M.P. ANAESTHESIOLOGY

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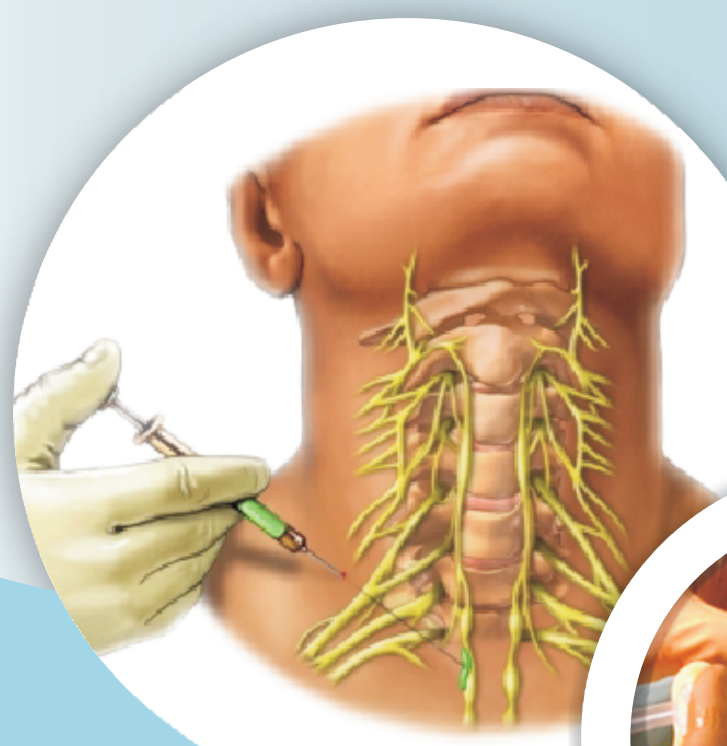

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FROM THE DESK OF THE PRESIDENT

Dear Friends,

Greetings and wish you all a very happy new year.

First and foremost I would like to thank all of you for entrusting me this responsibility of the President M.P. State 2019-2020. I will strive hard to achieve the goals set by our predecessors.

The Vision 2020 mission of ISA is :

- Safe Anaesthesia, Safe country
- Innovation and Research
- International Exchange Program
- Professional well being

The ISA National is doing an excellent job under the leadership of President Dr. Muralidhar Joshi and Secretary Dr. Naveen Malhotra, it is our duty to contribute our share to the Society.

This M.P. State Chapter journal took birth in the form of a small newsletter under the name of "Nishchetana" in the year2003 under the guidance of Dr. W.P. Thatte a legendarily figure in the field of Anaesthesia. It slowly became a journal for the Indore city branch and then the journal of the M.P. State Chapter. I hope the new team would take this journal to new heights and get it indexed so that it will serve as a good platform for publication of the postgraduates.

Our aim this year should be enrollment of maximum number of anesthesiologist as Life members, formation of more city branches, starting an M.P. P.G. Refresher course (which is need of the time for the post graduates) and lot of educational and public awareness activities under the banner of ISA.

I hope with the guidance of seniors and cooperation of all we will be able to achieve this with flying colours.

Long Live ISA

THANKING YOU

With warm regards

SECRETARY'S MESSAGE

I am honoured and privileged to shoulder the role of Secretary of the ISA MP State Chapter for last three years.

From its inception till now, AORA ISA has relentlessly contributed in training and education.

As an organization, ISA MP Chapter has expanded and continues to grow. ISA MP has created strong education programs, hosted workshops and conferences that bring together the best minds in Anaesthesia. After a wealth of achievements, however, a great deal of work still needs to be done.

My Vision is

To hold periodically Conferences / Workshops / CME in various cities for the betterment of Anaesthesiology.

To co-operate with other medical scientific associations.

To encourage clinical and scientific research in Anaesthesiology.

To hold scientific discussions in Anaesthesiology.

But in the present Corona Pandemic we are unable to organize conferences and workshops , so to fulfill the vision ISA MP chapter is planning to hold webinar for all members of MP state.

Before I conclude, I must admit that the real strength are the dedicated and enthusiastic life members!!

I would love to hear from you all if you have particular suggestion.

Stay at Home, wear Mask, keep social distancing and wash hand frequently.

Warm Regards,

● Dr. Jitendra Agrawal

EDITOR'S MESSAGE

"The secret of getting ahead is getting started. The secret of getting started is breaking your complex overwhelming tasks into manageable tasks, and then starting on the first one". – Mark Twain

Welcome to the first issue of MP journal of anaesthesiology for 2020. Amidst the havoc created by Novel Corona virus, it became difficult to publish the journal and therefore, we apologize for the delay. Nevertheless, we assure you it won't be repeated in the near future.

This issue could not have seen the light of day without the timely contributions from the authors, the expertise and constructive criticisms of our expert reviewers. It is the final product of the virtuosity of the authors, reviewers and the expertise. We are thankful to each and every participant who has contributed for this issue. And we hope, there are many more who contribute for the same.

The next issue lined up has "Covid-19" as its theme. The global pandemic has become a part and parcel of our life and we have been managing the patients as frontline warriors. Hence, we request the readers and members to share their experiences regarding the covid patients.

Jai Hind!

Long live ISA!!

● **Dr. Sadhana Sanwatsarkar**

Editor

MP-ISA

DIABETES AND IT'S ANAESTHETIC IMPLICATIONS

● Dr. Radhika Dua[1]

Abstract: Currently there are 35 million diabetics in India. By 2050, this number is expected to swell to 70 million, which means that a big chunk of patients coming in for surgery will require tailored anaesthetic management. Our aim would be to optimise chronic complications of the disease,prior to surgery, pevent the development of acute complications; all the whole maintaining blood glucose in a relatively narrow range without fluctuations to either extreme. Understanding glucose metabolism and the various drugs used to maintain a 'normal' blood sugar is a crucial part of it. It is vitally important to have an institutional protocol for insulin infusion ,to have a seamless transition between ward , theatres and intensive care admissiom, if rquired. Finally, it is desirable to have the patient back on presurgery dose schedule before discharge.

GLUCOSE METABOLISM AND REGULATION

Based on our present knowledge, glucose homeostasis is governed by the interplay of insulin,glucagon, amylin and incretin hormnes. These hormones are designed to maintain the circulating glucose concentrations in a relatively narrow range. Blood glucose levels are maintained through three mechanisms:

1. Intestinal absorption during fed state
2. Glycogenolysis
3. Gluconeogenesis

In the fasting state, blood glucose is being utilised at a constant rate,which has to be replaced. During the first 8- 12 hours, the main source of blood glucose is hepatic glycogenolysis, which is stimulated by glucagon. More prolonged fasting leads to hepatic gluconeogenesis from amino acids, which is regulated by insulin. [1-4]

In diabetic individuals in the fasting state,the same mechanism occurs. However, exogenous administered insulin is not able to properly regulate hepatic glucose output because it does not go into portal circulation. As also, in the fed state it is ineffective in suppressing glucagon secretion through the paracrine route resulting in an elevated hepatic glucose output.

