[illegible]

Short Notes :

in

Nephrology

Internal medicine

Polyuria

D.D. :

1- **Physiologic** causes :
   - During winter.
   - Excess intake of water.
   - Excess intake of " tea, coffee, beer & cola ".
   - Psychogenic polydypsia.

2- **Renal** causes :
   - Chronic renal failure.
   - Diuretic stage of acute tubular necrosis.
   - Nephrogenic D.I. [congenital tubular defect in reabsorption of water]
   - Intermittent hydronephrosis.

3- **Endocrinal** causes :
   - Diabetes mellitus.
   - Diabetes insipidus.
   - Hyperpitutarism.
   - Hyperthyroidism.
   - Conn's syndrome.
   - Cushing's syndrome.
   - Pheochromocytoma.

4- **Forced diurisis** after use of diuretics.

The Nephrotic Syndrome

Def. : A clinical condition resulting from kidney disease and consists of : (1) generalized oedema, (2) proteinuria, (3) hypoproteinaemia ± (4) hypercholesterolemia.

Pathology :

The most important pathological changes leading to nephrotic syndrome are alterations in the basement membrane of the glomerular capillaries and the cytoplasmic extensions of the epithelial cells of Bowman's capsule.
This results in increased glomerular permeability and proteinuria → hypoproteinaemia → oedema.

AE :

Alteration of the glomerular permeability leading to proteinuria may result from a primary glomerular lesion (Primary Nephrotic syndrome), or be secondary to a systemic disease affecting the kidney as well as other organs (Secondary Nephrotic syndrome).

A) Primary Nephrotic Syndrome :

This is the most important type and is due to glomerulo-nephritis (post-streptococcal or otherwise). According to the pathological changes in the glomeruli as shown by electron microscope; there are many types of 1ry nephrotic syndrome; the most important of which are :

1- Minimal lesion nephropathy :

In which there is only blunting and may be even fusion of the foot processesof the epithelial cells of Bowman's capsule. This is the commonest cause of nephrotic syndrome in children (previously called; Lipoid nephrosis).

- This type is characterized by heavy selective proteinuria i,e: proteinuria with more loss of small molecular weight proteins like albumin, transferring & $\alpha 1$ globulin.
- The lesion in the kidney is completely reversible with very good response to steroids with or without immuno-suppression with permanent remission in 85% of cases.
- It is often precipitated by upper respiratory viral infections.

2- Membranous glomerulo-nephritis :

Where thickening of the basement membrane results from deposition of immune complexes in its outer-part.

* The proteinuria is usually non-selective and the disease usually progresses to chronic renal failure with no response to any line of treatment.

3- Proliferative glomerulo-nephritis with proliferation and thickening of the epithelium of Bowman's capsule and appearance of capsular crescents.

* If the basement membrane is thickened as well, the condition is called " Membrano-proliferative glomerulo-nephritis "
* The prognosis here is also bad and most cases progress to chronic renal failure with no response to treatment.

B) Secondary Nephrotic Syndrome : caused by :

1. Diabetes. [Kimmel-Steil Wilson syndrome]
2. Amyloid deg. of the kidneys.
3. Chronic pyelonephritis. (rarely)
4. Collagen diseases as P.A.N. & S.L.E.
5. Thrombosis of the renal veins.
6. Drugs as mercury, gold and tridione (anti-epileptic).
7. Chronic CHF and constrictive pericarditis.

C/P :

- Age & sex : according to AE.
- Onset : gradual.

Symptoms & Signs :

1- **oedema** :
   - at 1st occurs in the face with puffiness of the eye-lids in the early morning (loose areolar tissue) to disappear during the day (due to blinking movements).
   - Oedema of the ankles appear in the evening (walking)
   - The oedema then becomes generalized & it involves L.Ls, Abdominal wall, U.Ls and face.
   - The oedema may disappear after sometime to reappear again & so on until it persists.
   - Hydrothorax, hydropericardium, ascitis & oedema of the scrotum or valva may occur.
   - It is soft pitting oedema.
2- Various dyspeptic symptoms; anorexia & st. diarrhea (oedema of GIT)
3- Recurrent infections esp. in the skin, sore throat with nasal catarrah. [due to loss of globulins in urine in advanced cases]
4- Anemia c/o as feeling of weakness, headache & pallor [due to loss of transferrin in urine]

Investigations :

- for D.M., collagen dse & amyloidosis.
- Surest AE by biopsy.

