NEWS LINE

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Perioperative Management of Endocrine Disorders

1. Perioperative Glycemic Control in Diabetes Mellitus
2. Management of Adrenal Insufficiency and Stress-Dose Steroids
3. Perioperative Management of Thyroid Disorders
4. Preoperative Optimization and Surgical Management of Pheochromocytoma
5. Calcium and Parathyroid Disorders in the Perioperative Setting
6. Perioperative Management of Pituitary Disorders
7. Endocrine Evaluation and Management in Bariatric Surgery

PRESIDENT'S MESSAGE

Greetings from Karnataka Endocrine Society!

Welcome to this special edition of the KES Newsletter, curated to commemorate **World Hormone Day on 24th April.**

Endocrinology is inherently multidisciplinary, often intersecting with various medical and surgical specialties. In this edition, we focus on the Perioperative Management of Endocrine Disorders.

Given the high prevalence of diabetes mellitus, and the frequency of surgical interventions for complications ranging from acute infections—such as pyelonephritis , diabetic foot and abscesses—to chronic cardiovascular and ophthalmic procedure is high. Suboptimal control leads to significant risks to patients with risk of mortality, morbidity, length of stay and re-admission.

Without meticulous perioperative care, there is a high risk of life-threatening emergencies, including Myxedema Coma, Thyroid Storm, and Addisonian Crisis Furthermore, functional tumours of the Adrenal and Pituitary glands, as well as hyperthyroidism, require rigorous hormonal evaluation and optimization to ensure surgical safety. There are also emergent situations where there is time constraint for optimisation.

We hope this edition serves as a valuable resource for the relevant specialities.

I would like to extend my heartfelt gratitude to my colleagues for their expert contributions and to the editors for their tireless effort in bringing this issue together at such short notice.

Your expert feedback is appreciated.
Thank you.

FROM THE EDITORS' DESK

This issue brings into focus the intricate and often underappreciated domain of perioperative management of endocrine disorders. As surgical outcomes increasingly depend on multidisciplinary precision, understanding the endocrine milieu before, during, and after surgery becomes essential. In this edition, we present a collection of concise mini reviews that distill current evidence, practical guidelines, and evolving perspectives across a spectrum of endocrine conditions.

From optimizing glycemic control in diabetes to navigating adrenal insufficiency, thyroid dysfunction, and calcium imbalances, each article aims to bridge the gap between theory and bedside application. The contributors have thoughtfully highlighted clinical challenges, risk mitigation strategies, and decision-making frameworks relevant to everyday practice.

We hope this compilation serves as a quick yet comprehensive reference for clinicians, trainees, and allied professionals striving for safer perioperative care and improved patient outcomes.

Happy reading.

Dr Rajeshwari
President
Karnataka Endocrine Society

Dr. Belinda George
Honorary Secretary

Dr Aditi Chopra

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Editorial Board

PERIOPERATIVE GLYCEMIC CONTROL IN DIABETES MELLITUS

Dr Jyothi Mariam Idiculla Dr Gurusangappa S Mudagall

Introduction

About 25% of patients with diabetes require surgery, and diabetes is present in ~17% of surgical patients, rising to nearly 40% in cardiovascular procedures. Additionally, over 20% may have hyperglycemia or undiagnosed diabetes. Diabetes is associated with poor wound healing, longer hospital stays, and increased cardiac and respiratory complications, leading to higher morbidity and mortality—especially with poor glycemic control.

Physiological Response to Surgery

Surgical stress triggers the release of catecholamines, glucagon, and cortisol, causing a hypermetabolic state and hyperglycemia. In patients with diabetes, this can overwhelm metabolic control and lead to complications such as diabetic ketoacidosis. Maintaining good glycemic control improves outcomes in both known and newly detected hyperglycemia.

Glycemic Targets

The American Diabetes Association recommends maintaining perioperative blood glucose levels between 100 and 180 mg/dL, a target that is widely adopted in clinical practice. An intensive glycemic control has not consistently shown a reduction in complications or mortality. Meta-analyses have demonstrated that this increases the incidence of hypoglycemia without significant clinical benefit.

Phases of Perioperative Management

The management of diabetes in the surgical setting can be broadly divided into **preoperative, intraoperative, and postoperative phases.**

Preoperative Management

A comprehensive risk assessment is essential, focusing on macrovascular and microvascular complications, as well as comorbidities such as malignancy. Lifestyle measures—including smoking cessation, weight optimization, and regular exercise—should be encouraged. For elective surgery, glycemic control should be optimized (target HbA1c <8% within 3 months or 14 day glucose management indicator <8% with >50% time in range).

Oral glucose-lowering agents are withheld on the day of surgery. Key considerations include:

- Metformin: risk of lactic acidosis in renal impairment
- Sulfonylureas: increased hypoglycemia risk
- Thiazolidinediones: fluid retention, may worsen heart failure
- SGLT2 inhibitors: risk of euglycemic ketoacidosis; stop ≥72 hours preoperatively

GLP-1 receptor agonists and dual GLP-1/GIP analogues require an individualized approach due to delayed gastric emptying and aspiration risk. Decisions should consider treatment duration, GI symptoms, and procedure type. A 24-hour liquid diet may be useful; if withheld, alternative glycemic strategies are needed.

Insulin therapy should be adjusted to avoid hypoglycemia while maintaining control:

- Long-acting insulin: reduce by ~25%
- NPH insulin: reduce by up to 50%
- Twice-daily regimens: reduce evening dose by 25% and morning dose by 25–50%

Close glucose monitoring is essential, with IV dextrose as needed. Insulin pumps require basal rate adjustments and specialist oversight. Insulin must never be discontinued in type 1 diabetes.

Intraoperative Management

Glycemic management during surgery depends on the duration and complexity of the procedure. For short procedures (<2–4 hours), blood glucose should be monitored every 1–2 hours, with correctional insulin given if levels exceed targets. In well-controlled patients, minimal oral intake restarts. For longer or complex surgeries, **intravenous insulin infusion with 5% dextrose** is recommended to maintain glucose between **100–180 mg/dL**. Monitoring includes hourly capillary glucose, laboratory glucose every 2 hours, and electrolytes/bicarbonate every 4–6 hours. Insulin infusion rates can be titrated using simple protocols (e.g., glucose/100). In case of hypoglycemia, insulin should not be stopped in type 1 diabetes; instead, reduce insulin and increase dextrose infusion.

