Preop. visit

ENDOCRINE DISORDERS

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Diabetes Mellitus

The two main disease categories are:

type 1 diabetes (previously called “insulin-dependent diabetes” or “juvenile-onset diabetes”) and

type 2 diabetes (previously called “non–insulin dependent diabetes” or “adult-onset diabetes”).

Type 1 diabetes, which accounts for about **5% to 10%** of all cases of the disease, is the result of autoimmune destruction of pancreatic β-cells.

Affected individuals have an **absolute deficiency of insulin**, but normal sensitivity to insulin.

Since the disease has a typically early onset at a young age, and is often **difficult to control**, adults with type **1 diabetes** are at risk of premature vascular disease, such as IHD, nephropathy, retinopathy, and peripheral neuropathy.

They are also **at risk of diabetic ketoacidosis**.

Type 2 diabetes is characterized by insulin resistance and a relative (but not absolute) deficiency in insulin.

Most affected individuals are obese and **seldom prone to ketoacidosis.**

Diabetes mellitus is associated with **multiorgan dysfunction**, including:

IHD, heart failure (independent of associated IHD), CVD, CKD, peripheral neuropathy, autonomic neuropathy (e.g., postural hypotension, gastroparesis), retinopathy, and reduced joint mobility (e.g., reduced cervical mobility affecting airway management).

In the perioperative setting, diabetes mellitus is a risk factor for **postoperative complications**, including:

cardiac events, acute kidney injury (AKI), and surgical site infections.

Insulin therapy is the main treatment for **type 1 diabetes mellitus**, either as **multiple** daily injections or a **continuous** subcutaneous insulin infusion.

In the case of **type 2 diabetes**, multiple **treatment** options are available, including:

- nonpharmacologic therapy (i.e., diet, weight loss, exercise),
- metformin,
- sulfonylureas (e.g., glyburide, glipizide),
- repaglinide,
- glucagon-like peptide-1 (GLP-1) agonists (e.g., liraglutide),
- sodium-glucose cotransporter 2 (SGLT2) inhibitors (e.g., empagliflozin),
- dipeptidyl peptidase-4 (DPP- 4) inhibitors (e.g., sitagliptin, saxagliptin, linagliptin, alogliptin), and
- insulin.

During the preoperative evaluation of a patient with diabetes mellitus, the anesthesiologist should **document**:

-the disease type (i.e., type 1 vs. type 2),

-current usual glycemic control,

-history of hypoglycemic episodes,

-current therapy,

-and the severity of any end-organ complications.

Given the effects of diabetes mellitus on other organ systems, the **history** and **physical examination** should especially **focus** on the cardiovascular, renal, and neurologic systems.

Inquiries about postural dizziness, early satiety, and postprandial vomiting can help assess for any **autonomic neuropathy.**

The **physical examination** should include an evaluation of pulses, skin breakdown, and joint (especially cervical spine) mobility.

Informative **preoperative laboratory tests** include:

- an ECG and
- blood sampling for electrolyte, creatinine, and blood glucose concentrations.

To help **better estimate** renal function, an **estimated GFR** should be calculated.

Since patients are **not typically fasting** when they are evaluated in a preoperative evaluation clinic, glucose concentrations measured in the clinic cannot be used to evaluate general glycemic control.

A diary of **multiple glucose values** (preprandial and postprandial) at varying times of the day is **more informative** for estimating the adequacy of therapy.

Alternatively, a **glycosylated hemoglobin (HbA1c)** concentration can help characterize the average plasma glucose concentration within the **prior 3 months**.

Among surgical patients, preoperative **HbA1C is more informative** than patients' self-reported history, fasting blood glucose concentrations, and random blood glucose concentrations in identifying preexisting poor glycemic control.

In the nonoperative setting, the American Diabetes Association recommends a target **HbA1c concentration under 7%** for most diabetic patients .

Although preoperative **HbA1c** is correlated with postoperative glycemic control, its role as a **predictor of postoperative complications is largely restricted** to diabetic patients undergoing **orthopedic** or **vascular surgery**.