Treatment :

A) general : [improve some patients alone]
   1. rest in bed.
   2. ↑ protein, salt free diet.
   3. prophylaxis & treatment of infections.

B) Corticosteroids :
   - very useful in minimal lesion nephropathy and children.
   - Prednisolone 60 mg/day in 10 ds, then gradual ↓ (in 4-8 weeks), then 10 mg/day until proteinuria disappears.

C) Diuretics.

D) Immuosppression :
   - in relapses; or steroid dependency.
   - Endoxan → good in minimal lesion nephropathy.
   - Indomethacin → tried in proliferative glomerulo-nephritis.

E) Others :
   - I.V. albumin.
   - I.V. dextran 1000 cc → ↑ blood volume.

Acute Renal Failure

Def. : passage of less than 400 mL. urine per day, of sudden onset.

Causes : [pre-renal, renal, post-renal]

A) **Pre-renal :** (causes of renal ischemia with diminution of renal blood flow)

1. Excessive fluid loss with dehydration (vomiting, diarrhea, burns and fistulae)
2. shock from any cause (hemorrhage esp. burns, crushing injuries and obstetric causes, general anesthesia, surgical operations, severe infections.... Etc.)
3. Acute severe hemolysis (incompatible blood transfusion, hemolytic crisis due to drugs, infections or in the course of hemolytic anemia).

- The oliguria in these conditions is due to the marked renal vasoconstriction → diminished renal blood flow → diminished G.F. (as the kidneys are part of the splanchnic area).
- The diagnosis is suspected in these cases by the presence of the cause and the occurrence of oliguria which is reversible in the early stages so long as the specific gravity of urine is normal or high. If not treated it will result in tubular necrosis from ischemia with low fixed specific gravity.

B) **Renal** :

1. Primary renal diseases as : acute glomerulonephritis and severe acute pyelonephritis with necrotizing papillitis.
2. Tubular necrosis : 2ry to renal ischemia or toxins (nephrotoxins) such as; mercury, arsenic, carbon tetrachloride, lead and sulpha. When necrotic foci are extensive and seen by the naked eye in cut sections, the condition is called acute cortical necrosis (it commonly complicates eclampsia and accidental hge)

C) **Post-renal** : means causes of acute urinary obstruction.

These are :

1. Bilateral impaction of ureteric or pelvic stones.
2. Unilateral obstruction by a stone and reflex susppression of function in the other kidney (calculus anuria) or unilateral obstruction of a single kidney.
3. Bilateral ureteric obstruction by pus (severe urinary infections), blood clots (hematuria) or crystals (crystalluria of sulpha & uric acid).
4. Bilateral ureteric invasion from outside by fibrosis (retro-peritoneal) or neoplasm.

- the diagnosis is suspected by the history of renal pain or colic, hematuria or other symptoms and then the sudden onset of anuria (no urine at all or urine less than 75 mL/ 24 hours) and not oliguria.

C/P :

In acute tubular necrosis the clinical course passes in **3 phases** (in other causes of renal failure the course is more or less similar). These phases are :

1- **The Oliguric phase** : the amount of urine is reduced, dark and thick (due to necrosed tubular cells and altered blood). There is mental dullness, headache, nausea, vomiting and may be convulsions. There may be increased JVP and pulmonary crepitations (overhydration). Breathing is deep and rapid (acidosis). Coma may result. Hiccough, hematemsis and melena may occur. Anxiety, pallor due to anemia develops rapidly.