In emergency surgeries, IV insulin and dextrose infusions should be initiated promptly to achieve glycemic control.

Postoperative Management

Close glucose monitoring should continue in the postoperative period to prevent both hyperglycemia and hypoglycemia. For patients on IV insulin, the infusion should be continued for up to 2 hours after initiating subcutaneous insulin and resuming oral intake to avoid ketosis. Subsequent insulin dosing should be individualized based on nutritional intake and clinical status.

Oral glucose-lowering agents can be restarted once oral intake is established, with precautions: metformin only if renal function is adequate (eGFR >30 mL/min), GLP-1–based therapies delayed in patients with nausea/vomiting, SGLT2 inhibitors used cautiously due to risks of dehydration and ketosis, and thiazolidinediones avoided or used carefully in those with fluid overload or heart failure.

Conclusion

Perioperative management of diabetes requires careful planning, individualized treatment strategies, and close monitoring across all phases of surgical care. Maintaining optimal glycemic control while avoiding hypoglycemia is critical to improving surgical outcomes and reducing complications in patients with diabetes.

Intraoperative Glycemic Management

Parameter	Short Procedures (<2–4 hours)	Long / Complex Procedures	Emergency Surgery
General approach	Minimal intervention if well controlled	Active glycemic management required	Immediate control required
Glucose monitoring	Every 1–2 hours	Hourly capillary + lab ≥2 hourly	Hourly capillary + lab ≥2 hourly
Insulin therapy	Correctional insulin if needed	Continuous IV insulin infusion	Continuous IV insulin infusion
Dextrose infusion	Usually not required	5% dextrose infusion	5% dextrose infusion
Target glucose	100–180 mg/dL	100–180 mg/dL	100–180 mg/dL
Adjustment method	Based on glucose values	Infusion rate = glucose ÷ 100	Infusion rate = glucose ÷ 100
Electrolyte monitoring	Not routinely required	Every 4–6 hours	Every 4–6 hours
Hypoglycemia management	IV dextrose	**Do not stop insulin in Type 1 DM; reduce insulin, increase glucose** Stop insulin, increase dextrose	**Do not stop insulin in Type 1 DM; reduce insulin, increase glucose** Stop insulin, increase dextrose
Parameter	**Short Procedures (<2–4 hours)**	**Long / Complex Procedures**	**Emergency Surgery**
		infusion rate in type 2 DM	infusion rate in type 2 DM
Post-procedure plan	Resume usual therapy after oral intake	Transition to SC insulin overlapping by 2 hours	Transition to SC insulin overlapping by 2 hours

MANAGEMENT OF ADRENAL INSUFFICIENCY AND STRESS DOSE STEROIDS

Dr Hema Venkataraman

Dr Nanda N

Adrenal insufficiency (AI) is a life-threatening endocrine condition characterized by the failure of adrenocortical function. This failure results in impaired secretion of glucocorticoids (GCs) ± mineralocorticoids (MCs) and adrenal androgens.

Primary AI occurs from a pathology affecting the adrenal gland. Secondary AI results from a decreased secretion of adrenocorticotrophin hormone (ACTH) from the pituitary gland.

This review focuses on the management of AI in wellness, sickness (stress dosing) and in the perioperative period.

1. Management of AI in wellness

Table 1: Treatment of PAI: Glucocorticoid replacement regimen

Adults:	Oral Hydrocortisone (HC): 15-25 mg in two or three divided oral doses / day Highest dose given in the morning at awakening, the next either in the early afternoon (2 hour after lunch; two dose regimen) or at lunch and afternoon (three dose regimen).
	Prednisolone: 3-5 mg/day (orally once or twice daily), considered specially in patients with reduced compliance
	Dexamethasone: Not to be used. Risk of cushingoid side effects due to difficulties in dose titration.
Pediatrics:	HC: Starting dose of 8mg/m2/day (doses adjusted on individual basis)
	Prednisolone/ Dexamethasone: Not recommended
	Monitoring: glucocorticoid replacement by clinical assessment, including growth velocity, body weight, blood pressure, and energy levels.
Pregnancy:	-Treatment based on individual clinical course, an increase in hydrocortisone dose should be implemented particularly during the third trimester. -Against using prednisolone and dexamethasone because it is not inactivated in the placenta. -Hydrocortisone stress dosing during the active phase of labor similar to that used in major surgical stress
Monitoring:	-Glucocorticoid replacement using clinical assessment including body weight, postural blood pressure, energy levels, signs of frank glucocorticoid excess -Against hormonal monitoring

Mineralocorticoid replacement regimen in PAI:

Adults	Fludrocortisone: 50-100mcg once daily and unrestricted salt intake
Paediatrics	Fludrocortisone: 100mcg/d. Infants – sodium chloride supplements in newborn period and up to age of 12 months.
Monitoring	Based on clinical assessment (salt craving, postural hypotension, or edema) and serum electrolyte measurement

Secondary adrenal insufficiency treatment:

At least 1% of the population use chronic glucocorticoid therapy as anti-inflammatory and immunosuppressive agents via multiple modes of administration (including oral, inhaled, intranasal, intra-articular, topical, and intravenous), and are at risk for glucocorticoid- induced adrenal insufficiency.

Tapering of glucocorticoids can be done more rapidly within a supraphysiological range and followed by a slower taper when on physiological glucocorticoid dosing. The degree and persistence of HPA axis suppression after cessation of glucocorticoid therapy are dependent on overall exposure and recovery of adrenal function varies greatly amongst individuals.

2. Management of AI in illness: Sick Day rules & perioperative care

All glucocorticoid-dependent patients are at risk of adrenal crisis as a consequence of surgical stress or illness, and it is essential for HCPs to be able to recognise and diagnose this medical emergency. If in doubt about the need for glucocorticoids, they should be given as there are no long-term adverse consequences of short-term glucocorticoid administration. Adrenal crisis in patients with known AI is best prevented by patient and HCP education of sick day rules / stress dosing (increased dose) of steroids during illness and surgery (Table 2).

