- Glucocorticoids are used acutely to diminish edema associated with neoplasm or abscess and may diminish associated intracranial Htn.
- Acetazolamide to diminish CSF production and reduce intracranial Htn.
- Furosemide to acutely decrease cerebrovascular volume.
- Mannitol to decrease ICP.

Assessment Points

System	Effect	Assessment by Hx	Physical Examination	Test
CV	Bradycardia, Htn	Late signs	Pulse, BP	
RESP	Impaired respiratory drive and airway reflexes		Cranial nerve exam, stridor, swallowing abnormality	Pulse oximetry
GI	N/V, aspiration, abnormal feeding	Hx of progression of N/V		
CNS	Depressed LOC, increased ICP, headache	Timing of onset	Arousability and neurologic exam, tense fontanel, inferior eye deviation	CT scan

Key References: Bober J, Rochlin J, Marneni S: Ventriculoperitoneal shunt complications in children: an evidence-based approach to emergency department management, *Pediatr Emerg Med Pract* 13(2):1–22, 2016; Christian EA, Melamed EF, Peck E, Krieger MD, McComb JG: Surgical management of hydrocephalus secondary to intraventricular hemorrhage in the preterm infant, *J Neurosurg Pediatr* 17(3):278–284, 2016.

Perioperative Implications

Preoperative Preparation

- Assess urgency of presentation. Catastrophic increased ICP requires emergent intubation and hyperventilation. In young infants, direct neurosurgical needle puncture of a proximal lateral ventricle or previously inserted shunt may diminish ICP sufficiently to avoid a catastrophe.
- Secure IV access if possible, and consider acetazolamide 10 mg/kg IV or furosemide 1 mg/kg IV.

Monitoring

- LOC
- Routine

Airway

- Head up 10-20 degrees and midline may diminish ICP
- Aspiration risk due to gastric atony

Preinduction/Induction

- Sedatives usually are not indicated so that resp compromise or sedation does not increase ICP. Minimal sedation or use of local anesthetic can secure IV access without causing increased ICP due to pain, crying, or struggling.
- Rapid-sequence IV induction is preferred (because of aspiration risk), unless in doubt of airway anatomy.
- Debate over use of succinylcholine versus rapid-onset nondepolarizing muscle relaxant (rocuronium). Thiopental, propofol, or etomidate IV agents preferred; avoid ketamine.
- Mask induction may increase ICP by increasing cerebral blood volume. Once fontanelles are closed, the brain is limited to a closed space within the cranium; prior to that time (<18 mo), the brain has some room to expand. Sevoflurane may be the preferable agent for inhalation induction (well tolerated and minimal effects on cerebrovascular tone). Isoflurane and desflurane are associated with coughing and are not recommended for induction.
- Lidocaine 1–1.5 mg/kg IV may be useful adjunct to minimize increase in ICP due to laryngoscopy and endotracheal intubation.

Maintenance

- Volatile anesthetic (most commonly sevoflurane or isoflurane) <1 MAC, N_2O 0–70% (debatable) and opioid (i.e., fentanyl 2–5 μg/kg or equivalent).
- Maintain normothermia, cardiac output. Hyperventilation may be acutely helpful until CSF is diverted and ICP reduced.
- Normal saline at restricted or maintenance rate. Glucose support should only be administered for infants; avoid hyperglycemia.

Extubation

- Ensure return of airway reflexes, LOC, and resp drive.
- Failure of achieving above criteria may require CT scan and/or ICU monitoring.

Postoperative Period

- Usually unremarkable; depressed LOC is concern for periop ischemic insult or hemorrhage.
- EBL should be minimal.

Adjuvants

- Lidocaine, mannitol, furosemide, and spontaneous hyperventilation by pt

Anticipated Problems/Concerns

- Immediate postop neurologic exam should demonstrate improvement. If not improved, urgent CT scan and secure airway must be maintained. Postop ICU admission not required unless impaired neurologic status continues.

Hyperaldosteronism, Primary

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Risk

- Responsible for up to 20% of moderate to severe systemic arterial Htn.
- End organ damage from long-standing Htn (e.g., chronic kidney disease, cardiomyopathy).
- Abnormal glucose tolerance in up to 50% of pts with hyperaldosteronism.