***N.B.** : Changes in the blood and body fluids : the renal blood flow is diminished → diminution in GFR + leak through the damaged tubules of a part of GF back to the blood = marked reduction of the glomerular filtrate. So there is retention of :*

- *water → increased blood volume and intracellular hydration → low plasma Na^+ (dilution hyponatremia)*
- *K^+ → hyperkalaemia, also Mg^{++} → hypermagnesemia.*
- *phosphates, sulphates and other anions and H^+ → acidosis and low serum Ca^{++}*
- *urea and creatinine.*

2- **The diuretic phase** : with recovery of the kidneys (this may take up to 2-3 weeks). The urine volume increases may be up to 5-6 L/day rarely. In this phase the changes of the oliguric phase gradually disappear. During this phase there is danger of dehydration and loss of Na^+ and K^+ .

3- **The Recovery (post-diuretic) phase** : urine volume and electrolyte changes gradually return to normal with complete recovery.

Causes of death in Acute renal failure :

A) In the oliguric phase :
   1. cardiac arrest (hyper-kalemia, hypo-calcaemia and hyper-magnesaemia).
   2. intercurrent infections.
   3. pulmonary oedema (heart failure) and cerebral oedema from over-hydration.

B) In the diuretic phase : from dehydration and electrolyte loss (rarely).

Treatment : The important steps are :

1. to exclude urinary retention as the cause of anuria.
2. To exclude urinary obstruction as a cause of post-renal failure.
   - this is done by (1) history (sense of bladder fullness), (2)examination (e.g.: full bladder), (3) catheterization, (4) cystoscopy and (5) ureteric catheterization.
3. Prophylaxis is better than cure, so diagnose pre-renal failure before tubular necrosis has occurred (when the sp. gr. of urine is still above that of the plasma) and treat shock and hypotension properly. One of the best osmotic diuretics to use is mannitol 500 cc 10% sol., it is said to reduce renal vasoconstriction esp. in the efferent arterioles, so improves the blood supply to the tubules and prevents tubular necrosis.

The main differences ( ) Pre-renal failure and acute tubular necrosis are :

	Pre-renal	**Acute tubular necrosis**
Sp. gravity : Urinary Na+ : Response to mannitol :	High Low Increase in the amount of urine (e.g. doupled in 1hour)	Fixed at 1010 High No change in urine amount

4. Treat urinary infections e.g. : acute pyelonephritis early and promptly.

Treatment of the Oliguric phase :

if renal failure is severe : Dialysis is indicated, either peritoneal dialysis (usually) or haemodialysis (artificial kidney).

This is known by : [indications for dialysis]

1. blood urea above 250 mg% (even above 100 mg% according to the facilities).
2. high serum K^+ (above 7 mEq/L.).
3. marked acidosis (serum HCO_3^- below 15 mEq/L.).
4. bad general condition : coma, twitching, GI-bleeding,.... etc.

Otherwise; conservative treatment is done which consists of :

1. Only 500 mL of electrolyte free water + amount equal to the amount excreted in the day-before is given in 24 hours.
2. No or very little protein e.g.: 10 gm/day is given.
3. An excess of carbohydrate is given (to spare protein breakdown). If oral feeding is possible as much carbohydrates as tolerated by the patient is given. If not, 300 – 400 gm glucose are given IV.

4. Fats are not dangerous, they also spare protein but orally they may cause diarrhea and they can't be given IV, so fats are not given.
5. Calcium gluconate and 50 units insulin daily may be used to reduce plasma K^+.
6. Anabolics as durabolin may be used, anti-emetics (for nausea) and bactericidal antibiotics are used when indicated.
7. Follow-up the patient by fluid chart, daily estimation of blood urea, electrolytes, glucose and haematocrite. Weight the patient daily.

Treatment of the diuretic phase : watch and correct any water, Na^+ or K^+ deficiency.

Prognosis : mortality in the best centers is still about 40%. The rest either recover completely (usually) or recover with some degree of renal impairment.

Chronic renal failure

Def.: means failure of the kidneys to keep the composition of the internal environment within normal. This results when at least 75% of the nephrons are destroyed. This can complicate practically any form of bilateral renal disease. The clinical picture is non-specific and always nearly the same irrespective of the cause.

Causes : [renal & metabolic]

A) **Renal cause** :
   a. Diseases of the renal parenchyma :
      i. Chronic glomerulonephritis.
      ii. Chronic pyelonephritis
      iii. Advanced renal T.B.
   b. Diseases of the renal vessels :
      i. Hypertension, (malignant & benign).
      ii. Collagen diseases (PAN & SLE).
   c. Obstruction to the urinary tract causing bilateral pyo- or hydro-nephrosis.
   d. Congenital lesions : as polycystic kidneys or hypoplasia.