The discovery of amylin, which is coexpressed and cosecreted by B cells, has made the intricacies of glucose metabolism clearer. It has been shown that it complements the effects of insulin on plasma glucose concentrations. [5]

Additionally the role of gut peptides must be covered. It has been shown that ingested food causes more potent release

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of insulin than glucose administered intravenously,which is termed the incretin effect. The dominant incretin hormone is GLP[1, 6]

CLASSIFICATION OF DIABETES 7

1 Autoimmune destruction of B cells leading to absolute insulin deficiency.

2 Loss of insulin secretion by B cells in the background of insulin resistance

3 Gestational diabetes is diagnosed in 2nd or 3rd trimester that was not clearly overt diabtes prior to gestation.

4 Specific types of diabetes due to other causes such as monogenic diabetes syndrome, disease of exocrine pancreas, drug induced etc.

WHO DIAGNOSTIC CRITERIA

Fasting plasma glucose >=126mg/dl or

2 hour plasma glucose >= 200 mg/dl during 75 gm oral glucose tolerance test Or

HbA1C > 6.5 % Or

Random plasma glucose >= 200mg/dl in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis.

PREDIABETES/IMPAIRED FASTING GLUCOSE

Fasting plasma glucose between 100 to 125 mg/dl Or

2 hr plasma glucose between 140 and 199 mg/dl. Or

HbA1C between 5.7 and 6.4%

PATHOPHYSIOLOGY

In type 1 diabetes there is deficiency of insulin and amylin. Rise in glucose occurs due to loss of insulin regulation

In type 2 DM there is abnormal B cells action and peripheral insulin resistance

PHARMACOTHERAPY

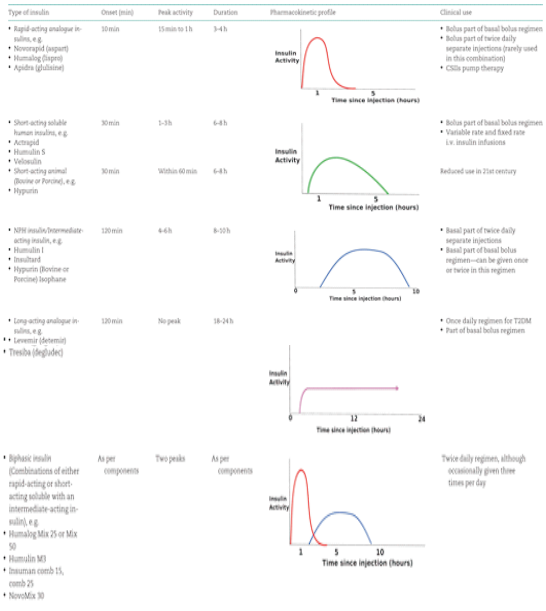
Diabetes pharmacotherapy includes

1. INSULIN

2. NON INSULIN GLUCOSE LOWERING AGENTS

INSULIN

There are several types of insulin commercially available. They can be classified into short acting insulins and background insulins. Table 1 summarizes the different insulins and pharmacokinetics. [8]



1. Short acting insulin:

There are three types of short acting insulins: they are usually administered to control glucose excursions during meals as part of basal bolus regimen

a) Very rapid acting insulin analogues e.g. Insulin aspart, insulin lispro, insulin glulisine. They have a small number of amino acid substitutions to the human insulin molecule, thus able to be absorbed faster. These are recommended by Joint

British Diabetes Society (JBDS) and The Association of Anaesthetists of Great Britain and Ireland(AAGBI) to treat transient hyperglycemia in the surgical patient.[9, 10]

b) Human soluble insulin e.g. Actrapid, humulins

These insulin are exactly the same as human insulin. These are recommended to be used in fixed rate and variable rate i v insulin infusion.

C) Animal soluble insulin(rarely used nowadays)

2. Background insulin

These insulins provide a steady systemic concentration,to promote cellular glucose uptake.

a) Intermediate acting insulin e.g. NPH insulin, HUMULIN I. They have a delayed onset of action at 90-120 minutes after s.c injection due to the addition of a protamine molecule.

b) Long acting insulin analogies e.g. Insulin glargine, insulin detemir, insulin degludec. They take 2-3 days to reach a steady state, and are usually administered as a once daily injection.

INSULIN REGIMENS

There are several regimens that can be used ,to mimic normal physiological concentrations of insulin.The more commonly used regimens are listed here .

1) Once daily

This regimen is commonly used in type 2 diabetics where once daily long acting insulin analogue is injected in addition to the oral medication to prevent hyperglycemia associated with hepatic neoglucogenesis.

2) Twice daily

Premixed insulin is injected at breakfast and evening meal.

3) Basal bolus

In this regimen long acting /intermediate acting insulin is given at night and three doses of very rapid acting insulin analogies given at mealtimes.

4) continuous subcutaneous insulin infusion(CSII)

The pump delivers a fixed hourly basal rate of a Very rapid acting insulin analogue ,the rate of which can be changed hourly. In addition, blouses are given to cover meals.

NON INSULIN GLUCOSE LOWERING DRUGS

All these drugs work essentially via four different mechanisms:

1) Increase the release of endogenous insulin (sulphonylureas and meglitinides)
2) Affect the G I absorption and renal reabsorption of glucose(intestinal alpha glucosidase inhibitors and SGLT2 inhibitors)
3) Drugs that alter the sensitivity to endogenous insulin and reduce glycogenolysis/Gluconeogenesis(metformin and Thiazolidinediones)
4) Drugs acting on Incretin pathway(GLP 1 analoghe's and DPP4 inhibitors).[11]

The pertinent aspects of various drugs are described below:

1) sulphoylureas e.g. Glibenclamide, gliclazide, glimepiride, glipizide, tolbutamide.

These agents are insulin secretagogues.These rely on adequate B cells function, also increased risk of

hypoglycemia. These drugs are discouraged in elderly and renally impaired progressively for hepatically metabolized gliclazide. Their long term use has been associated with deterioration of glycolic control, weight gain and risk of cardiovascular complications. Due to risk of hypoglycemia, these are to be omitted preoperatively.