Perioperative Risks

- Hypernatremia and hypervolemia with high total body sodium.
- Htn may be refractory to treatment, with increased risk of cardiovascular complications, including malignant hypertensive crisis.
- Hypokalemia and hypomagnesaemia with low intracellular potassium and magnesium may cause cardiac arrhythmia and general muscle weakness.

Worry About

- Hypertensive response to intubation or surgical incision
- Hypokalemia and associated muscle weakness or potential for arrhythmia
- Metabolic alkalosis

Overview

- Also known as Conn syndrome; described by Jerome W. Conn, University of Michigan, in 1955.
- Characterized by Htn, hypernatremia, hypokalemia, metabolic alkalosis, and low plasma renin level.
- Classically caused by a unilateral aldosterone producing adrenal adenoma.
- Primary hyperaldosteronism is a renin-independent and incompletely suppressible over secretion of the mineralocorticoid aldosterone secreted from the zona glomerulosa of the adrenal cortex.
- Aldosterone acts on the mineralocorticoid receptor in the distal convoluted tubule of the nephron and the collecting ducts to enhance sodium and water reabsorption, at the expense of potassium. Excess loss of potassium leads to loss of hydrogen ions to maintain electroneutrality.
- Usually aldosterone secretion is controlled by the renin-angiotensin feedback system in response to thirst, hypovolemia, reduced renal juxtaglomerular apparatus perfusion pressure, and reduced tubular sodium concentration.
- Aldosterone promotes restoration of circulating volume by correcting water and sodium depletion.
- Dx is by combination of clinical suspicion of persistent Htn, hypernatremia and spontaneous hypokalemia, and metabolic alkalosis in the absence of diuretics. Dx is confirmed by measuring the plasma aldosterone to renin ratio—a value over 35 ng/dL per ng/mL/h has sensitivity of 100% and specificity of 92%. Post test specificity can be improved by measuring post-sodium infusion aldosterone. Values above 7 ng/dL showed specificity of 100%.

Etiology

- 60–70% idiopathic hyperaldosteronism or bilateral idiopathic adrenal hyperplasia
- 30–40% unilateral aldosterone-producing adrenal adenoma (first described by Conn in 1955)
- Uncommon causes:
  - Unilateral adrenal hyperplasia
  - Familial hyperaldosteronism
  - Aldosterone producing adrenocortical carcinoma and ectopic aldosterone secreting tumors

Usual Treatment

- Aldosterone antagonists such as spironolactone (up to 300 mg/d) or the newer drug eplerenone (up to 100 mg/d).
- Both spironolactone and eplerenone are mineralocorticoid receptor antagonists and therefore have a potassium-sparing diuretic effect. Eplerenone is more selective for the mineralocorticoid receptor than spironolactone and has fewer glucocorticoid or androgen receptor antagonist side effects.
- Htn may be refractory to treatment with aldosterone antagonist alone and may require formal

antihypertensive therapy with ACE inhibitor or beta-blocker.
- Treatment of Htn may be needed for several wk before any benefit to periop morbidity.
- Hypervolemia should be treated with a potassium-sparing diuretic, such as amiloride, to avoid exacerbating potassium loss.
- Potassium deficit is likely to be severe and larger than apparent from serum levels.
- Unilateral primary hyperaldosteronism (from either aldosterone producing adenoma or unilateral adrenal hyperplasia) should be treated with surgical adrenalectomy.
- Bilateral primary hyperaldosteronism should be treated medically with long-term mineralocorticoid receptor antagonist therapy.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Htn, often resistant to therapy; increased sympathetic activity, cardiac output often increased except where there is hypertensive cardiomyopathy	Exercise tolerance, dyspnea, orthopnea, Htn—headache, visual disturbance	BP (compare R to L or arms with legs with coarctation) Third heart sound Fine inspiratory crackle	BP, ECG, ABG, CXR, ECHO
HEME	Hypervolemia results in Htn, hypokalemia, metabolic alkalosis, and congestive cardiac failure	Weakness, fatigue, headache	JVP, motor power	Hematocrit, serum lytes, biocarbonate, ABG
RENAL	Hypokalemia, alkalosis	Weakness, palpitations	Motor power, arrhythmia	ECG, serum and urine lytes, ABG

Key References: Jano A, Domi R, Derdica H, et al.: Anesthetic considerations of Conn syndrome, *Clin Med Res* 3(5):123–135, 2014; Reilly CS: Adrenal disease: cortex and medulla. In Hall GM, Hunter JM, Cooper MS, editors: *Core topics in endocrinology in anaesthesia and critical care*, Cambridge, 2010, Cambridge University Press, pp 45–56.