In the perioperative setting, the goals of glycemic management are:

- to avoid hypoglycemia,
- prevent ketoacidosis, and
- avoid marked hyperglycemia.

Normal treatment regimen for most non-insulin diabetic medications (**metformin, sulfonylureas, repaglinide, GLP-1 agonists, DPP-4 inhibitors**) **should be:**

- **continued until (and inclusive of) the day before surgery** but
- **held on the morning of surgery.**

The possible **exception** pertains to **SGLT2** inhibitors, which have been associated with **euglycemic diabetic ketoacidosis** in the postoperative setting.

Thus, some guidelines recommend that the medications be **discontinued at least 24 hours before elective surgery.**

Diabetic patients should **discontinue *short-acting* insulin while fasting**.

The **exception** pertains to patients with **continuous subcutaneous insulin infusion pumps**.

These individuals should **continue their infusion at the lowest basal rate**, which is usually the nighttime fasting rate.

patients with type 1 diabetes mellitus to take a **small amount** (one third to one half) of their usual morning dose of **intermediate-acting or long-acting insulin** (e.g., lente, isophane) **to avoid diabetic ketoacidosis**.

Patients with **type 2 diabetes** mellitus can either take no insulin or up to **one half** of their usual dose of intermediate-acting, long-acting, or combination (e.g., 70/30 preparations) insulin on the morning of surgery.

Thyroid Disease

The major concern pertains to significant **hyperthyroidism** or **hypothyroidism**, which appears to **increase perioperative risk**.

Symptoms and signs of hypothyroidism and hyperthyroidism can be **subtle** and **nonspecific**, especially with **milder disease** in **older adults**

Hyperthyroid individuals may manifest:

-tachycardia,

-arrhythmias,

-palpitations,

-tremors,

-weight loss, and

-diarrhea.

Patients with **hypothyroidism** may demonstrate:

-hypotension,

-bradycardia,

-lethargy,

-weight gain,

-depressed cardiac function,

-pericardial effusions, and

-impaired ventilatory response to hypoxia or hypercarbia

Patients may also have **goiters** with related symptoms such as:

- dysphagia,
- dyspnea,
- wheezing, and
- orthopnea.

Individuals with **hyperthyroidism** due to **Graves disease** may also demonstrate **proptosis** .

The **preoperative evaluation** should clarify the patient's **current medical therapy** as well as any recent changes.

In patients with **known thyroid disease**, additional preoperative thyroid function testing is not needed if the patient is on a **stable medication** dose and was assessed as being **euthyroid** within the **previous 6 months**.

If additional preoperative testing is clinically indicated, thyroid-stimulating hormone **(TSH)** assays are best to evaluate for **hypothyroidism**, while free triiodothyronine **(T3),** free thyroxine **(T4),** and **TSH** levels are useful in **hyperthyroid** patients.

Surgery, stress, or illness can **precipitate** myxedema or thyroid storm in patients with **untreated or severe thyroid dysfunction.**

In general, if a patient has **moderate or worse hypothyroidism** (i.e., **elevated TSH** and **low free T4—with or without associated symptoms**), elective surgery should be postponed until the individual is euthyroid.

Similarly, elective *non-thyroid* surgery should also be delayed to facilitate treatment of patients with **overt hyperthyroidism** (i.e., **suppressed TSH** with **elevated free T4 or T3 concentrations—with or without associated symptoms**)

Consultation with an endocrinologist is necessary if surgery is urgent in patients with thyroid dysfunction.

If surgery is urgent, **hyperthyroid patients** can be **treated** with β-adrenergic blockers, antithyroid medications (e.g., methimazole, propylthiouracil, potassium iodide), and corticosteroids.

Other potentially useful tests include **chest radiography** or **computed tomography scans** to evaluate **tracheal or mediastinal involvement by a goiter.**

All thyroid replacement therapy and antithyroid drugs should be continued on the day of surgery.

Parathyroid Disease

Parathyroid hormone regulates **calcium**.

Most cases of **hyperparathyroidism** are discovered based on an incidental elevated calcium level found during diagnostic testing.

***Primary* hyperparathyroidism** is caused by a primary disorder of the parathyroid glands (**adenomas or hyperplasia**).