2) Meglitinides e.g. Nateglinide and Repaglinide. These are shorter acting insulin secretagogues; require more frequent dosing; also to be omitted preoperatively.
3) Intestinal alpha glucosidase inhibitors e.g. Acarbose . This drug inhibits absorption of monosacchrides from the gut. Since in the preoperative fasting state there will be no monosacchrides to be absorbed ; this drug maybe discontinued in the perioperative period.
4) SGLT 2 inhibitors e.g. Dapagliflozin, canagliflozin, empagliflozin.These are a class of drugs which prevent glucose reabsorption from the proximal convoluted tubules. It is advised that these drugs be stopped preoperatively as they have been associated with DKA.
5) Biguanides e.g. Metformin. This is a first line treatment for type 2 diabetics. It inhibits flushing signalling leading to a reduction in glycogenolysis and endogenous glucose production.It also increased insulin receptor expression leading to increased insulin sensitivity. It is associated with the maximum HbA1c reduction. The dreaded complication of lactic acidosis is rate and most frequent in patients with renal failure of poor peripheral perfusion such as heart failure. Earlier it was recommended to stop 48 hours before surgery , however, the JBDS and AAGBI recommend it's continuation in elective surgeries with short fasting times, if no other risk factors for acute kidney injury are present.
6) Thiazolidinediones e.g. Pioglitazone; increased insulin sensitivity. The use of this drug is reducing because of it association with cardiovascular complications, macular edema and bladder cancer.
7) GLP 1 Analogues e.g. Exenatide, dulaglutide, liraglutide, lixisenatide. These peptides are subcutaneously administered and stimulate insulin release in response to food, reduce hepatic Gluconeogenesis, reduce gastric emptying and promote satiety.
8) Dipeptidyl peptidase 4 inhibitors(DPP4 inhibitors) e.g.alogliptin, linagliptin, saxagliptin, sitagliptin, vildagliptin. These inhibits degradation of GLP 1 INHIBITORS by DPP 4 .

SURGICAL STRESS AND ANAESTHESIA

The stress of surgery and anaesthesia leads to a surge of counterregulatory hormones such as glucagon, cortisol, catecholamines and growth hormone, producing what is termed stress induced hyperglycemia. This is defined by the American Diabetes Association as transient hyperglycemia in a previously non diabetic patient during an acute illness or invasive procedure. It is characterised by plasma glucose> 180mg/dl with levels returning to normal after removal of the

stressor.

RISKS OF POOR GLUCOSE CONTROL

A substantial body of literature exists which demonstrates a clear association between perioperative hyperglycemia and adverse clinical outcomes. Some studies have shown that there is greater than 50% increase in mortality. There maybe 2- 4 times increase in postoperative respiratory infections and nearly twice the risk of surgical site infection. Other complications such as acute kidney injury and myocardial infarction are also increased in poorly controlled diabetics. 12-15

On the other side of the spectrum, hypoglycemia is equally detrimental to a good outcome. It is associated with increased mortality, neurocognitive defects, seizures, myocardial ischemia.

AIMS OF PREOPERATIVE ASSESSMENT

1) Optimisation of glycemic control(to aim for HbA1c < 8.5%)
2) Recognition and optimisation of comorbodities
3) Tailored glucose management plan and ICU admission for high risk patients
4) Identification of a difficult airway

PREOPERATIVE ASSESSMENT

The preoperative assessment should focus on identifying and optimising the chronic complications of diabetes , in addition to the usual history taking. The history and examination must include specific questions pertaining to the CNS such as peripheral neuropathy, TIA, stroke.

The cardiovascular system ,perhaps requires the most attention ,as about 75% diabetics die of atherosclerotic complications. The incidence of silent myocardial ischemia is 30- 50%. It is recommended to investigate for SMI if Lee Index score>= 2 and METS < 4. TESTING for autonomic neuropathy should be carried out in all type 2 diabetics and type 1 diabetes with disease duration of 5 years or more.if autonomic neuropathy is detected and If QT interval is prolonged, then 24 hour ECG monitoring is advised to look for ectopic ventricular beats.

Diabetic neuropathy is the most common cause of end stage renal failure. Therefore detection is even more significant. Nephrotoxic drugs must be avoided and the mean arterial pressure should be maintained between 60-70 mms Hg.

Gastroparesis occurs in about 30-50% of either types of diabetes.It can be detected by history of anorexia,nausea, vomiting, abdominal pain and bloaring. The gold standard for diagnosis is gastric scintigraphy.In the absence of definitive test the patient should be given prokinetic agents and a rapid sequence induction of anaesthesia should be carried out.

The airway examination assumes particular importance because hyperglycemia leads to glycosylation of collagen in the cervical and temporomandibular joints leading to stiffness as possible difficult airway. The palm print test is the most sensitive test to detect the presence of a difficult airway. Other sign includes the prayer sign.16,17

PERIOPERATIVE MANAGEMENT GOALS

These include reduction of patient morbidity and mortality,which are achieved by avoiding hyper or

hypoglycemia by keeping the blood glucose below the target of <180mg/dl, maintenance of fluid and electrolyte balance and prevention of ketosis. [18,19]

The glycemic target in the preoperative level has been recommended by several societies to be < 180mg/dl. This value resulted from the NICE- SUGAR trial which showed that intensive glycemic control(81-108 mg/dl) was associated with higher mortality. A subsequent follow up publication demonstrates that the excess mortality was due to hypoglycemia.

Choice of anaesthetic technique depends on the surgery; no study has consistently shown benefit of general anaesthesia or regional anaesthesia over the other.

PREOPERATIVE INSULIN DOSING ADJUSTMENT

For type I diabetics who are obligate insulin dependent ,it must not be stopped perioperatively.

On the day before surgery, all the usual insulin should be given per the dose, except for the evening dose of long acting insulin which should be reduced by 20%.

Insulin regimens on the day of surgery are slightly more challenging.80% of the usual dose of long acting insulin should be administered. For intermediate acting insulins, 50% of the dose to be given if blood glucose is > 120 mg/dl . If blood glucose< 120 mg/dl it should be withheld. The short acting insulin should be withheld on the morning of the surgery. For premixed insulins , total insulin dose is added and 1/2 of it to be given as long acting, the night prior , skip the morning dose and decide insulin dosing based on blood glucose.

INTRAOPERATIVE INSULIN REGIMENS

Intraoperative glucose control is done most perfectly with intravenous insulin infusion. I V insulin infusion can be either fixed rate of variable rate. Fixed Rate of Insulin Infusion (FRIII) is usually used only in diabetic ketoacidosis or hyperglycemia hyperosmolar state. For preoperative management, Variable Rate iv insulin Infusion (VRIII) is preferred. There are several different protocols for VRIII ,which are based on the first such protocol called the Alberti regimen. The important thing to note is that there should be a single protocol pan hospital, to avoid errors in dosing and consequently glucose variability and other complications. An example of a protocol is give below .[20]

Blood glucose	**Action**
< 80 mg/dl	Stop insulin infusion25 g of glucose Restart insulin 0.3-0.6U/he when BG >120mg/dl
80-120 mg/dl	Reduce insulin infusion by 0.3U/hr
120-180mg/dl	No change
180-220 mg/dl	Increase insulin infusion by 0.3U/hr
>220mg/dl	Increase insulin infusion by 0.5 U/hr

FLUID MANAGEMENT

The ideal fluid should have sufficient glucose to minimise metabolism and allow insulin infusion, should contain potassium and be isotopic with plasma. This can be achieved by adding 20mmo!/l of potassium to 5% dextrose in 0.45% saline. Of note, is the fact that lactate ringers is no

longer contraindicated as a fluid choice ,in the absence of severe renal impairment.

POSTOPERATIVE MANAGEMENT

Pain management is crucial because it release counterregulatory hormones thereby increasing blood glucose. Multimodal analgesia is preferred.