Condition	Stress Steroid dosing in AI
Minor illness with fever (> 38 C)	Hydrocortisone (HC) replacement doses doubled or tripled (>39 c) until recovery (usually 2-3 days) and increased consumption of electrolyte-containing fluids as tolerated.
Unable to tolerate oral medicines during illness (eg Gastroenteritis)	Adults– Injection HC 100mg (im/iv); followed by I.V. infusion / I.V. boluses of HC as below Children Injection HC 50mg/m2; ● Infants – 25mg ● School age children -50mg ● Adolescents- 100mg
Minor surgery / stress not requiring GA	HC, 25–75 mg/24 h (usually 1 to 2 d) Children, i.m HC 50 mg/m2 or HC replacement doses doubled or tripled
Major surgery with general anaesthesia, trauma, delivery / disease that requires intensive care	I.V. HC 100 mg at induction, followed by continuous iv infusion of 200 mg HC/24h (alternatively 50 mg every 6 h iv or im) until able to eat and drink followed by double dose oral replacement steroids and gradually to appropriate maintenance dose (over 48 hours to 7 days) -Children, HC 50 mg/m2 iv followed by HC 50 –100 mg/m2/d divided q6 h, weight-appropriate continuous iv fluids with 5% dextrose and 0.45% NaCl Rapid tapering and switch to oral regimen depending on clinical state
Acute adrenal crisis	-Rapid infusion of 1 ltr of NS within first hour or DNS followed by continuous iv NS on individual basis - HC 100 mg iv immediately followed by hydrocortisone 200 mg/d as a continuous infusion for 24 h until patient is able to eat and drink -Children, rapid bolus of normal saline (0.9%) 20 mL/kg. Can repeat up to a total of 60 mL/kg within 1 h for shock. Children, hydrocortisone 50 –100 mg/m2 bolus followed by hydrocortisone 50 –100 mg/m2/d divided q6h Hypoglycemia: Dextrose 0.5–1 g/kg of dextrose or 2– 4 mL/kg of D25W (maximum single dose 25 g) infused slowly at rate of 2 to 3 mL/min. Alternatively, 5–10 mL/kg of D10W for children 12 y old.

The steroid emergency card

The Steroid Emergency Card is a critical tool supported by the Society for Endocrinology to prevent adrenal crises by alerting healthcare professionals to a patient's steroid dependency. The society recommends that patients at risk of adrenal insufficiency carry this card at all times to ensure prompt treatment in the ER. A sample card designed at our institute is shown below.


KHM24 00001

25 kauvery hospital

IMPORTANT MEDICAL INFORMATION

Steroid Emergency Card (Adult)

This patient is physically dependent on daily steroid therapy as a critical medicine. It must be given/taken as prescribed and never omitted or discontinued. Missed doses, illness or surgery can cause adrenal crisis requiring emergency treatment.

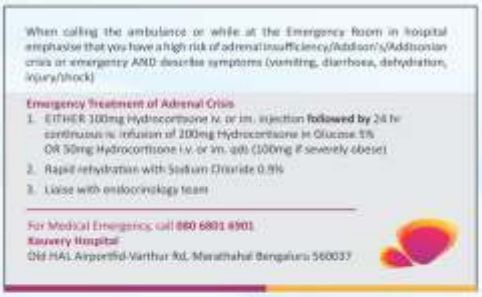
Patients not on daily steroid therapy or with a history of steroid usage may also require emergency treatment.

Name

Date of Birth

Why Steroid Prescribed

Emergency Contact


When calling the ambulance or while at the Emergency Room in hospital emphasise that you have a high risk of adrenal insufficiency/Addison's/Addisonian crisis or emergency AND describe symptoms (vomiting, diarrhoea, dehydration, injury/shock)

Emergency Treatment of Adrenal Crisis

1. EITHER 100mg Hydrocortisone iv. or im. injection **followed by** 24 hr continuous iv. infusion of 200mg Hydrocortisone in Glucose 5%
   OR 50mg Hydrocortisone i.v. or im. qds (100mg if severely obese)
2. Rapid rehydration with Sodium Chloride 0.9%
3. Liaise with endocrinology team

For Medical Emergency, call 080 6801 4901

Kauvery Hospital

Old HAL Airport Rd, Varthur Rd, Marathahal Bengaluru 560037

Conclusion:

The prevention and treatment of Adrenal crisis is by timely administration of steroids higher than usual maintenance doses, either orally or parentally determined by the severity of stress. Patient and HCP education is key to preventing and optimising treatment of acute adrenal crises.

PERIOPERATIVE MANAGEMENT OF THYROID DISORDERS

Dr Afsar Fatima

Dr Rashmi K G

Perioperative Management of Hypothyroidism

Managing the hypothyroid patient in the surgical setting requires balancing the risks of untreated hormone deficiency against the potential cardiovascular, respiratory and anaesthesia related complicationsof non-treatment. While most cases are straightforward, moderate to severe undiagnosed hypothyroidism can lead to life-threatening complications.

1. Who should be screened for thyroid disorder prior to procedure?

Though universal screening is not followed, high-risk and major surgeries, clinical history suggesting thyroid dysfunction,goitre, central hypothyroidism, high prevalence regions should have a baseline thyroid profile done. Known stable hypothyroid (evaluated in past 3 months), minor procedures may not necessarily be screened

2. What is the peri-operative risks in patients with uncontrolled hypothyroidism?

- Cardiovascular effects– peri-operative hypotension, low cardiac output, conduction abnormalities, arrhythmia
- Respiratory- ventilatory dysfunction, effusions, difficult airway, delayed extubation
- Hyponatremia, hypothermia
- Coagulopathy, dyslipidaemia
- Myxoedema, post-operative paralytic ileus, delayed wound healing

3. What are the preferred agents during GA(general anaesthesia) in hypothyroidism?

Induction- ketamine

Maintenance – Nitrous oxide, short acting agents

Intubation – rapid sequencing:succinylcholine or non-depolarising muscle relaxants

4. Should elective surgery be postponed for a patient with subclinical hypothyroidism?

Current guidelines suggest that subclinical hypothyroidism (elevated TSH with normal freeT3,T4) is not a contraindication to surgery. There is no evidence that these patients have increased perioperative morbidity or mortality

5. How should levothyroxine (LT-4) be managed on the morning of surgery?

The patient can take their usual dose of levothyroxine with a sip of water. In bowel surgery/ prolonged NPO state, as LT-4 has a long half-life (~7 days), missing a single dose rarely has acute impact, but maintaining a steady state is preferred.If the patient cannot tolerate oral intake for more than 5 to 7 days, critically ill, parenteral replacement(IV dose ~ 60% to 80% of usual dose) is necessary.