***Secondary* hyperparathyroidism** is parathyroid gland hyperplasia induced by the **hyperphosphatemia** and **hypocalcemia** that occur during **chronic renal failure**.

***Tertiary* hyperparathyroidism** occurs when the parathyroid hyperplasia in secondary hyperparathyroidism functions autonomously.

Hypercalcemia from parathyroid disease is associated with **osteoporosis** and **bone loss**.

It is very **unlikely** that parathyroid glands become **sufficiently enlarged to compromise the airway**.

Hypoparathyroidism is very **uncommon**, but it can be the consequence of a **prior total parathyroidectomy.**

Pheochromocytoma

Pheochromocytomas are **catecholamine-secreting tumors** that arise from **chromaffin cells** of the adrenal medulla.

Similar tumors arising from **sympathetic ganglia** are termed **catecholamine-secreting paragangliomas** or **extraadrenal pheochromocytomas**.

These **rare tumors** (incidence **~1 per 100,000** person-years) occur most commonly between **ages 40 and 60 years**, with equal incidence in males and females.

About **40% of cases** occur as part of a **familial disorder** (i.e., von Hippel-Lindau syndrome, MEN type 2, neurofibromatosis type 1).

The classic triad of **associated symptoms** are:

-episodic headaches (90% of symptomatic patients),

-sweating (60%-70% of symptomatic patients), and

-tachycardia.

About **half of patients have paroxysmal hypertension**, **5% to 15% have normal blood pressure**, and the remainder have what appears to be essential hypertension.

Other manifestations include:

-orthostatic hypotension,

-psychiatric disorders (i.e., panic attacks),

-pallor,

-blurred vision,

-weight loss,

-hyperglycemia, and

-cardiomyopathy.

A diagnosis of **pheochromocytoma should be considered** if any of the following features is present:

- Triad of episodic headaches, sweating, and tachycardia
- Hyperadrenergic spells (e.g., nonexertional palpitations, diaphoresis, headache, tremor)
- Hypertension that is difficult to control, or occurs at a young age

- Hypertension associated with new-onset or atypical diabetes mellitus.
- Idiopathic dilated cardiomyopathy.
- Family history of pheochromocytoma or suspicious familial syndrome (von Hippel-Lindau syndrome, MEN type 2, neurofibromatosis type 1)
- A history of gastric stromal tumors or pulmonary chondromas.
- Incidentally discovered adrenal mass.

Measurements of **fractionated metanephrine** and **catecholamine concentrations** in the **urine** and **plasma** generally establish the diagnosis of pheochromocytoma,

with **recent guidelines** focusing on initial testing with either **plasma-free metanephrines** or **urinary fractionated metanephrines.**

Patients scheduled for **pheochromocytoma resection** should undergo surgery **at centers with experienced teams of anesthesiologists and surgeons**.

They also require about **10 to 14 days of medical preparation before surgery** to mitigate perioperative risks.

The overarching **goals of this preparation** are to:

-control hypertension,

-control tachycardia, and

-normalization of intravascular volume status.

The mainstay of medical therapy is preoperative **α-adrenergic blockade** started 7 to 14 days before surgery.

The preferred drug at many centers is **phenoxybenzamine,** which is an **irreversible, long-acting, nonspecific α-adrenergic blocking drug.**

The **initial dose is 10 mg once or twice daily**, and the dose is **increased by 10 to 20 mg every 2 to 3 days as needed.**

Most patients eventually need doses ranging from **20 to 100 mg daily.**

The arterial blood pressure target is less than 130/80 mm Hg in the seated position, with systolic pressure less than 90 mm Hg while standing.

Typical **side effects** include orthostatic dizziness, fatigue, and nasal congestion.

Given these side effects, as well as higher rates of postoperative hypotension after preoperative phenoxybenzamine treatment, some centers instead use selective α1-adrenergic blocking drugs (e.g., prazosin, terazosin, doxazosin).

These agents are also preferable when longterm pharmacologic treatment is indicated (e.g., metastatic pheochromocytoma).

The disadvantage of selective α1-adrenergic blocking drugs is their **incomplete degree of α-adrenergic blockade**, thus resulting in **more episodes of intraoperative hypertension**.

