

- Fasting causes mobilization of fat stores
- Stressed patients — Proteolysis causes muscle wasting

Measurement of Organ Function

- Malnutrition can lead to impaired muscle strength which in turn leads to increased fatigability of small muscles especially of the hand.
- Impaired muscle function is measured by *Hand Grip Dynamometry*
 - Measured in non-dominant hand
 - Highest value of three readings is taken
 - Force-frequency characteristics of muscles and rate of recovery from fatigue after electrical stimulation of the ulnar nerve is measured
- Bioelectric impedance analysis measures adipose reserve, intracellular and extracellular water and third space fluid in a stable surgical patients
- **Indirect calorimetric tests:** Measures oxygen consumption, respiratory quotient

Energy

- It is the loss of cutaneous responses to antigens, e.g. PPD, Candida
- Total leukocyte count ($<1800/\text{mm}^3$) is seen in malnutrition
- Decreased level of
 - S. Albumin ($t_{1/2}$ of 14–18 days) $< 3 \text{ g/dL}$. As serum albumin has long half-life its level falls late in malnutrition
 - S. Transferrin ($t_{1/2}$ of 7 days) $< 150 \text{ m-mol/L}$. It can be elevated in iron deficiency irrespective of nutritional status
 - S. Pre-albumin ($t_{1/2}$ of 3–5 days) $< 12 \text{ mg/dL}$. *It is the first to fall even in early stage of malnutrition. It is useful in ICU patients and should only be used if creatinine clearance is more than 50 ml per minute as levels of pre-albumin may be raised in renal failure inspite of poor nutritional status.*
 - S. Retinol binding protein ($t_{1/2}$ of 12 hours). Retinol binding proteins are bound to pre-albumin in circulation
 - Insulin like Growth Factor-1 (IGF-1)
 - Iron, calcium, magnesium, folic acid, vitamin B_{12} , 25-OH vitamin D
- Specific functional defects like increased prothrombin time

Urinary Creatinine Height Index

- It is measured by following formula:

$$\frac{\text{Actual 24 hours urinary creatinine excretion}}{\text{Ideal urinary creatinine excretion as per height}}$$

- Creatinine excretion is an indicator of muscle mass and total body nitrogen
- Value of urinary creatinine height index less than 80 suggests malnutrition

Requirements for Nutritional Support

- Adequate energy
- Protein nitrogen
- Water and electrolytes
- Trace elements
- Vitamins

Energy requirements

- 25–35 kcal per kg of body weight/day
- Should not exceed more than 40 kcal/kg/day

Energy Sources

- Hypertonic sugar
 - Glucose 10–50%
 - Fructose 15%
 - Insulin may be required when high concentration of glucose is used
- Fat emulsion
 - 1–2 grams/kg body weight/day
 - Given as intra-lipid 10% or 20%
 - Soyabean oil emulsion is commonly used which also contains egg lecithin
 - Glycerol is added to make it isotonic
 - 50% of the fatty acid content is linoleic acid
 - 500 units of heparin is used to facilitate fat utilization.

Nitrogen Source

- To meet substrate requirements for protein synthesis, the non-protein: nitrogen ratio of 150:1 should be maintained. This is required to prevent proteins from being used up to meet the caloric requirements
- Approximately 0.25 to 0.35 g of nitrogen per kilogram of body weight should be provided daily (to be adjusted depending on renal and hepatic function)

- ◊ Normal or raised ACTH levels are seen in ACTH-dependent Cushing's syndrome
- CT scan abdomen/chest – detects adrenal adenoma and adrenocortical carcinoma. May help to detect peripheral source of ACTH.
- MRI pituitary fossa – detects pituitary microadenomas that are more than 6 mm.
- High-dose dexamethasone suppression test – 2 mg dexamethasone q6 hourly given for 48 hours and then urinary or serum cortisol is measured. Suppression by high dose dexamethasone suggests pituitary microadenoma, non-suppression suggests peripheral source of ACTH (like bronchogenic tumor). Dexamethasone is chosen because it does not cross-react with present biochemical assays for cortisol.
- Bilateral inferior petrosal sinus ACTH sampling with CRF stimulation – study done if in case of ACTH-dependent Cushing's syndrome, the MRI fails to detect microadenoma.

Management

- Perioperative glucocorticoid administration is necessary to prevent the adrenal crisis.
- For patients undergoing adrenalectomy for adrenal adenoma or adrenocortical carcinoma,
 - Hydrocortisone 100 mg q8 hourly for 24 hours post-operatively is given. The steroid is then tapered slowly over weeks.
 - Perioperative antibiotics for 24 hours are given as these patients are prone to surgical site infection.
- For patients undergoing pituitary surgery (through transnasal transsphenoidal approach)
 - Steroids are withheld for first 48 hours. Subnormal serum cortisol estimation on post-op day 1 or 2 is suggestive of cure. The glucocorticoid supplementation is then started for usually 6 months, to give time to HPA axis to recover.
 - If the post-op cortisol levels remain high, repeat pituitary surgery, radiation to pituitary fossa or bilateral laparoscopic adrenalectomy remain options.