Prophylaxis of postoperative nausea and vomiting is desirable to prevent any electrolyte imbalance.

CHANGE OVER FROM IVII TO PREVIOUS REGIMENS

The changeover to sc insulin should occur after insulin levels have been stable for 24 hours and normal oral intake is resumed. Half the total of insulin dose should be given as long acting insulin at least half hour prior to stopping of infusion. The remaining half can be divided in three very rapid acting insulin analogues. In patients with type 1 diabetes ,resumption of basal bolus regimen is essential and a follow up consultation with an endocrinologist must be sought.

For Patients with type 2 diabetes , on non insulin glucose lowering agents, when the HbA1c is less than equal to 8%, previous drug treatment is resumed at same doses after 48 hours,with roaring of very rapid acting insulin analogues. If HbA1c is between 8-9% , the previous treatment is resumed; additionally long acting insulin analogue is added and follow up with a specialist is to be arranged.21,22

To summarise, the anaesthesiologists are uniquely placed to play a key role in patient optimisation and glycemic control.with universal protocols in place, patients can be safely managed from admission to discharge with good clinical outcomes.

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IMPLICATION OF THYROID DISORDERS ON ANAESTHESIA

● Dr. Abhimanyu Rana[1]

In India, there is a significant burden of thyroid diseases. According to a projection from various studies on thyroid disease, it has been estimated that about 42 million people in India suffer from thyroid diseases. Thus, the significance of in-depth knowledge about functioning of Thyroid is required in day to-day practice.

Anatomy

Thyroid is bilobular gland joined by isthmus, located at anterior aspect of neck between the medial border of sternocleidomastoid muscles.

It extends from C5 –T1 cartilage, below thyroid cartilage.

- 20g weight
- 2 lobes – at level of 2-4 tracheal cartilage
- 4 parathyroid glands
- **Arterial supply –** Superior & inferior thyroid Artery.
- **Venous drainage –**Superior, middle and inferior thyroid veins which form a venous plexus.
- **Blood flow -** 4-6g/ml/min – (which goes higher as the thyroid gland enlarges)
- **Innervation –** sympathetic nerve supply.

Superior thyroid artery is first branch of External Carotid Artery , it supplies the superior and anterior portions of the gland.

The inferior thyroid artery arises from the Thyrocervical trunk, it tends to supply the posteroinferior aspect of the gland.

Histology –

Thyroid is subdivided into irregular lobular units. Each lobule contains a cluster of follicles, which are the structural and functional units of the thyroid gland. Follicular cells produce Thyroglobulin (an iodine rich, inactive form of the thyroid hormones), which is then stored as colloid in the lumen of the follicles.

Parafollicular cells are a subtype of neuroendocrine cells (amine precursor uptake and de-carboxylation – AUPD – system) that produce **thyrocalcitonin** (calcitonin)

EMBRYOLOGY –

The thyroid originates from two main structures: the primitive pharynx and the neural crest. The rudimentary lateral thyroid develops from neural crest cells, while the median thyroid, which forms the bulk of the gland, arises from the primitive pharynx.

- Origin – 24 day
- **Site –** Foramen cecum.
- Primitive pharynx and neural crest
- **Descends –** Thyroglossal duct
- 7 week gestational age (GA)
- Secretion starts at 11 week GA
- **Parathyroid glands –** 3rd and 4th pharyngeal pouches.

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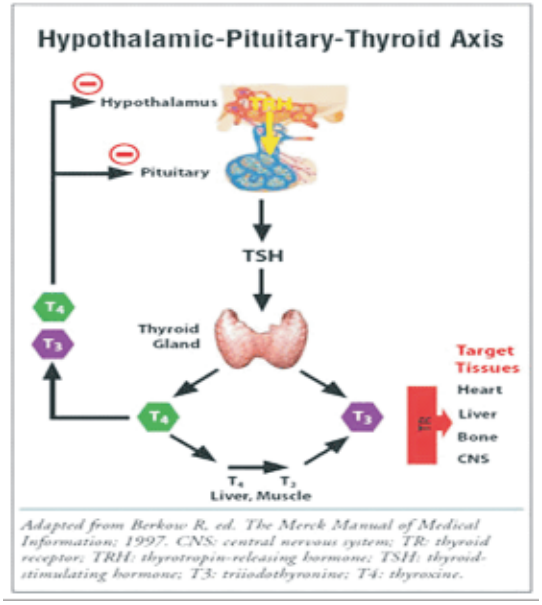
PHYSIOLOGY

The thyroid gland is part of the hypothalamic-pituitary-thyroid axis, and control of thyroid hormone secretion is exerted by classical negative feedback, as depicted in the diagram.

Thyroid-releasing hormone (TRH) from the hypothalamus stimulates TSH from the pituitary, which stimulates thyroid hormone release.



Adapted from Berkow R, ed. The Merck Manual of Medical Information; 1997. CNS: central nervous system; TR: thyroid receptor; TRH: thyrotropin-releasing hormone; TSH: thyroid-stimulating hormone; T3: triiodothyronine; T4: thyroxine.

TSH secretion increased by

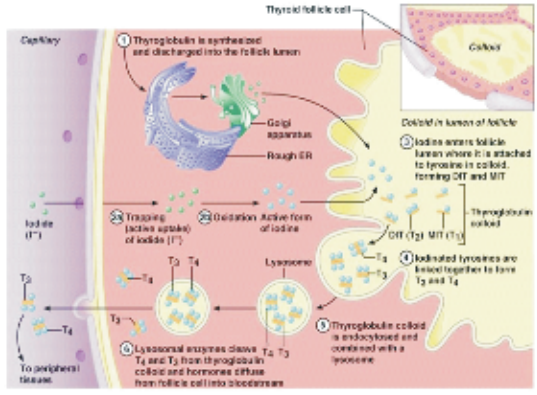
- TRH
- Cold (children)

TSH suppression is done by

- Glucocorticoids
- Somatostatin
- Dopamine

Metabolic step	Inhibitor
Iodine transport	SCN (Thio cyanate) ion
Iodisation	PTU
Coupling	PTU

SYNTHESIS OF THYROID HORMONES



HYPER THYROIDISM

- Causes-
- **Intrinsic –** MNG, toxic adenoma
- **TSH stimulation –** Grave's trophoblastic tumor
- Thyroiditis
- **Exogenous thyroid –** iatrogenic, iodine induced
- **Extrathyroidal source –** Struma ovari

Classification -

	T3 T4	TSH
Sub clinical	NORMAL	0.1 to 0.4
Overt hyperth-yroidism	Elevated	0.03

Drugs inducing Hyperthyroidism -

- Amiadarone – AIT1& AIT2
- Lithium
- Para –amionosalicylic acid
- Potassium iodide
- AMIODARONE - contains 37% iodine by weight.