6. If emergency surgery is required in a severely hypothyroid patient, what is the management strategy?

If surgery cannot be delayed, management includes:

Stress-dose Glucocorticoids: Hydrocortisone 100 mg IV every 8 hours until coexisting adrenal insufficiency is ruled out.

IV Levothyroxine: 200–500µg bolus immediately, followed by maintenance(50 -100 µg IV daily).

IV Liothyronine considered if suspicion for myxoedema coma

Perioperative management of hyperthyroidism

1. When to plan for elective surgery in patients with hyperthyroidism?

- Patients scheduled for elective surgery should be made euthyroid before surgery, and cardiovascular stability should be ensured.
- Patients with Subclinical hyperthyroidism (low TSH with normal free T4 and T3) can proceed for surgery under the cover of beta-blockers.
- Patients with overt hyperthyroidism (suppressed TSH with elevated free T4 and/or T3 concentrations) can undergo surgery once they have achieved a euthyroid state and are hemodynamically stable

2. How to manage patients with hyperthyroidism undergoing emergency surgery?

Patients with overt hyperthyroidism requiring emergency surgery needs close monitoring with the use of cardiovascular monitoring devices. Premedication with beta-blockers, anti-thyroid agents and corticosteroids should be administered. Preferred beta-blockers include propranolol which may be used intravenously during surgery and has additional benefit of inhibiting conversion to active hormone, or an esmolol infusion, which allows for rapid titration because of its short acting. Anti-thyroid drugs (methimazole and propylthiouracil),

decrease thyroid hormone synthesis and should be given orally or rectally. When there is urgent need to stabilize thyrotoxicosis, iodine should be given 1 hour after thionamide administration to block the organification of iodide and decrease thyroid hormone synthesis. Additionally, stress dosing of glucocorticoids (Inj hydrocortisone 100mg Q8h; tapered over 3 days), should be given to address relative adrenalinsufficiency and prevent conversion of thyroxine to triiodothyronine

3. Management of subclinical/overt hyperthyroidism in patients with thionamide intolerance?

In patients with subclinical or overt hyperthyroidism who are thionamide-intolerant and require urgent surgery, rapid optimization with beta-blockers, iodine (if not contraindicated), glucocorticoids, and adjuncts such as cholestyramine or lithium, along with close perioperative cardiac and hemodynamic monitoring, is essential to reduce the risk of thyroid storm

4. Rate control in patients with contraindication to betablockers

Patients who have relative contraindications to beta blockade may tolerate beta-1-selective agents better, such as atenolol or metoprolol, or calcium channel blockers. Glucocorticoids may also be given because these agents are thought to decrease the release and peripheral conversion of thyroid hormone. Additionally, in the setting of thyrotoxicosis, glucocorticoids may be necessary to treat relative adrenal insufficiency

5. What are the optimal strategies for administering ATDs in hyperthyroid patients who are NPO or RT, NG tube, or gastrostomy tubes in the perioperative period

In hyperthyroid patients who are NPO during the perioperative period, it is important to continue antithyroid therapy. Administer crushed methimazole or carbimazole via a nasogastric (NG), refeeding (RT), or percutaneous endoscopic gastrostomy (PEG) tube whenever enteral access is available. If enteral access is not possible, use rectal propylthiouracil (PTU) or methimazole, or intravenous methimazole if available. Additionally, administer beta-blockers and, if necessary, steroids to maintain euthyroidism and prevent thyroid storm.

6. Preferred anaesthetic agents during general anaesthesia in patients with hyperthyroidism

- During surgery, sodium thiopental may be a preferred induction agent because it has antithyroid activity.
- Drugs such as ketamine and ephedrine that increase sympathetic tone should be avoided.
- Succinylcholine or a nondepolarizing muscle relaxant devoid of having deleterious cardiovascular effects is acceptable for facilitation of intubation after induction.
- When reversing neuromuscular blockade, anticholinergics (atropine or glycopyrrolate) may lead to exaggerated sympathetic responses, particularly profound tachycardia. Glycopyrrolate, which has less chronotropic effect than atropine, may be the preferred choice

7. Post-operative complications in patients with subclinical/overt hyperthyroidism

Patients with overt hyperthyroidism, and to a lesser extent subclinical hyperthyroidism are at increased risk of postoperative complications such as tachyarrhythmias (especially atrial fibrillation), hypertension, heart failure, myocardial ischemia, thyroid storm, hyperthermia, delirium/agitation, increased metabolic/catabolic stress, and poor glycaemic/hemodynamic control, with risk being highest in overt hyperthyroidism.

PREOPERATIVE OPTIMIZATION & SURGICAL MANAGEMENT OF PHAEOCHROMOCYTOMA

1. Introduction

Phaeochromocytomas are rare, catecholamine-producing neuroendocrine tumours arising from the chromaffin cells of the adrenal medulla. Similar tumours arising from extra-adrenal chromaffin tissue are known as paragangliomas.

These tumours represent a critical cause of secondary hypertension. While most cases in adults are sporadic and unilateral, bilateral or early-onset cases often indicate an underlying genetic predisposition. With an annual prevalence of 2 to 8 per million people, early suspicion and meticulous management are vital to preventing life-threatening cardiovascular complications.