B) **Metabolic** :
   a. Diabetes
   b. Gout.
   c. Hypercalcaemia.
   d. Amyloidosis.

C/P :

This condition usually occurs gradually.

Age and sex incidence according to the aetiological condition.

The patient complains of : [symptoms]

1. Urinary symptoms : nocturia, thirst and polyuria. The urine is usually about 3-4 1/24 hours (it can't be a gross polyuria due to limited number of the renal nephrons).
2. General weakness, fatigability and vague aches and pains (anemia & disturbed calcium metabolism).
3. Gastrointestinal symptoms : anorexia, nausea, vomiting and hiccough (of central origin), dyspepsia, retching and rarely GI-bleeding from GI ulcerations (perhaps due to ammonia formation from urea secreted in the GIT) + increased capillary fragility. Diarrhea or more commonly constipation is present.
4. Neurological symptoms : headache, apathy, reversed sleep rhythm and finally coma may occur in advanced cases. Convulsions may occur. Burning feet, progressive numbness and weakness may result from polynuritis (uraemic polyneuropathy).

On Examination : [signs]

1. The patient is pale, yellow with an earthy (dirty beige) complexion (combination of anemia and retention of urochromogen). In more advanced cases the patient will be found confused and may be in coma (uraemic coma).
2. In advanced cases, the respiration is deep and rapid (air-hunger or Kussmaul's resp. due to the acidosis). The breath may smell like urine (urineferous odour).
3. The tongue is dry, also the skin is dry and loose its turgor (dehydration).
4. High B.P. is present in most of the cases (primary or secondary) with evidence of hypertensive heart diseases (e.g.: L.V.H.). Terminally aseptic fibrinous pericarditis with an extensive pericardial rub may occur. Occasionally a large haemopericardium develops (unknown cause).
5. Evidence of latent (or manifest) tetany or polyneuritis may be present.
6. The patient is liable to develop recurrent infections especially in the skin (recurrent boils or carbuncles) and the lungs (pneumonias).

Investigations : (exclude surgical causes of renal failure i.e. examination, plain U.T., I.V.P., cystoscopy and others)

1- Kidney function tests : high blood urea, usually above 60 mg%, high plasma creatinine usually above 2 mg%, low creatinine and urea clearance. Usually the glomerular and tubular functions are impaired (one maybe affected more than the other according to the underlying cause).
2- Urine exam.
3- Blood picture : there is anemia which is usually normocytic normochromic (deficient erythropoietin and bone marrow depression). Leucocytosis and high ESR may be found.
4- Blood chemistry : low blood Ca^{++}, high blood phosphates, low $HCO3^-$ (alkali reserve), high blood uric acid, serum K^+, serum Mg^{++} and low serum Na^+.
5- Fundus examination : may show evidence of arteriosclerotic (chronic) or hypertensive (acute) retinopathy.
6- X-ray chest and heart : show LVH and rarely "uraemic lungs" (dense bilateral opacities radiating from the hilum into the lung fields due to transudation from abnormal capillaries + pul. Venous congestion from L.V.F.).
7- Renal biopsy : may be done at least to determine the exact cause of the failure.
8- Specific investigations : directed towards detection of the cause e.g.; for collagen diseases, amyloidosis and diabetes.

So we diagnose renal failure, its cause and its severity.

Treatment of chronic renal failure :

A) if a specific cause is known : **causal treatment** is given e.g.: pyelonephritis, obstruction of the urinary tract, malignant hypertension, diabetes, renal T.B. ,..... etc.

B) **Conservative treatment** : consists of :

1. Treatment of nitrogen retention : by a low protein diet. 40 gms proteins per day are given with blood urea from 70 – 100 mg% and 20 gms + the essential amino acids and adequate calories if blood urea is above 100 mg% and esp. if it is rising under treatment. Anabolics such as durabolin are given. High doses of frusemide (lasix) about 250 mg daily are found to increase urea clearance.
2. Correction of water, electrolyte and acid-base disturbances by :
   - high fluid intake (up to 3 L/day) to prevent dehydration and increase urea clearance. If dehydration is present from nausea, vomiting or diarrhea, IV fluids e.g.: 5%glucose should be given.