After adequate α-adrenergic blockade, ß-adrenergic blockade may be started cautiously with short-acting drugs.

As an example, **10 mg of propranolol every 6 hours** can be used.

After 24 to 48 hours, a long-acting preparation (e.g., metoprolol, atenolol) can be substituted, provided that the patient tolerates ß-adrenergic blockade.

The dose is then adjusted to achieve a **heart rate between 60 and 80 beats/min.**

ß-Adrenergic blockade should *never* be initiated before α-adrenergic blockade.

In the setting of unopposed α-adrenergic receptor stimulation, blockade of vasodilatory peripheral ß-adrenergic receptors **worsen hypertension**, while acute depression of cardiac function can **precipitate acute heart failure**.

In addition, **initiation of ß-adrenergic blockade may unmask a catecholamine-induced cardiomyopathy**, with resulting **acute pulmonary edema**.

Alternatives to perioperative **α-adrenergic blockade** include calcium channel blockers and metyrosine

Nicardipine is the **most commonly used calcium channel blocker** for this indication, with a **starting oral dose of 30 mg twice daily** (sustained release preparation).

The **main role** for calcium channel blockers is likely to supplement α-and β-adrenergic blockade **when blood pressure control is inadequate**, or **to treat patients with intolerable side effects from usual therapy**

Metyrosine, which inhibits catecholamine synthesis, has many side effects (e.g., sedation, diarrhea).

As a consequence, it is also **reserved** for cases where conventional treatment is insufficient or not tolerated.

The **preoperative evaluation** of a patient with known pheochromocytoma should focus on the cardiovascular system (including orthostatic vital signs) and current medical treatment for pheochromocytoma (including adequacy of treatment).

Laboratory testing includes: an **ECG**, as well as blood sampling for a **CBC**, **electrolyte** concentrations, **creatinine** concentrations, and **glucose** concentrations.

The patient may also warrant **echocardiography** or a **cardiology consultation.**

KIDNEY DISEASE

During preoperative evaluation, it is important to **establish** the severity, type, and underlying cause of preoperative renal impairment.

CKD is defined as a GFR less than 60 mL/min/1.73 m2 for at least 3 months, regardless of the underlying cause

Chronic kidney failure is defined as a GFR less than 15 mL/min/1.73 m2 or the need for renal replacement therapy (i.e., dialysis).

End-stage renal disease generally refers to chronic kidney failure that requires either dialysis or transplantation .

GFR decreases with age; the renal reserve of a normal 80-yearold person is less than half that of a 40-year-old person.

Thus, creatinine concentration is often not an accurate indicator of renal function, especially in older individuals

The GFR can be reduced by 50% without a rise in creatinine concentration, while creatinine concentration does not exceed normal limits until GFR has fallen to less than 50 mL/min.

In the United States, the **leading causes** of end-stage renal disease are **diabetes mellitus** and **hypertension** .

AKI is a sudden decrease in renal function with the possible decrease in urine output.

Episodes of AKI can occur in individuals with or without CKD .

Classifying AKI into:

-prerenal,

-renal, and

-postrenal causes allows for a systematic approach.

Prerenal causes can often be differentiated by calculating the blood urea nitrogen-to-creatinine ratio.

A ratio **more than 20** suggests prerenal etiologies, with **hypovolemia** or **hypotension** the most common .

Obstruction, which results in dilated ureters and enlarged kidneys, should always be considered in the differential diagnosis of AKI.

Prompt identification with **ultrasound** should lead to attempts to decompress the outflow tract.

Patients with CKD have many **associated comorbidities**, both related to the underlying diseases that led to CKD and its resulting end-organ complications.

Cardiovascular issues include: hypertension, IHD, ventricular dysfunction (diastolic and systolic), heart failure, CVD, PAD, pericarditis, pericardial effusions, and valvular heart disease (valvular calcification with resulting regurgitation or stenosis)

CKD is also associated with **chronic anemia** due to reduced erythropoietin production by the kidneys.

