (CKD)	A long and usually slow process where the kidneys lose their ability to function
End-stage renal disease (ESRD)	When the kidneys have completely and permanently shut down

Types of Kidney Function Tests

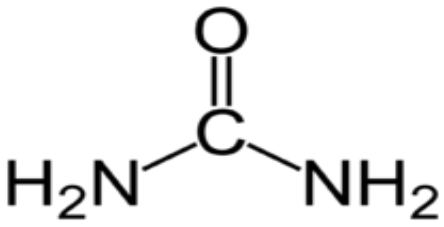
To test the kidney function, doctor will order a set of tests that can be used together to estimate the glomerular filtration rate (GFR). The GFR tells the doctor how quickly the kidneys are clearing waste from your body. Tests include the following:

1. Urinalysis
2. Serum creatinine Test
3. Blood urea nitrogen(BUN)
4. Estimated glomerular filtration rate (GFR)
5. 24-Hour urine sample
6. Electrolytes (Dissolved salts).

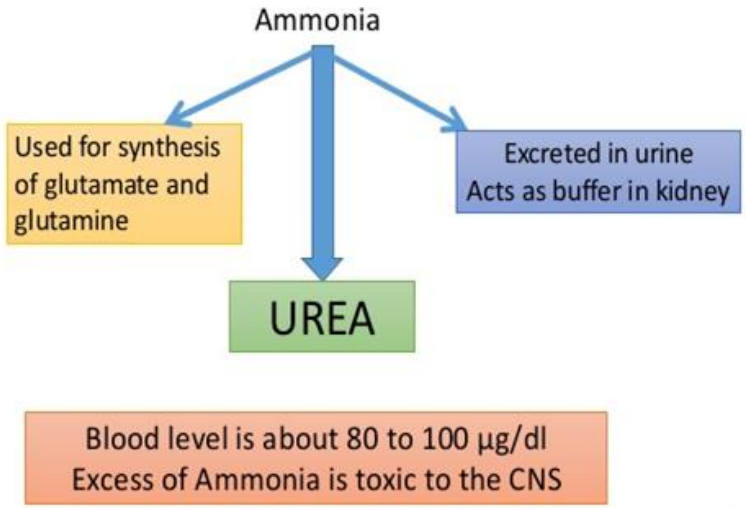


Blood Urea

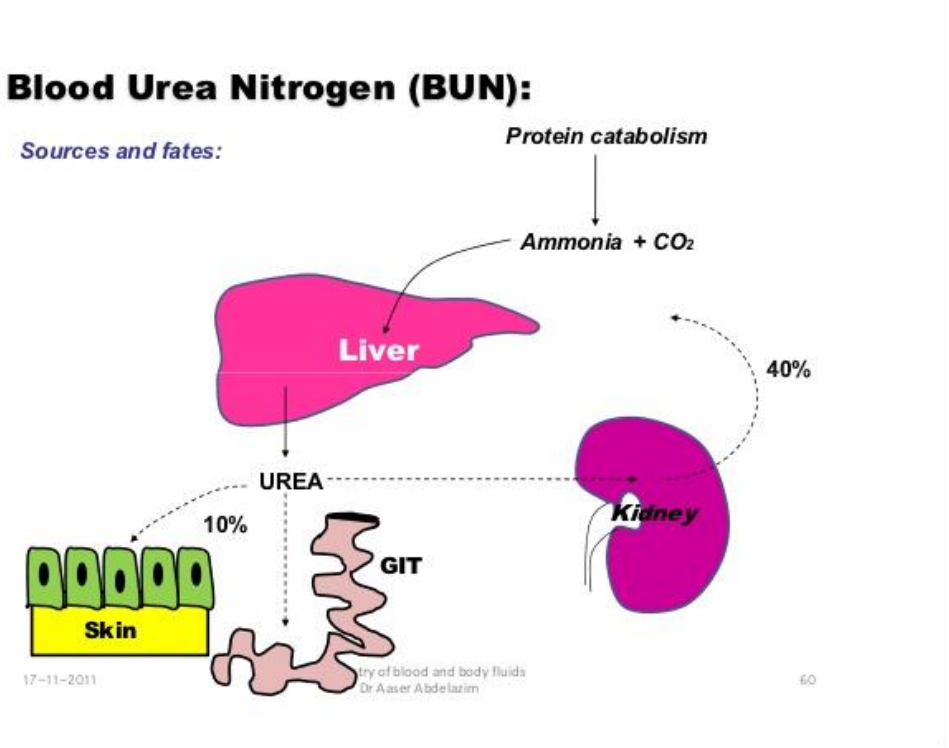
Catabolism of proteins and nucleic acids results in the formation of the so-called non-protein nitrogenous compounds. Urea is the major nitrogen-containing excretory product of protein catabolism in humans. It accounts for more than 75% of non-protein nitrogen eventually excreted. Urea is one of the primary components of urine. It can be defined as an excess of amino acid and protein metabolism end products, such as urea and creatinine, in the blood that would be normally excreted in the urine.