2. Goals of Preoperative Management

Phaeochromocytoma patients are exposed to chronic, high levels of circulating catecholamines, which can lead to multi-organ damage. While surgery is the definitive treatment, it is a high-risk procedure that requires a multidisciplinary team (MDT) approach involving endocrinologists, surgeons, and anaesthetists.

The primary goals of preoperative optimization are:

- **Haemodynamic Stability:** Achieving consistent control of blood pressure and heart rate.
- **Crisis Prevention:** Minimizing the risk of a hypertensive crisis during induction of anaesthesia or tumour manipulation.
- **Volume Expansion:** Reversing chronic intravascular volume contraction caused by catecholamine-induced vasoconstriction.

3. Pharmacological Optimization (The 7–14 Day Protocol)

Proper pharmacological preparation is mandatory to minimize catecholamine-induced adverse events.

Alpha-Adrenergic Blockade (The First Step)

Alpha-blockade must be initiated at least **10–14 days before surgery** to prevent a hypertensive crisis due to "unopposed alpha stimulation."

- **First-line Agent: Phenoxybenzamine** (a non-selective, irreversible alpha-blocker).
  - **Dosing:** Start at 10 mg twice daily; titrate every 48 hours.
  - **Maintenance:** Typically 20–40 mg per day.
- **Alternative Agents:** Selective Alpha-1 blockers like **Doxazosin** or **Prazosin** may be used for a more favourable side-effect profile.
- **Clinical Indicators of Success:** The presence of nasal congestion and mild orthostatic hypotension. Target BP should be approximately **130/80 mmHg.**

Beta-Adrenergic Blockade (The Second Step)

Beta-blockers are used to control reflex tachycardia or arrhythmias.

- **CRITICAL RULE: Never start a beta-blocker before an effective alpha-blockade is established.** Doing so can cause a paradoxical rise in blood pressure.
- **Timing:** Wait at least **2–3 days** after achieving effective alpha-blockade.
- **Agents:** Propranolol, Metoprolol, or Esmolol.

Second-Line or Adjunctive Agent:

Calcium Channel Blockers (e.g., Amlodipine):

Role: Used as an add-on to alpha-blockers for refractory hypertension or as monotherapy in patients who cannot tolerate alpha-blockers.

Advantages: Reduces the risk of severe postoperative hypotension and carries a lower risk of orthostatic hypotension compared to Phenoxybenzamine.

Limitation: May be less effective at suppressing severe hypertensive paroxysms during direct surgical manipulation of the tumour.

Volume Restoration

Chronic catecholamine excess leads to a contracted plasma volume.

- **Management:** Encourage a high-sodium diet and increased fluid intake. This prevents profound hypotension after the tumour's blood supply is ligated.

4. Intraoperative Management

The surgical standard is laparoscopic adrenalectomy, though open abdominal excision may be required for large, complex, or invasive tumours.

- **Anaesthesia:** General anaesthesia is preferred. **Propofol** is recommended for induction, and **Fentanyl** for analgesia.
- **Crisis Management:** Hypertensive surges during tumour manipulation are managed with rapid-acting vasodilators: **Sodium Nitroprusside**, **Nitroglycerine**, or **Nicardipine**.
- **The Role of Magnesium:** Intravenous Magnesium Sulphate is a highly effective adjunct for controlling both hypertension and arrhythmias.
- **Post-Ligation Hypotension:** Once the tumour vein is ligated, a sudden drop in catecholamines can cause severe hypotension. This is managed with aggressive **intravenous fluid boluses** (crystalloids or colloids) and, if necessary, inotropic/vasopressor support (e.g., **Noradrenaline or Phenylephrine).**

5. Postoperative Care

Patients require intensive monitoring in an ICU or HDU for 24–48 hours to manage potential complications:

- **Persistent Hypotension:** Due to residual alpha-blockade and sudden withdrawal of catecholamines; treated with fluids and vasopressors.
- **Hypoglycaemia:** Caused by a sudden rebound in insulin secretion and increased glucose uptake once the inhibitory effect of catecholamines is removed.
- **Hypertension Monitoring:** While most patients become normotensive, persistent hypertension may indicate residual disease or fluid overload.

6. Long-Term Follow-up

Due to the risk of recurrence or the development of new tumours (especially in hereditary cases), long-term follow-up is essential. Annual biochemical screening is often recommended to ensure the patient remains in remission.

CALCIUM AND PARATHYROID DISORDERS IN THE PERIOPERATIVE SETTING

Dr Medha Rao

Dr Aditi Chopra

Calcium homeostasis is tightly regulated by parathyroid hormone (PTH), vitamin D, and calcitonin. In the perioperative setting, this balance is frequently disrupted by surgical stress, fluid shifts, blood transfusions, and direct glandular manipulation. Unrecognised hypo- or hypercalcaemia can precipitate cardiac arrhythmias, seizures, and haemodynamic instability. Here, through three clinical vignettes, we review the key scenarios every physician should recognise.

Case 1: Post-Thyroidectomy Hypocalcaemia

A 42-year-old woman presents with perioral tingling and carpopedal spasm 18 hours after total thyroidectomy for multinodular goitre. Corrected calcium is 7.1 mg/dL; iPTH is 6 pg/mL (low).

Discussion: Inadvertent parathyroid devascularisation or removal during thyroidectomy is the commonest cause of acute post-surgical hypocalcaemia, reported in 20–30% of total thyroidectomies (transient) and 1–2% permanently. A post-operative iPTH< 15 pg/mL at 4–6 hours strongly predicts symptomatic hypocalcaemia. Immediate management includes IV calcium gluconate, followed by oral elemental calcium 1–3 g/day and calcitriol 0.25–0.5 µg twice daily. Monitoring corrected calcium every 6–12 hours for the first 48 hours is essential.Concomitant hypomagnesaemia should be corrected, and underlying vitamin D deficiency increases risk and severity Patients should be discharged with clear instructions to report symptoms of hypocalcaemia.

Case 2: Hypercalcaemic Crisis Before Elective Surgery

A 58-year-old man with known primary hyperparathyroidism is scheduled for elective cholecystectomy. Pre-anaesthetic labs reveal a corrected calcium of 13.8 mg/dL with a QTc of 320 ms.