- No limitation of salt intake unless there is oedema, heart failure or severe hypertension.
- Correction of acidosis by 3 – 9 gm NaHCO3 daily. In severe cases IV NaHCO3 can be used.
- Terminally when there is hyperkalaemia, fruits are eliminated from the diet and 15 – 20 gm cation exchange resin in Na or Ca phase are given.

3. Ca metabolism : Vit. D3 10 mg and Ca carbonate 6 – 10 gm are given daily.
4. Treatment of hypertension and heart failure by suitable drugs ; noting : avoid rapid drop in the B.P., use small dose of diuretics and half the doses of digoxin.
5. Treatment of anemia.
6. Treatment of present infection and guarding against infection.
7. Symptomatic treatment : for nausea, vomiting, hiccough, anxiety and convulsions.

C) **Intermittent haemodialysis :** if there is no response to conservative treatment or if the kidney function is grossly impaired from the start, e.g.: serum creatinine from 5 – 10 mg% after correcting all the reversible factors in the patient.

D) Think of **renal transplantation** in suitable cases; if there is bad response to intermittent haemodialysis or the serum creatinine is above 10 mg% from the start and after correcting all the reversible factors present.

H_2O and electrolyte balance

We have :

- Intra-cellular water (ICW).
- Extra-cellular water (ECW) : plasma, interstitial fluid and lymph.
- Trans-cellular water : CSF, synovial fluid, pleura, peritoneum....etc.

* Non-Electrolytes e.g.: urea, glucose, creatine and A.As are distributed equally through-out all body water compartments.

* For the electrolytes, these are special mechanisms in the cell membranes → important differences between the ICW & ECW.

	E.C.W.	I.C.W.
Na^+	135 -145 mEq/L.	0 – 40
K^+	3.5 - 4.5 mEq/L.	150
Cl^-	90 - 110 mEq/L.	25
HCO_3^-	22 - 26 mEq/L.	---

* Total body water 50 – 70% of body weight. [about 40 - 45 L.]
  - ICW 60% → [25 L.]
  - ECW 40%→ [15 L.]
  - Plasma [3 L.] while the interstitial fluid & lymph [12 L.]

* Obligatory H_2O loss = 800 mL insensible respiration.
  500 mL least amount of urine.
  400 mL evaporation in the lungs.

* about 400 mL H_2O are produced per day during the normal metabolism (metabolic H_2O). So, at least 1300 cc H_2O should be supplied from outside (500 in the solid part of the diet + 800 free H_2O).

Hypokalaemia

AE :

1) Decreased intake :
   - IV fluid therapy deficient of potassium.
   - Prolonged decrease of dietary intake.
2) Decreased absorption : as in malabsorption syndrome.
3) Increased loss :
   a. **Renal** causes :
      - Dieuresis :
        * Diuretic especially loop diuretics & thiazides.
        * Diuretic phase of acute tubular necrosis.
        * Osmotic diuresis e.g.: severe diabetes.
        * Tubular disorders e.g.: Fanconi syndrome.
      - Conn's syndrome & Cushing's syndrome.
      - Alkalosis.
      - Antibiotics e.g.: carbenicillin.
   b. **Gastrointestinal** loss :
      - Diarrhea especially "secretory diarrhea".
      - Vomiting.
   c. Excessive **tapping** of **ascitis**.
4) Intra-cellular shift :
   - Alkalosis.
   - Insulin therapy.
   - Hypokalaemic periodic paralysis.

C/P :

1- Muscle weakness or paralysis, hypotonia & hyporeflexia.
2- Constipation, distension & paralytic ileus.
3- Arrhythmias especially in digitalized patients.
4- Prolonged hypokalaemia may result in renal tubular damage (hypokalaemic nephropathy).
5- Alkalosis which may lead to tetany.