Systemic toxicity due to Amiodarone

- Endocrine- Thyroid (35%)
- Cardiovascular (<10%)
- Respiratory (17%)

- Skin (75%)
- Hepatic (30%)

Treatment for -

Type I amiodarone-induced thyrotoxicosis- Anti thyroid drugs, thyroidectomy

Type II amiodarone-induced thyrotoxicosis – Prednisolone

Amiodarone induced hypothyroidism – thyroxine

CLINICAL FEATURES –

- Exophthalmos –

Grave' eye disease is a autoimmune Infiltrative orbitopathy.

TRAb – TSH Hormone Receptor Antibody

Eye signs –

- **Dalrymple's sign -** Eyelid retraction is the most common presenting sign of TED, present in upto 90% of patients.
- **Von Graefe's sign -** Lid lag of the upper eyelid on downward gaze and lid edema.
- **Stellwag sign -** infrequent or incomplete blinking. It is accompanied by Dalrymple's sign
- **Joffroy sign -** There is a lack of wrinkling of the forehead when a patient looks up with the head bent forwards.
- **Moebius sign -** an inability to maintain convergence of the eyes.It is found in patients with Graves' disease.

Respiratory system –

- Increase BMR
- Increased CO2 production – Hyperventilation
- **Airway obstruction -** If the obstruction is intrathoracic, flow limitation will show up as a plateau in flow during exhalation.

If it is extra-thoracic, flow limitation and a plateau will occur during inhalation.

Cardio vascular system -

- Systolic Hypertension
- Hyperdynamic cirulation
- Arrhythmias – most common AF
- Thyrotoxic cardiomyopathy – CCF (rare)

Sleeping pulse rate – if >80/min . in an adult indicates probability of thyrotoxicosis.

Dermatopathy –

The pathogenesis is due to expression of TSH- receptor antigen in the skin fibroblasts, triggering the auto-immune response. Usually associated with ophthalmopathy.

- Warm, smooth
- Diffuse alopecia
- Acropachy (swelling of distal digits with overgrown nail plates that may lift off the nail bed

Pigmentation – mostly on pre-tibial skin. Due to ACTH secretion.

ANTI THYROID MEDICATIONS -

HYPOTHYROIDISM –

Hypothyroidism may be caused by intrinsic thyroid disease or failure of the hypothalamo - pituitary axis.

CAUSES -

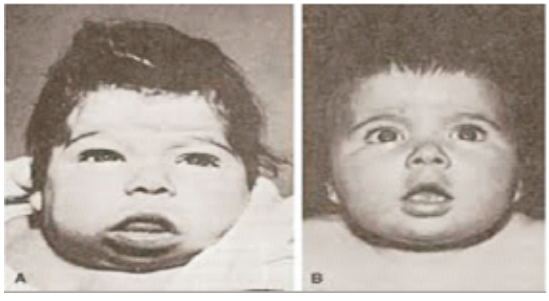
- Iodine deficiency.
- Iatrogenic- Radioactive Iodine therapy, anti thyroid drugs , Post thyroidectomy
- Hashimoto thyroiditis
- Sheehan' syndrome.

	T3 ,T4	TSH	Symptoms
Sub clinical	normal	<11	-
Mild	Normal	6-11	+
Moderate	Low	11-90	++
Severe	Low	>90	comatose

DRUG	DOSE	MECHANISM OF ACTION	SIDE EFFECTS
Carbimazole	Initial:15-40mg daily Maintenance: 5-15mg daily Takes 6-8 weeks to work	Prodrug rapidly converted to methimazole. Prevents synthesis of T3 and T4 by blocking oxidation of iodide to iodine and inhibiting thyroid peroxidase	Rashes, arthralgia, pruritis, myopathy. Bone marrow suppression Agranulocytosis (0.1%) Crosses placenta: foetal hypothyroidism
Propylthiouracil	Initial:200-400mg daily Maintenance: 50-150mg daily Takes 6-8 weeks	Blocks iodination of tyrosine residues present in thyroglobulin. Inhibits conversion of T4 – T3	Thrombocytopenia, Aplastic anaemia, Agranulocytosis Hepatitis, nephritis, Crosses placenta: foetal hypothyroidism
Iodide/Iodine	Lugol's solution: 5g Iodine solution in 10g Potassium iodide: 0.1-0.3ml TDS	Large doses of Iodide inhibit hormone production. Reduced the effect of TSH. Marked reduction in thyroid vascularity over 10-14days	Antithyroid effects diminish with time. Hypersensitivity reactions. Crosses placenta: foetal hypothyroidism
Propanolol	Oral: 40-80mg TDS (May need higher dose as metabolism increased) IV: 0.5mg titrated to effect	Controls sympathetic effects of thyrotoxic crisis. Blocks peripheral conversion of T4 to T3	Negative inotropy & chronotropy. Bronchospasm Poor peripheral circulation. CNS effects

- **Congenital hypothyroidism (CHT)** is a condition resulting from an absent or under-developed thyroid gland (dysgenesis) or one that has developed but cannot make thyroid hormone because of a 'production line' problem (dyshormonogenesis)

Sleepy and difficult to feed, prolonged jaundice (with an associated yellow skin) after birth. Other later symptoms may include constipation, low muscle tone (floppiness), cold extremities, and poor growth.



Myxedema facies – Myxedema describes a specific form of cutaneous and dermal oedema secondary to increased deposition of connective tissue components.

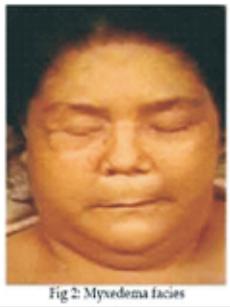


Fig 2: Myxedema facies

The features such as expressionless face, puffiness around the eyes and pallor.

- **Respiratory –**
- Difficult airway- (>5%) Diposition of mucopolysaccharides (MPS) inside the airways: leads to narrowing of airway, large tongue, and increased neck circumference.
- Deviation of trachea, difficulty in neck flexion causing difficult to align axes.
- CO_2 curve shifted to right.
- Maximum breathing capacity and diffusion capacity are decreased .
- Ventilation responses to hypoxia and hypercarbia is depressed.
- CVS

Bradycardia – due to deposition of MPS in conduction pathways.

Reduced myocardial contraction (decreased beta activity) - Decreased SBP, increased DBP (increased alpha action) leads to low cardiac output

These changes make them prone for CHF. More susceptible for hypotension effects of anaesthetic agents and neuraxial blocks.

- **GIT**

Delayed gastric emptying – Risk of aspiration is high.

- **CNS -**

Poor temperature regulation

Prone for hypothermia – more chances of re-curarization. Deep tendon reflexes demonstrate a prolonged relaxation phase.

CLINICAL METHODS TO EXAMINE THYROID GLAND –

- **Lahey's method -** Place the thumb against the lower lateral portion of the thyroid cartilage and push the trachea laterally.
- **Crile method –** thumb on the gland, patient is asked to swallow.
- **Pizillo method –** palpation using fingers of both hands, standing behind the patient.
- **Kocher's test –** scabbard trachea- pushing lateral lobes of thyroid will cause stridor. Common in Carcinoma thyroid and multi-nodular goitre.
- **Berry's sign –** Lack of carotid pulsation (carcinoma thyroid)
- **Pemberton's sign –** patient is asked to raise both arms over his head until they touch ears. This is maintained for a while leads to congestion of face and distress increases due to venous obstruction. Seen in retrosternal goitre.