Discussion: Severe hypercalcaemia (≥ 14 mg/dL) is a medical emergency and a contraindication to elective surgery. The risk of intra-operative cardiac arrhythmias (shortened QTc, bradycardia, heart block) rises steeply above 12 mg/dL. Initial management includes aggressive isotonic saline hydration (200–300 mL/hour, titrated to urine output), followed by calcitonin 4 IU/kg for rapid but short-lived effect, and a bisphosphonate such as zoledronic acid 4 mg IV for sustained control. Furosemide should be reserved strictly for volume-overloaded patients. Elective surgery must be postponed until calcium is consistently below 12 mg/dL. In this case, definitive parathyroidectomy for the PHPT should also be considered.

Case 3: Hungry Bone Syndrome After Parathyroidectomy

A 35-year-old woman undergoes parathyroidectomy for a solitary adenoma. Pre-op calcium was 12.4 mg/dL with elevated ALP (320 U/L) and radiological evidence of osteitis fibrosa cystica. On post-operative Day 2, corrected calcium drops to 6.8 mg/dL despite IV supplementation.

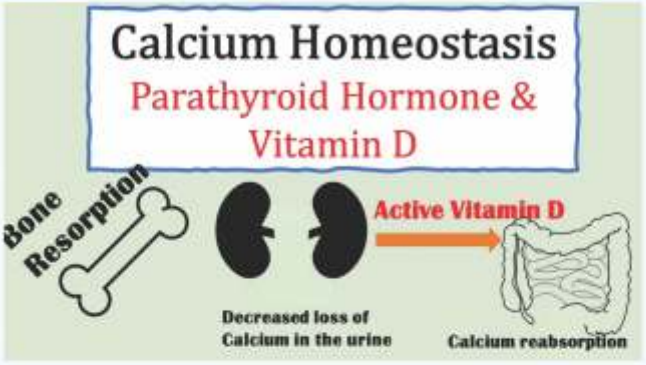
Discussion:

Hungry bone syndrome (HBS) results from rapid skeletal remineralisation following abrupt withdrawal of PTH excess. Risk factors include high pre-operative alkaline phosphatase, significant bone disease, and larger adenoma size.

Unlike hypoparathyroidism, PTH levels are typically normal or elevated in response to hypocalcaemia. The condition is often associated with hypophosphataemia, although phosphate levels may vary depending on timing and renal handling. Hypomagnesaemia is also common and should be corrected. Management requires aggressive and often prolonged calcium replacement, including intravenous calcium infusions, high-dose oral elemental calcium (up to 4–6 g/day), calcitriol (0.5–1 µg/day), and magnesium supplementation. Recovery may take weeks to months.

Scenario	Key Action
Post-thyroidectomy	Check early PTH (4–6 h); initiate calcium and calcitriol if low
Pre-op hypercalcaemia	Postpone elective surgery if Ca > 12; hydrate aggressively
Hungry bone syndrome	Anticipate in high bone turnover; requires prolonged calcium and magnesium replacement

Perioperative calcium disturbances are common but predictable. A structured approach—integrating calcium levels, PTH, and clinical context—allows early intervention and prevents serious complications. Anticipation remains the most effective strategy.



PERI-OPERATIVE MANAGEMENT OF PITUITARY TUMOURS

Introduction:

Pituitary adenomas are quite common, making up about 18.1% of all brain tumours and ranking as the second most frequent intracranial pathology. The standard treatment for most of these tumours is trans-sphenoidal surgery, while prolactin-secreting tumours (prolactinomas) are usually treated with dopamine agonists rather than surgery.

A thorough endocrine evaluation of pituitary adenoma preoperatively is crucial to identify hormonal compromise caused by the large sellar mass and identifying functional tumours to improve patient care and outcome.

Although trans-sphenoidal surgery is generally safe when performed by experienced surgeons, it still carries a risk of postoperative hypopituitarism. Therefore, careful monitoring during the immediate postoperative period is important to reduce complications and mortality.

I.Preoperative Evaluation:

1.History and physical examination: Symptoms of pituitary tumors depend on size, local invasion, and hormonal activity, presenting with visual disturbances, headache, or features of hypo-/hyperpituitarism. Preoperative evaluation includes detailed neuro-ophthalmic assessment, systemic examination for endocrine signs (e.g., galactorrhea, acromegaly, Cushing's), and ENT review for surgical planning.

2. Pre-Surgical testing - Baseline Evaluation

Assessment of hormonal excess and deficiencies is recommended in all patients with pituitary adenomas regardless of symptoms.

Table 1: Pre-operative hormonal assessment of pituitary adenomas.

Pituitary axis	Tests	Comments
Prolactin Axis	Prolactin	• Avoid exercise and nipple stimulation 30 min before exercise • Prolactin levels correlate with the size of adenoma, prolactin levels exceeding 250 ng/mL typically indicative of a macroadenoma. • There may be discrepancies in prolactin level and size in the setting of cystic prolactinoma and drugs like risperidone and metoclopramide. • Consider Endocrinology evaluation for further work up of hyperprolactinemia if needed
Adrenal axis	AM cortisol Dynamic testing (CST) if needed 24-h urine cortisol or Dexamethasone Suppression Testing	• A morning serum cortisol concentration above 10 ug/dl suggests an intact HPA axis • Obtain Co-Syntropin stimulation test if AM cortisol 3–10 µg/dL • AI is confirmed if AM cortisol is below 3 µg/dL and or if CST is abnormal • A normal CST result is defined by an increase in cortisol greater than 18 µg/dL after an injection of 250 mcg of an ACTH analog • Treat with GC preoperatively if AI is confirmed or suspected: Hydrocortisone 15 mg in divided doses or prednisone 5 mg daily • Stress dose GC is needed preoperatively in all patients undergoing surgery: Hydrocortisone 50–100 mg IV at anesthesia induction followed by a quick taper
Growth hormone axis	IGF-1	• Normally elevated IGF-1 in adolescence and pregnancy • Falsely low IGF-1 can be seen with liver/renal failures, uncontrolled diabetes, hypothyroidism, malnutrition, and oral contraceptive use. • A mildly elevated IGF-1 or equivocal test should be confirmed with a glucose suppression test
Thyroid axis	TSH and free T4	A low free T4 level with inappropriately normal or low TSH is diagnostic for central hypothyroidism • Treat central hypothyroidism with levothyroxine preoperatively • Always rule out AI prior to initiating levothyroxine. • TSH is not reliable, always follow free T4 levels. Target free T4 level in the mid to high normal range.