Investigations :

1- serum K^+ : is reduced [normal : 3.5 – 5 mEq/L]
2- ECG :
   - sagging depression of **ST** segment.
   - Flat or inverted **T** wave.
   - Prominent **U** wave.
3- Investigations for the cause.

Treatment :

1- Potassium replacement :
   a. Oral potassium is preferred to prevent hyperkalaemia.
   b. IV potassium maybe gives in severe cases with careful monitoring.
2- Treatment of the cause.

Hyperkalaemia

AE :

1- Increased intake :
   - Excessive therapy with IV potassium.
   - Transfusion of stored blood.

2- Decreased excretion :
   a. **Renal** causes :
      i. Acute renal failure.
      ii. Severe chronic renal failure.
      iii. Tubular disorderes. (e.g.: type IV RTA)
   b. **Adrenal** causes : Addison's disease.
   c. **Drugs** :
      i. Potassium sparing diuretics. (e.g.: spironolactone)
      ii. ACE inhibitors. (e.g.: captopril)

3- Extra-cellular shift :
   - Tissue damage. [haemolysis, rhabdomyolysis & tumor-lysis syndrome]
   - Acidosis.
   - Insulin deficiency.
   - Hyperkalaemic periodic paralysis.
   - Drugs. (e.g.: succinylcholine)

4- Pseudohyperkalaemia :
   - polycythemia. - In-vitro haemolysis.

C/P :
- Muscle weakness or paralysis, hypotonia & hyporeflexia.
- Bradycardia, heart block & cardiac arrest.

Investigations :
1- Serum K^+ : is increased. [normal 3.5 – 5 mEq/L.]
2- ECG :
   - tall peaked **T** wave.
   - Prolonged **PR** interval.
   - loss of P wave.
   - widened **QRS** complex.
   - bradycardia, heart block & arrest.
3- Investigations for the cause.

Treatment :
1- Decreased intake of potassium :
   a. Restriction of potassium on diet.
   b. Avoidance of drugs that increase potassium.
      (e.g.: potassium sparing diuretics & ACEIs)
2- Decreased absorption of potassium :
   a. Cation-exchange resins : e.g.: kayexalate 20 gm t.d.s. in sorbitol solution.
3- Increased excretion of potassium :
   a. Diuretics e.g.: frusemide.
   b. Dialysis in severe cases.
4- Intra-cellular shift of potassium :
   a. Regular insulin 10 U with glucose 25% (as 10% solution) IV over 30 min.
   b. NaHCO3 50 – 100 mEq IV especially if acidosis is also present.
   c. B2-agonists. (e.g.: nebulized albuterol)
5- Counteract the effect of hyperkalaemia on the heart :
   a. Calcium gluconate 10 mL of 10% solution over 10 min.
      (1-3 ampoules could be given).

Acidosis [Metabolic & Respiratory]

Metabolic Acidosis

AE :

1- increased acid :
   a. **increased** acid **production** :
      i. *Endogenous* : ketoacidosis; diabetic, alchoholic & starvation & Lactic acidosis.
      ii. *Exogenous* : ingestion of salicylates, methyl alcohol, ammonium chloride
   b. **decreased** acid **excretion** : Acute & chronic renal failure.

2- decreased alkali :
   a. **GIT** causes :
      i. Severe diarrhea.
      ii. Pancreatic, biliary or intestinal fistula.
   b. **Renal** causes :
      i. Renal tubular acidosis.
      ii. Carbonic anhydrase inhibitors.
      iii. Spironolactone & amiloride.

C/P :

1- Kussmaul's breathing : hyperventilation or air-hunger.
2- Myocardial depression.
3- Drowsiness & impaired consciousness in severe cases.
4- Bone decalcification in chronic cases.

Investigations :

1- low blood pH.
2- Low HCO_3^- & $P.CO_2$
3- Investigations for the cause.

Treatment :

1- treatment of the cause.
2- $NaHCO_3$ orally or IV.

Respiratory acidosis

AE :

All causes of hypercapnic respiratory failure that occur due to hypoventilation. [refer to Chest]

C/P : Features of respiratory failure. [mention]

Investigations :

1) low blood pH.
2) high HCO_3^- & $P.CO_2$
3) Investigations for the cause.