Subclinical Cushing's Syndrome

- It is characterized by
 - Incidentaloma

- Biochemical evidence of hypercortisolism
- No typical clinical evidence of Cushing's syndrome
- Natural history is not known
- The patients are at risk of hypertension, dyslipidemia, and impaired glucose tolerance
- Surgical therapy does not show consistent improvement
- Therapy is primarily medical.

Adrenocortical Carcinoma

- Rare – incidence 1:1,000,000
- No gender predilection
- Mostly 40 to 50 years old, minor peak at 5 years age
- Presentation
 - More than half of them are functional tumors and present with Cushing's syndrome or virilization
 - Mean tumor size at presentation is 9–12 cm
- Diagnosis
 - CT scan shows a heterogeneous mass with irregular/indistinct borders, central necrosis, and invasion of adjacent structures.
 - Large tumors, esp. right sided, may have intravascular extension into inferior vena cava or right heart
 - Metastases to the lymph nodes, liver, and lungs may be found.
- Treatment
 - Radical open surgery — En-bloc resection of the tumor with adjacent organs and lymph nodes. Patients with vascular tumor thrombus may need cardiopulmonary bypass to prevent tumor thromboembolism.
 - Chemotherapy — It is a relatively chemoresistant tumor. Only mitotane (o,p-DDD, or 1,1-dichloro-2-[o-chlorophenyl]-2-[p-chlorophenyl] ethane), a derivative of the insecticide DDT, has shown some efficacy in adjuvant/neoadjuvant settings.
- Prognosis
 - Overall adrenocortical carcinoma is associated with poor survival with 5 years survival reported between 15–20% range.
 - Patients who undergo incomplete resection of adrenocortical carcinomas have extremely limited life expectancy (median survival, < 1 year).

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Bladder Outflow Obstruction (BOO)

BOO is a urodynamic concept and the diagnosis is based upon:

- **Low flow rate:** Peak urinary flow < 10 ml/second for a voided volume > 200 ml at least on two occasions (Fig. 42.1)
- High voiding pressure > 80 cm of water (normally it is < 60 cm of water).

Pathogenesis

BOO can result from:

- Detrusor instability
- Neurological dysfunction
- Weak bladder contractions

The common disease entities resulting in bladder outlet obstructions are:

- Benign hyperplasia of the prostate
- Bladder neck stenosis
- Bladder neck hypertrophy
- Prostate cancer
- Urethral stricture
- Neuropathic bladder with sphincter dys-synergia.

Complications

- Acute retention
- Chronic retention with upper tract damage
- Impaired emptying resulting in stasis and infections
- Bladder calculi

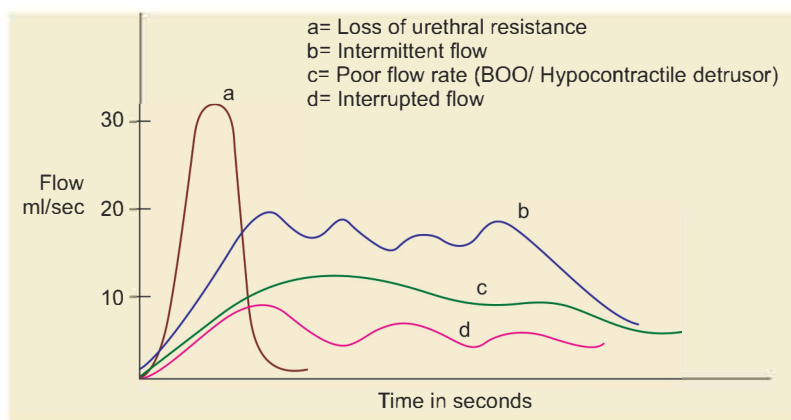


Fig. 42.1: Uroflowmetry patterns

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Voiding Dysfunction

Includes Problems of

- Bladder emptying
 - Obstructive conditions, e.g. benign prostatic hyperplasia (BPH)
- Bladder storage
 - Overactive bladder
 - Stress incontinence
 - Mixed incontinence
 - Overflow incontinence

Overactive Bladder

Present with

- Frequency
- Urgency
- Urge incontinence

Pathophysiology of urinary incontinence:

- Due to pelvic floor weakness with normal sphincteric mechanism (Stress incontinence)
- Due to detrusor instability with normal anatomy of pelvis floor and normal sphincter (True incontinence)
- Neuropathic incontinence due to nerve lesions
- Congenital incontinence due to ectopic ureter, duplicate or single system, with epispadias or exstrophy.
- False incontinence as a result of obstructive lesions (overflow incontinence).
- Traumatic

Urinary Incontinence

- **Definition:** Urinary incontinence is uncontrolled leakage of urine causing hygienic and social problems.
- Occurs mostly in older adults, more common in females.

Stress Incontinence

- Stress incontinence occurs when a small amount of urine escapes while the person coughs, sneezes, laughs, jumps or lifts something heavy.
- Occurs due to weakness in external urethra sphincter, mostly in women. Main pathology is hypermobility of vesicourethral segment, pelvic floor weakness and intrinsic sphincter deficiency.

Urge Incontinence

- Associated with sudden strong desire to pass urine
- Often results from detrusor overactivity.

Overflow Incontinence

- Overflow incontinence happens when urine leaks from an overfilled bladder.
- Occurs due to detrusor failure.

Mixed Incontinence

- Mixed incontinence occurs when a person has both the symptoms of urge incontinence and stress incontinence.