Urea is formed by the liver and carried by the blood to the kidneys for excretion. Because urea is cleared from the bloodstream by the kidneys, a test measuring how much urea nitrogen remains in the blood can be used as a test of renal function. However, there are many factors besides renal disease that can cause BUN alterations, including protein breakdown, hydration status, and liver failure.



The biosynthesis of urea from (amino-nitrogen)- derived ammonia is carried out exclusively by hepatic enzymes of the urea cycle. More than 90% of the urea is excreted by the kidney. Losses through the gastrointestinal tract and skin account for most of the remaining minor fraction.



Normal range:

- ***In young adults***: 15 - 40 mg/dL (2.5-6.7 mmol/L)
- ***In older age group:*** up to 50 mg/dL (8.3 mmol/L)
- ***In children:*** 5 - 15 mg / dL

Clinical significance:

Azotemia is a biochemical designation referring to any significant increase in the plasma concentration of non-protein nitrogenous compounds, principally urea and creatinine.

Azotemia has three classifications, depending on its causative origin, but all three types share a few common features. All forms of azotemia are characterized by a decrease in the glomerular filtration rate (GFR) of the kidneys and increases in blood urea nitrogen (BUN) and serum creatinine concentrations.

The BUN-to-creatinine ratio (BUN:Cr) is a useful measure in determining the type of azotemia. A normal BUN:Cr is equal to 15.

Prerenal azotemia

Prerenal azotemia is caused by a decrease in blood flow (hypoperfusion) to the kidneys, diminished glomerular filtration in the presence of otherwise normal renal function. It can occur following shock, volume depletion, congestive heart failure, adrenal insufficiency, and narrowing of the renal artery among other things.

The BUN:Cr in prerenal azotemia is greater than 20. The reason for this lies in the mechanism of filtration of BUN and creatinine.

Primary renal azotemia

Renal azotemia (acute renal failure) typically leads to uremia. It is an intrinsic disease of the kidney, generally the result of renal parenchymal damage. The pathogenesis primarily a diminished glomerular filtration due to glomerular damage.

Causes include renal failure, glomerulonephritis, acute tubular necrosis, or any other kind of renal disease.

The BUN:Cr in renal azotemia is less than 15.

In cases of renal disease, glomerular filtration rate decreases, so nothing gets filtered as well as it normally would. However, in addition to not being normally filtered, what urea does get filtered is not reabsorbed by the proximal tubule as it normally would be

Postrenal azotemia

Blockage of urine flow in an area below the kidneys results in postrenal azotemia. Obstruction to the outflow of urine through the renal tract (ureters, ladder or urethra leading to high plasma urea by causing both increased re-absorption of urea through tubules (back diffusion) as well as causing diminished glomerular filtration (back pressure). This may occur in renal calculi, enlarged prostate and urological neoplasms.

It can be caused by congenital abnormalities such as vesicoureteral reflux, blockage of the ureters by kidney stones, pregnancy, compression of the ureters by cancer, prostatic hyperplasia, or blockage of the urethra by kidney or bladder stones. Like in prerenal azotemia, there is no inherent renal disease. The increased

resistance to urine flow can cause back up into the kidneys, leading to hydronephrosis.

The BUN:Cr in postrenal azotemia is initially >15. Over time the BUN:Cr will decrease due to tubule epithelial damage. The increased nephron tubular pressure causes increased reabsorption of urea, elevating it abnormally relative to creatinine.

Low serum urea:

1. ***Physiological:*** a- In pregnancy: due to water retention and increased glomerular filtration.

   b- In people on low protein.

2. ***Pathological:*** In severe liver disease due to decreased urea synthesis.
3. ***Iatrogenic:*** In people treated by massive fluid infusion.

Estimation of serum urea

Methods of Estimation

1. ***Indirect methods***: Based on preliminary hydrolysis of urea with unease (urea amidohydrolase) that result in forming ammonia in solution followed by some process that quantitates the formed ammonium ions.
   Spectrophotometric approaches to ammonium quantitation include methods such as Berthelot reaction and Nesslerization.

2. ***Direct methods***: Based usually on some variation of Fearon reaction. In thiosemicarbazide method, there is condensation of diacetyl with urease to form the chromogen, diazine.