Laboratory investigations –

- T4 – 4.5 – 12 mcg/ dL
- T3 – 80-180 ng/dL
- TSH - 0.4 -5 milunits/mL
- 80% T3 T4 bound to TBG , 10-15% to Albumin 0.1% free unbound form

Radiological investigation -

- **Ultrasonography –** to assess airway, nodules and size of thyroid.
- **Thyroid scan –** using I-123 or Tc 99 .
- Radioactive iodine uptake – 10-25%

Other investigations required for pre op assessment

- CBC
- Serum electrolytes and Serum Creatinine
- ECG
- X- ray - neck AP, Lateral
- CT scan
- Indirect laryngoscopy
- Serum calcium

Table 2
Advantages and disadvantages of treatment modalities for Graves' disease

Treatment Modality	Advantages	Disadvantages
Thionamides	No radiation hazard No surgical and anesthesiologic risk No permanent hypothyroidism Outpatient therapy	Recurrence rate high (>50%) Frequent testing required Common mild side effects Rare but potentially lethal side effects
Radioactive iodine	Definitive treatment of hyperthyroidism No surgical or anesthesiologic risk Outpatient therapy, rapidly performed Rapid control of hyperthyroidism in most Low cost Side effects mild, rare, and transient Normalizes thyroid size within 1 year	Potential radiation hazards Worsening of thyroid eye disease Adherence with radiation regulations Decreasing efficacy with increasing goiter size May need to be repeated Hypothyroidism eventually develops in most cases
Thyroidectomy	Definitive treatment of hyperthyroidism No radiation hazard Rapid normalization of thyroid dysfunction Definitive histology Most effective in cases with pressure symptoms	Cost Inpatient therapy Anesthesiologic risk[a] Hypoparathyroidism (1%–2%)[b] Damage to the recurrent laryngeal nerve (1%–2%)[b] Risk of bleeding, infection, unsatisfactory scarring[b] Hypothyroidism in most patients[b]

[a] Hyperthyroidism at time of surgery increases the risk associated with anesthesia.
[b] The surgical risk generally is higher for total thyroidectomy than for subtotal thyroidectomy. The risk of hypothyroidism and the risk of recurrence are inversely related and depend on the amount of thyroid tissue removed.

PRE OP OPTIMISATION

●Hyperthyroid –

i) Anti thyroid drugs – (6-8 weeks)

ii) Lugol's iodine – 3 drops q8h 14 days - 5% iodine in 10% KI(8mg iodide/drop)

iii)Beta blockers- propanolol, esmolol

iv) Lithium carbonate – 300 mg PO q6h

Wolf –Chaikoff effect - The Wolff–Chaikoff effect has been used as a treatment principle against hyperthyroidism by infusion of a large amount of iodine to suppress the thyroid gland.

Jod Basedow effect -_ Iodine-induced hyperthyroidism**.**

● Hypothyroid – Oral thyroxine

PRE –MEDICATION –

●**Hyperthyroid –** Beta blockers , anti thyroid drugs to be continued

Anxiolytic drugs .

Hypothyroid – Thyroxine to be continued Sedative pre-meds not preferred.

In patient with Obstructive Sleep Apnoea – sedative pre meds avoided.

● **Monitoring –**

- ECG lead II & V5
- SpO2
- NIBP/IBP – invasive monitoring in patients with rhythm irregularities and major hemodynamic shift.
- Temperature monitoring.
- EtCO2
- NMT

INDUCTION –

Cockpit drill check of the anaesthesia work station to be done. Difficult airway cart to be prepared thoroughly.

Esmolol, propranolol, hydrocortisone, propylthiouracil/ carbimazole, Nasogastric tube to be kept on the work station.

IV Induction –

Thiopentone is drug of choice .

Propofol is a good alternative .

Steal induction –

in pre anti thyroid era, patient was taken to Operation theatre as scheduled for Operation daily and brought out. Once patient is accustomed with OT, induction is done without informing the patient.

General Anaesthesia -

● Probability of difficult airway – Check ventilation and proceed.

● Anticipated difficult airway , H/o OSA –

i) Awake fiberoptic bronchoscope for intubation

ii) Tracheostomy.

Adequate depth to blunt the laryngoscopy response using lignocaine 1-1.5 mg/Kg , fentanyl dose 2mcg/Kg with adequate dose of muscle relaxant.

Types of ETT –

I. PVC

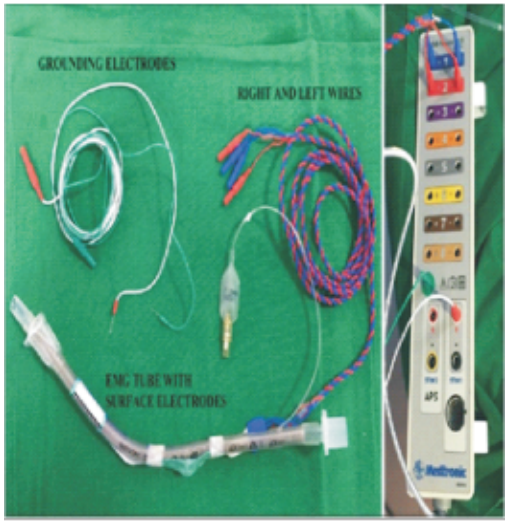
II. Armoured ETT/ Flexo-metallic ETT

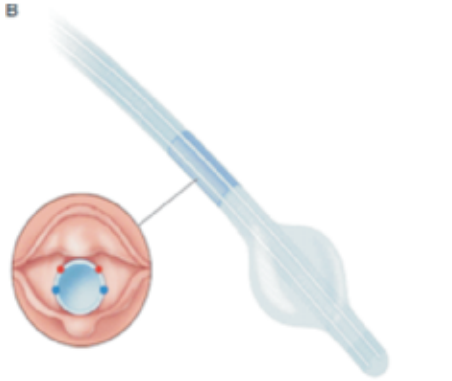
III. NORTH POLE ETT

IV. Neural Integrity Monitoring (NIM) ETT. –

ETT Placement to be done keeping the sensor strip should be placed in proximity to vocal cords.

To monitor the recurrent laryngeal nerve response. Prior to placement, the cuff of the NIM EMG tracheal tube should be covered with an aqueous lubricant rather than a local anesthetic gel. It is also advisable to use a low FiO2 given the associated potential for an electrical fire.





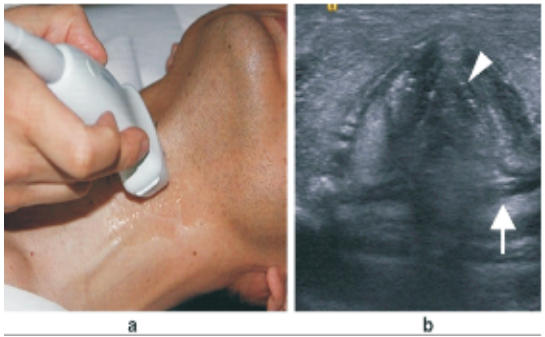
Use of Supra-glottic Airway Device –

Relative contraindications – tracheal deviation, large thyroid mass.