Treatment : Treatment of respiratory failure.

Alkalosis [Metabolic & Respiratory]

Metabolic Alkalosis

AE :

1- increased intake of alkali :
- excessive treatment with bicarbonate.
- Milk-alkali syndrome.

2- loss of acid :
- GIT loss. [vomiting & gastric drainage]
- Renal loss :
  * diuretics especially loop diuretics and thiazides.
  * Conn's syndrome & Cushing's syndrome

3- hypokalaemia.

C/P :

1- hypoventilation. (shallow slow breathing)
2- tetany.
3- manifestations of hypokalaemia may occur.

Investigations :

1- high blood pH.
2- high HCO_3^- & $P.CO_2$
3- Investigations for the cause.

Treatment :

1- of the cause.
2- In severe cases : dilute HCl or arginine HCl may be given IV.
3- Correction of hypokalaemia.

Respiratory Alkalosis

AE : All causes of hyperventilation; e.g.:
- hyperventilation syndrome.
- Head injury, heat stroke & encephalitis.
- Pneumonia, pulmonary embolism & heart failure.

C/P :

1- Hyperventilation.
2- Parasthesia, light-headedness & rarely syncope.
3- Tetany in severe cases.

Investigations :

1- High blood pH.
2- Low HCO_3^- & $P.CO_2$
3- Investigations for the cause.

Treatment :

1- of the cause.
2- Breathing in a paper bag.
3- Sedation may be needed in severe anxiety.

Hyponatraemia [True & dilutional]

True hyponatraemia (Na^+ depletion) in Dehydration

Dehydration : loss of H_2O, Na^+ & Cl^-

AE :

1- GIT : vomiting, diarrhea & fisulae.
2- Renal : uncontrolled D.M., DKA, CRF with polyuria, diuretic phase of ARF & in Addison's disease.
3- Skin : ↑↑ sweating : as in miners and burns.

C/P : ↓↓ ECW results in :

- the skin loses its natural turgor, with sunken-eyes.
- ↓ BP, ↑ HR ± ↑ temp.
- may lead to circulatory failure, ARF or even death.
- If H_2O drinking continues → ↑ E.C. fluid and becomes hypotonic → intra-cellular over-hydration → drowsiness, restlessness, apathy, headache, nausea, vomiting plus muscle cramps develop (miners' cramps)

Treatment :

- IV saline + treatment of the cause.
- If big amounts of saline are needed, add Na-lactate or NaHCO3

N.B. : 5% glucose is dangerous as it increases I.C. over-hydration.

Hypervolaemia with dilutional hyponatraemia
(Over-hydration or Water-intoxication)

AE :

1- Acute renal failure especially if excessive fluids are given.
2- Post-operative & post-traumatic states.
3- Syndrome of inappropriate secretion of ADH [SIADH] :
   * Paramalignant e.g.: bronchogenic carcinoma & lymphoma.
   * CNS lesions e.g.: tumor, trauma & infections.
   * Chest diseases e.g.: tumor, pneumonia & T.B.
4- Drugs e.g.: cyclophosphamide.
5- Oedematous conditions (e.g.: Heart-failure, constrictive peri-carditis, cirrhosis) especially with diuretic therapy.

C/P :

- Dizziness, weakness, headache & vomiting.
- Confusion, muscle twitches, convulsions, coma & finally death.

Treatment :

1- Fluid restriction.
2- Hypertonic saline IV in severe cases with frusemide.
3- Drugs inhibiting action of ADH on the kidney e.g.: demeclocycline.
4- Treatment of the cause if possible.

	Plasma HCO_3^-	PCO_2	Bl. pH	Plasma Cl^-	Urine
M. acidosis:	↓	↓	↓	↑	Acidic with ↓ HCO_3 & ↑ ammonia.
M. alkalosis:	↑	↑	↑	↓	Alkaline ē ↑ HCO_3 & ↓ ammonia.
Resp. acidosis:	↑	↑	↓	↓	Acidic with ↓ HCO_3 & ↑ ammonia.
Resp. alkalosis:	↓	↓	↑	↑	Alkaline ē ↑ HCO_3 & ↓ ammonia.