Pituitary axis	Tests	Comments
Gonadal Axis	LH, and FSH in both sexes Serum Testosterone in males Serum Estradiol in premenopausal female	The assessment of pituitary-gonadal function is contingent on the patient's gender and age • Always check testosterone via LCMS in the morning in males • No need for biochemical testing in a female patient with a normal menstrual cycle • In selected patients, a progesterone challenge test may be employed to confirm the presence of estrogen deficiency • For postmenopausal females, assessing serum FSH levels can aid in delineating pituitary insufficiency • No need to treat preoperatively.
Fluid and electrolyte imbalance	Serum sodium	• If patient symptomatic with polyuria /polydipsia and has high normal or elevated serum sodium, check urine osmolarity • Treat with desmopressin if needed.

Abbreviations: AI, adrenal insufficiency; CST, cosyntropin stim test; DST, dexamethasone suppression test; GC, glucocorticoids; LCMS, liquid chromatography mass spectrometry.

II. Postoperative Management

1. Adrenal Axis

Use steroids peri-operatively as intravenous hydrocortisone 25-50mg Q8H and once D3-D4 post-operative period, once patient's oral intake is stable, oral steroids either hydrocortisone or prednisone can used. Dose is reduced to physiological or slightly higher dose at the time of discharge. We generally check for HPA axis after stopped steroids only after 2 weeks after the surgery as there is a risk of hyponateremia upto 2 weeks after pituitary surgery and its recommended to keep the adrenal and thyroid axis adequately replaced in this time window to avoid worsening of hyponatremia.

Table 2: Postoperative assessment of HPA axis in Non-functioning pituitary adenoma

Intact HPA axis preoperatively	Continue GC perioperatively Check cortsiol POD 14 after stopping GC	Check AM cortisol POD14 >10ug/dl no GC and check labs in 3 months Monitor clinically for AI
Central AI diagnosed preoperatively	Continue GC peri operatively Check AM cortisol on follow up (2-4 weeks)	Discontinue GC if AM cortisol level on follow up is >10ug/dl on two consecutive labs, check AM cortisol 1-2 weeks later after stopping GC Monitor clinically for AI

AI, adrenal insufficiency; GC, glucocorticoids; HPA, hypothalamic pituitary adrenal axis; POD, postoperative day.

2. Water and Sodium Balance

Table 3: Monitoring and treatment water and electrolyte disorder after trans-sphenoidal surgery

	Polyuria/AVP-D	Hyponatremia/SIADH
Mechanism	Decrease AVP secretion due to surgical traction and damage to the nerve terminal in the posterior pituitary and due to depletion of ADH deposits	Excessive AVP secretion from degenerating axon terminals
Timings / duration	Symptoms start within 24-48 hours Transient-lasts for 7 days Can be permanent	Symptoms start within 5-7 days Transient-lasts up to 14 days
Symptoms / labs	Polyuria/Polydipsia urine osmolality <300/ urine specific gravity <1.005 serum sodium high normal or>145Meq/L	Urine osmolality >300 Serum sodium low normal or <130meq/L
Monitoring/ treatment	Labs*on POD1 and if needed Monitor for symptoms Drink to thirst Treat with DDAVP	Labs *POD5 Mild hyponatremia: fluids restriction Moderate to severe hyponatremia: hospital admission for management Normal sodium: can initiate fluid restriction of 1L/day on POD 5-10 especially if serum sodium is <140mEq/L

AVP-D: Arginine vasopressin deficiency SIADH: Syndrome of inappropriate antidiuretic hormone, Labs* serum sodium, serum osmolarity, urine osmolarity/urine specific gravity

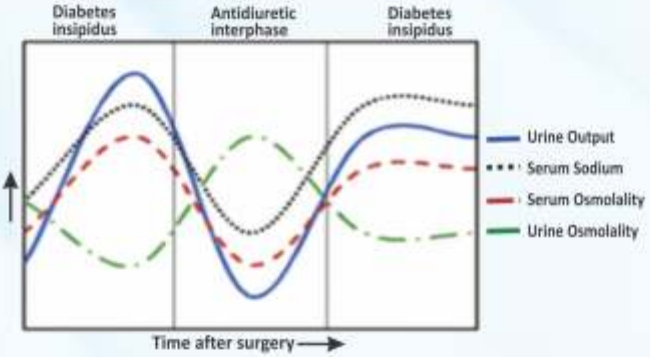
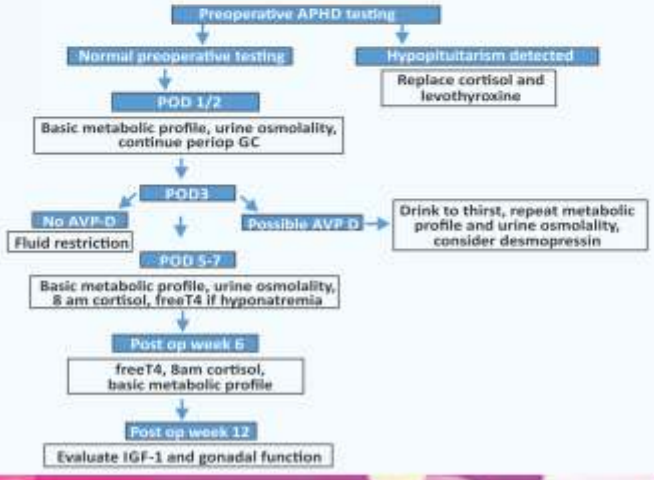


Figure 1:The triphasic response starts with symptoms of DI caused by "stunning" of the magnocellular neurons and a lack of AVP secretion. This initial phase is followed by a period of increased AVP secretion whereby the injured hypothalamic cells degenerate and release their stored AVP. At the end of this antidiuretic interphase, if the majority of AVP-producing neurons are destroyed, a permanent phase of DI begin.