- Dysphagia and surgical incision pain scores were also significantly lower in the LMA group, compared with the ETT group, at 6 h and 12 h after surgery.
- The frequency of postoperative paracetamol consumption was significantly increased in the ETT group, compared with the LMA group

Extubation

- Difficult airway cart to be kept ready
- Cuff-Leak test – Defalte the ETT cuff and listen for the sound of air leak between Trachea and ETT. Absence of cuff leak suggests Airway edema.
- **Full reversal –** confirm with NMT.
- Vocal cord examination –
  - i. Direct Visualisation
  - ii. USG
- **Bailey' s manoeuvre -** for deep extubation. The placement of LMA before extubation when patient is in deep plane and allowing the patient to be awake with LMAA.



a b

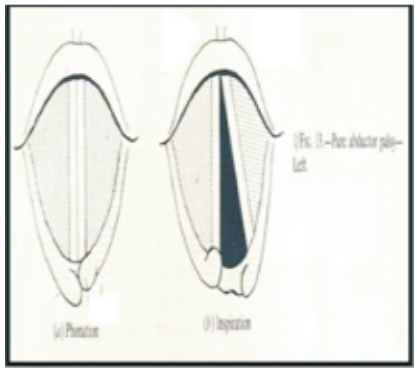
PERIOPERATIVE COMPLICATION -

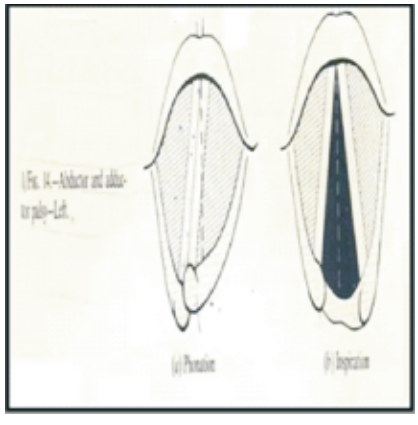
- **During positioning –** Extubation, ETT kinking
- **During maintenance –** kinking of tube
  - Hemorrhage
  - Dysrrhythmias
  - Air embolism
  - Thyroid storm

POST OPERATIVE COMPLICATIONS-

- **Immediate -** tracheomalacia, recurrent laryngeal nerve palsies, hypocalcemic tetany, laryngospasm
- **In the ward -** laryngeal edema and haematoma producing respiratory distress and hypocalcemic tetany (20%)
- **Delayed –** hypothyroidism.

NERVE PALSY -

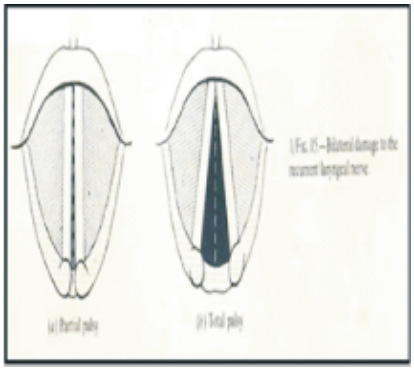


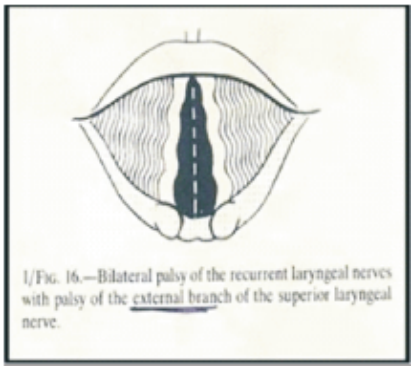


Injury to external laryngeal nerve can cause weakened phonation because the vocal folds cannot be tightened.

Injury to Unilateral Recurrent Laryngeal Nerves produces Hoarseness.

If B/L RLN are damaged the voice may or may not be preserved, but breathing distress is present. Tracheostomy is mandatory.





I/Fig. 16.—Bilateral palsy of the recurrent laryngeal nerves with palsy of the external branch of the superior laryngeal nerve.

HYPERTHYROIDISM IN PREGNANT PATIENT -

- Surgery can be performed safely in the second trimester.
- Propylthiouracil is the preferred antithyroid drug as it is higly protein bound and is secreted in the breast milk to a lesser extent.

BETA Blockers can be used for control of CVS symptoms.

ROLE OF REGIONAL ANAESTHESIA -

Cervical Epidural anaesthesia – risk of bradycardia, hypotension, phrenic nerve paralysis.

Cervical plexus block – Superficial & Deep cervial plexus block.

Superficial cervical plexus unilateral and bilateral has been used for thyroid surgeries . Deep cervical plexus block can be used only unilateral.

Contraindications – patient refusal

Coagulopathy

Massive thyroid gland – causing Respiratory obstruction.

OSA

PERI OPERATIVE COMPLICATIONS – THYROID STORM

How to identify during surgery

- Early exhaustion of soda lime, canister becoming very hot,
- hyperpyrexia,
- Increased ETCO2
- Patient requiring increased muscle relaxants and anaesthetic agents.
- unexplained tachycardia and dysrrythmias

GOALS OF MANAGEMENT

- Reduction of circulating thyroid hormone levels.
- Inhibition of the peripheral effects of circulating thyroid hormones
- Supportive care
- Treatment of the underlying precipitating event.

MANAGEMENT -

- 100% O2
- COLD SALINE – Infusion of cold saline / or Irrigation of Urinary bladder with Cold saline.
- Treat metabolic acidosis if pH is < 7.1 by sodium bicarbonate.
- Infusion of esmolol or propranolol .

- Dexamethasone 2mg every 6hrs or cortisol 100 -200mg every 8 hrs.
- Propylthiouracil 200-400mg every 8 hrs via Ryle's Tube.
- If shock – phenylephrine infusion
- Digoxin for CCF,
- Serum thyroid hormone levels generally return to normal within 24-48 hrs & recovery occurs within 1 week.

MYXEDEMA COMA -

- Patient presents in stupor or coma,
- Severe hypothermia, hypoventilation with hypercarbia, hypotension, CCF with severe bradycardia
- Often seen in elderly women.

Hyponatremia associated with SIADH may be seen

MANAGMENT -

- Inj Atropine 0.02mg/kg for bradycardia
- Controlled ventilation
- Warm IV Fluids, Warming blankets.
- Hyponatremia and Metabolic Acidosis to be corrected.
- Specific Therapy :
- Parenteral T4 : 100-200mcg IV bolus, followed by 50mcg IV Q6h
- Inj Hydrocortisone 100mg IV stat, 25mcg IV Q6h

If the pt has coronary artery disease, we should start with low dose of T4.