While treatable with erythropoiesis stimulating agents, **complete "normalization" of hemoglobin** concentration (i.e., 135 g/L vs. 113 g/L) may actually **increase morbidity and vascular events.**

recommend using erythropoiesis stimulating agents to treat hemoglobin concentrations less than **90 g/L**, but avoid increasing the concentration **to above 130 g/L**.

Other hematological abnormalities include **platelet dysfunction** and increased bleeding, **despite normal platelet counts**, **prothrombin times**, and **activated partial thromboplastin time (aPTT)** .

Once dialysis is begun, patients become more prone to **hypercoagulable** states.

Patients with CKD can develop **autonomic and peripheral (sensory and motor) neuropathies**.

Unsurprising, CKD is associated with many **electrolyte disturbances.**

Chronic metabolic acidosis is common, but it is usually mild and **compensated for by chronic hyperventilation.**

Hyperkalemia is the most serious electrolyte disturbance.

Hypocalcemia is common in patients undergoing dialysis, although with long-term disease, secondary and tertiary **hyperparathyroidism eventually develops.**

Chronically **elevated troponin** concentrations are common in end-stage renal disease, which does influence interpretation of any postoperative troponin elevations.

Since **insulin is metabolized by the kidneys**, worsening renal function should be suspected in diabetic patients with end-stage **renal disease who develop improved glycemic control or unexpected hypoglycemia**.

Preexisting **CKD is a risk factor** for increased postoperative complications, including cardiac complications,AKI, acute stroke, and death.

In noncardiac surgery, identified **risk factors for AKI** include:

increased age, male sex, symptomatic heart failure, hypertension, liver disease (including ascites), CKD, PAD, COPD, nonelective surgery, and intraperitoneal surgery.

Nonsteroidal antiinflammatory drugs (**NSAIDs**) and cyclooxygenase-2 (**COX-2) inhibitors** **interfere with renal perfusion autoregulation** and **should be avoided or discontinued** in patients with CKD.

In contrast, these drugs do not increase the risk of postoperative AKI in patients with normal renal function.

LMWHs are cleared by the kidneys and are not removed during dialysis; thus, they have a prolonged duration of action in patients with CKD.

Similarly, dosing of **DOACs** have to be adjusted in CKD.

The preoperative evaluation of patients with CKD should emphasize the:

-cardiovascular system,

-cerebrovascular system,

-intravascular volume status, and

-electrolyte status.

The **early stages of CKD** typically cause **no symptoms**.

The **anesthesiologist should inquire about the:**

-cardiovascular systems (i.e., chest pain, orthopnea, paroxysmal nocturnal dyspnea),

-urine output, associated comorbidities, medications, dialysis schedules, and any hemodialysis catheter problems (e.g., infection, thrombosis)

Information on the patient's target and current **weight** may be helpful for assessing **volume status**.

Patients with CKD need an **ECG** and blood sampling to measure **electrolyte**, **calcium**, **glucose**, **albumin**, and **creatinine** concentrations.

Further evaluation is needed if the **ECG** shows:

-LVH (hypertension),

-peaked T waves (hyperkalemia),

-flattened T waves, a

-prolonged PR interval, or a **prolonged QT interval** (hypokalemia).

-A **chest radiograph** (infection, volume overload),
-**echocardiogram** (murmurs, heart failure), and cardiology evaluation may be necessary in some cases.

Venous access sites or **blood draws** from the brachial, cephalic (antecubital), and central veins in the nondominant upper extremity **should be avoided** in patients who may eventually **need fistulas** in those locations for dialysis

Preoperative renal replacement therapy (**dialysis**) schedules should be coordinated with the timing of the planned surgery. Dialysis is **important for correct:**

-volume overload,

-hyperkalemia, and

-acidosis before planned surgery.

Ideally, **elective surgery** should be performed about **24 hours after dialysis**.

Performance of surgery shortly after dialysis should be avoided, because of the risks of acute volume depletion and electrolyte alterations.

Specifically, **dialysis leads** to:

-fluid shifts and

-electrolyte (i.e., sodium, potassium, magnesium, phosphate) imbalance, especially related to shifting of electrolytes between intracellular and extracellular compartments.


Have nice day