3. Disease-Specific Monitoring

- GH should be measured on POD 3-5 and IGF-1 should be measured at 12 weeks post-operatively in patients with Acromegaly.

Flowchart 1:Hormonal testing after trans-sphenoidal surgery in patients with pituitary masses other than functioning adrenocorticotropic hormone (ACTH)-adenomas



APHD: Anterior pituitary hormone deficiency, POD: postoperative day, AVP-D: Arginine vasopressin deficiency.

4. Discharge planning

- Discharge on a modestly restricted fluid intake of approximately 1 litre/day — this has reduced readmissions for delayed hyponatraemia.
- Advise periodic serum sodium monitoring and regular contact with the endocrine team. (post discharge day 3 and day 7)
- Out-of-station patients should stay near the hospital for the first 1–2 weeks to avoid emergency presentations.

5. Follow-up and axis reassessment

- HPA axis: reassess 14 days post-op; steroids can be weaned if recovered.
- HPT axis: reassess alongside HPA.
- HPG axis and overall recovery: reassess at approximately one month, once the patient is clinically stable.
- Adjust oral desmopressin dose at follow-up based on fluid balance and sodium profile.

Conclusion:

To optimize the care of patients undergoing pituitary surgery, a multidisciplinary approach is paramount. Collaboration between endocrinologists, ophthalmologists, neurosurgeons, and otolaryngologists, coupled with vigilant monitoring, plays a pivotal role in ensuring the best possible outcomes. Furthermore, a thorough preoperative evaluation of pituitary hormones serves as a valuable tool for guiding perioperative decisions, ultimately enhancing patient care.

ENDOCRINE EVALUATION AND MANAGEMENT IN BARIATRIC SURGERY

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Bariatric surgery is a profound metabolic intervention that triggers massive recalibration of the endocrine system, extending far beyond simple weight loss. Long-term success and patient safety depend on meticulous endocrine evaluation and management before, during, and after the procedure.

Preoperative Endocrine Evaluation The preoperative phase must focus on uncovering hidden nutritional deficits and optimising metabolic control.Iron, vitamin B12, vitamin D, and thiamine deficiencies are common in candidates for bariatric surgery and must be corrected prior to surgery.Glycaemic optimisation is also a time-sensitive priority; achieving a preoperative HbA1c level of <7% is associated with significantly fewer perioperative complications.Additionally, endocrine evaluation should screen for underlying conditions that drive obesity, such as excluding hypercortisolism (Cushing's syndrome) through appropriate testing if clinically suspected, as bariatric surgery will not resolve the underlying pathology of Cushing's disease.

Gut Hormones and Glycaemic Control Bariatric surgery induces a rapid, weight-independent improvement in type 2 diabetes, driven by extensive remodelling of the gut hormone axis.Following procedures such as Roux-en-Y gastric bypass (RYGB), ghrelin levels fall, while satiety-promoting hormones such as glucagon-like peptide-1 (GLP-1) and peptide YY (PYY) rise substantially.Fasting plasma glucose levels often normalise before patients leave the hospital.Because insulin sensitivity improves rapidly, physicians must proactively reduce insulin doses and discontinue insulin secretagogues (such as sulfonylureas and meglitinides) in the immediate postoperative period to prevent severe iatrogenic hypoglycaemia.

Post-Bariatric Hypoglycaemia (PBH) While the exaggerated GLP-1 response helps resolve diabetes, it can later drive a dangerous condition known as Post-Bariatric Hypoglycemia (PBH).Occurring months to years post-surgery and is triggered by rapid gastric emptying, which delivers a concentrated carbohydrate bolus to the small intestine, sparking an excessive insulin surge and a subsequent precipitous drop in blood glucose levels.PBH manifests 1–3 h after eating with neuroglycopenic symptoms such as confusion, visual disturbances, and syncope. It must be distinguished from early dumping syndrome, which occurs within 30–60 min of a meal and presents mainly with vasomotor and gastrointestinal symptoms.

The first-line management for PBH is strictly dietary: patients must avoid refined carbohydrates, consume small frequent meals high in protein and fibre, and separate liquids from solid foods by at least 30 min.If symptoms are refractory to dietary changes, pharmacotherapy, such as acarbose (to slow carbohydrate absorption) or somatostatin analogues, such as octreotide (to blunt the incretin response), is recommended.

Bone Health and Calcium Metabolism Bariatric surgery induces a high bone turnover state, posing a silent threat to skeletal health.Procedures such as RYGB bypass the duodenum and proximal jejunum, which are the primary sites for active vitamin D-mediated calcium absorption.Consequently, patients are at an exceedingly high risk for calcium and vitamin D deficiencies, leading to secondary hyperparathyroidism as the body resorbs bone to maintain serum calcium levels. Lifelong supplementation is therefore mandatory.Patients require 1,200 to 2,400 mg/day of elemental calcium, which **must** be prescribed as calcium citrate, as it does not require gastric acid for absorption.This should be paired with at least 3,000 IU of vitamin D3 daily.Physicians should monitor intact parathyroid hormone (PTH), calcium, and 25-hydroxyvitamin D levels and utilise DXA scans to assess baseline and ongoing fracture risk.

Thyroid and Reproductive Axes Bariatric surgery also alters thyroid and reproductive hormone dynamics.The absorption of standard levothyroxine tablets often decreases by 30% to 40% after bypass surgery because the medication requires gastric acid for dissolution.Physicians should closely monitor TSH levels and consider switching to liquid or soft-gel formulations if malabsorption prevents adequate TSH suppression.

Finally, rapid weight loss quickly restores the fertility in women, anovulation and polycystic ovary syndrome (PCOS) often resolve quickly.Because oral contraceptives may be poorly absorbed postoperatively, non-oral contraceptive methods are strongly recommended to prevent unplanned pregnancies during the active weight-loss phase.In men, the reduction of adipose tissue lowers the aromatisation of testosterone to oestrogen, frequently resolving functional hypogonadism and improving free testosterone levels without pharmacological intervention.

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