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SEPSIS - A COMPREHENSIVE REVIEW

● **Dr. Mayank Massand**[1]

DEFINITION- Derived from the ancient Greek word for rotten flesh and putrefaction. Evolution of definitions

1992-SIRS due to confirmed or suspected infectious process

SEVERE SEPSIS – Sepsis plus organ dysfunction

SEPTIC SHOCK-Sepsis induced hypotension persisting despite adequate fluid resuscitation

SIRS- TEMP>38*or <36*C, HR>90/MIN,RR>20MIN OR PACO2 <32,TLC>12000OR <400

SEPSIS- A life threatening organ dysfunction due to dysregulated host response to infection

ORGAN DYSFUNCTION- A change in total SOFA score of more than 2 points due to infection .Baseline score as 0 in pts with no pre-existing organ dysfunction .

Septic shock – Sepsis with persistent hypotension requiring vasopressors to maintain a MAP >65MMHG and serum lactate of more than 2mmol/Lt despite adequate volume resuscitation

SOFA SCORE – Resp, Liver , CVS, CNS ,coagulation ,renal each having score from 0to 4. SOFA SCORE for bed side monitoring

SYSTOLIC BP<100MMHG

ALTERED MENTATION

RR>22/MIN

2 OR MORE OF THE FINDINGS.

PIRO SYSTEM- 28 days mortality, predisposition, insult /infection, response, organ dysfunction

BACERIOLOGY AND SITES OF INFECTION

GRAM POSITIVE 25%

GRAM NEGATIVE 25%

GRAM POSITIVE PLUS GRAM NEGATIVE 25%

CULTURE NEGATIVE 25%

FUNGAL PATHOGENS 3TO5%

SITES OF INFECTION

LUNG , ABDOMINAL, URINARY TRACT MAINLY

PATHOPHYSIOLOGY OF SEPSIS

Interaction between macrophages and pathogens resulting in expression of various receptors like PRPS, finally PAMPS AND DAMS are expressed resulting in various presentations

CLINICAL MANIFESTATIONS

CVS – tachycardia, hypotension, vasodilatation, decreased contractility

RESP- Tachypnoea, ALI / ARDS, decreased PAO_2/FIO2 RATIO

HAEMATOLOGICAL-thrombocyto-penia, decreased protein C, increased D- dimer, increased PT/APTT

1. Consultant Anaesthesiology & Critical Care, Medanta Hospital, Indore

NEUROLOGICAL- confusion, agitation, cerebral oedema ,neuropathy

RENAL – oliguria, increased creatinine

LIVER – decrease in albumin, increase in enzymes

METABLOIC- AG acidosis, increased lactate, hyperglycemia , hypophosphatemia ,

Sometimes varied features in elderly as well in debilitated immuno compromised young people

DIAGNOSTIC CRITERIA

PCT most useful . PCT is more than 5ng/ml in gram negative infections. Predictor of outcome and trend of infection .

PRINCIPLES OF TREATMENT

Multiple trials have been done on sepsis management

Early goal directed therapy , PROCESS, ARISE , PROMISE

SURVIVAL SEPSIS CAMPAIGN 2018

Combining 3hrs to 6hrs bundle into 1hr bundle .

BUNDLES DEFINITION –

They are a group of therapies built around the best evidence based guidelines which when implemented together produce greater benefit in terms of outcome than the individual therapeutic interventions.

KEY ELEMENTS

Measure lactate levels , re-measure when the lactate levels were >2MMOL/LT

Obtain blood cultures prior to administration of antibiotics

Administer broad spectrum antibiotics

Rapid administration of crystalloid 30ml/kg for hypotension or lactate >4mmol/lt

Apply vasopressors if pt is hypotensive during or after fluid resuscitation to keep MAP >65MMHG

SOURCE CONTROL-

Should be diagnosed or treated as early as possible

ANTIBIOTIC REGIMEN-

Immunocompetent adults – PIPERACILLIN AND TAZOBACTAM, MEROPENEM, IMEPENEM, VANCOMYCIN AND CIPROFLOXACIN

NEUTROPENIA-CASPOFUNGIN

FLUID THERAPY –

Crystalloid (RL, BALANCED SALT SOLUTION , NS)

ALBUMIN – should be used in fluid refractory septic shock, 200ml albumin over 30 to 60 min .

RESPONSIVENESS

STATIC MEASUREMENTS –CVP,SCVO2, PULSE CONTOUR ANALYSIS ,FOCUSSED ECHO

LABILE MEASUREMENTS-PASSIVE LEG RAISING OR IVC COLLAPSIBILITY on USG

VASOPRESSORS AND IONOTROPES

NORADRENALINE IS 1ST CHOICE

Vasopressin 0.03u-0.04u/min salvage therapy to raise MAP>65MMHG OR to decrease the dose of norepinephrine

Dopamine – in selected pts where there is absolute bradycardia or low risk of tachycardia .

STEROID THERAPY –

Inj Hydrocortisone 200mg/day if the MAP is not being maintained inspite of fluid and vasopressors

BLOOD REGIMEN –

HB <7GM% then only blood to be

given .acute mi, acute hypoxemia, acute haemorrhage then only give blood with Hb>7gm%

Not to use erythropoietin and antithrombin

FFP not to be used to correct clotting parameters until to perform an invasive procedures or if there is bleeding

INFECTION PREVENTION

Limited pt contact, hand washing repeated, propped up position, Chlorhexidine mouthwash

GLUCOSE CONTROL

Target blood glucose <180MG%, REPEATED blood measurements of 1 to 2 hrs and then 4 hrly .

BICARBONATE

NOT to be used if ph is >7.15. Don't correct rapidly, use after calculating deficit

MECHANICAL VENTILATION

TV <6ML/KG, PPLAT ,30CMOF H2O ,APPLY PEEP ,RECIRTMENT MANYVERS FOR severe hypoxemias, propped up position 30 to 45 degrees, PRONE POSITION VENTILATION

DVT PROPHYLAXIS –

Graduated compression stockings , DVT pumps , Inj Enoxaparin 40 mg sc.If Creatinine clearance is less than 30ml/min use Dalteperin

STRESS ULCER PROPHYLAXIS

H2 blockers and proton pump inhibitors

PLATLETS

<10000if there is no bleeding

<20000if there is active bleeding

<50000if active bleeding or surgery

NUTRITION

As far as possible enteral feeding within first 48hrs . IV glucose should be started within 7 days along with enteral feeding rather than TPN alone .

WEANING PROTOCOL

Arousable, haemodynamically stable, Fio2 <40%, low ventilator and PEEP requirement

OTHER THERAPIES

Activated protein C, IL7, ANTITHROMBIN 3

EXTRACORPOREAL THERAPIES

CRRT, HIGH CUT OFF HAEMOFILTRATION

B BLOCKERS

LV DIASTOLIC DYSFUNCTION , SHORT ACTING ESMOLOL B1 SELECTIVE ALONG WITH PHENYL EPHRINE (ALPHA 1 AGONIST).

So sepsis is still a very confusing disease for the physician and the bundles are still being sorted out to better the outcome of the disease .