Perioperative Implications

Preinduction/Induction/Maintenance
- Correct hypokalemia and associated electrolyte disturbance (e.g., magnesium); this may require IV supplementation preop and intraop.
- Htn may be refractory; require treatment with several classes of drugs with the potential for extreme cardiovascular instability, and there may be a disproportionate hypertensive response to laryngoscopy or surgical stimulation.
- Avoid hyperventilation and hypocapnia to prevent worsening metabolic alkalemia and subsequent intracellular potassium shift.
- Anticipate increased sensitivity to nondepolarizing neuromuscular antagonists due to hypokalemia. Consider using drugs with spontaneous organ independent metabolism (e.g., atracurium or cisatracurium, or rocuronium and sugammadex combination).

Monitoring
- Consider arterial pressure monitoring.
- Consider central venous cath insertion for access and administration of concentrated potassium and magnesium in the face of hypervolemia, or for monitoring filling pressure where there is cardiomyopathy and heart failure.
- Urinary cath.
- Peripheral nerve stimulation for neuromuscular function.

General Anesthesia
- Hypokalemia may potentiate muscle relaxants and arrhythmia.
- Surgical manipulation of the aldosterone producing structure may cause severe Htn.
- High pH decreases availability of intracellular calcium.

Regional Anesthesia
- Local anesthetic mixtures with sympathomimetic may exacerbate preexisting Htn.
- Altered pharmacokinetics resulting from hypervolemia and end organ damage such as renal dysfunction may need dose adjustments.

Postoperative Period
- Appropriate care predicated on surgical procedure, co-morbidities, and hemodynamic stability.
- Monitor and correct ongoing electrolyte abnormalities.
- Consider glucocorticoid supplementation, though it should not be required if there is at least one intact adrenal gland.

Anticipated Problems/Concerns
- Labile blood pressure.
- Increased sympathetic activity leads to activation of the renin-angiotensin system.
- Arrhythmia from severe hypokalemia.
- Generalized muscle weakness from hypokalemia.

Hyperaldosteronism, Secondary

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Risk
- High renin states, and greater risks may be associated with the primary problem, leading to hyperreninemia.
- End organ damage from long-standing Htn (e.g., chronic kidney disease, cardiomyopathy).
- Abnormal glucose tolerance in up to 50% of pts with hyperaldosteronism.

Perioperative Risks
- Risks include hypernatremia and hypervolemia with high total body sodium.
- Htn may be refractory to treatment, with increased risk of cardiovascular complications, including malignant hypertensive crisis.
- Hypokalemia and hypomagnesaemia with low intracellular potassium and magnesium may cause cardiac arrhythmia and general muscle weakness.

Worry About
- The underlying primary medical disorder that leads to increased renin and, hence, increased aldosterone secretion.
- Hypertensive response to intubation or surgical incision.
- Hypokalemia and associated muscle weakness or potential for arrhythmia.
- Metabolic alkalosis.

Overview
- Secondary hyperaldosteronism is a renin-dependent oversecretion of the mineralocorticoid aldosterone secreted from the zona glomerulosa of the adrenal cortex.
- Renin is released from the JGA as a response to decreased renal perfusion pressure. Osmoreceptors in the macula densa will also stimulate renin release in the presence of decreased sodium concentration in the distal tubule.
- Renin enzymatically alters angiotensinogen to angiotensin I. ACE (found in the pulmonary and renal vascular endothelium) then converts angiotensin I to angiotensin II. Angiotensin II, a potent vasoconstrictor, then stimulates release of aldosterone from the zona glomerulosa of the adrenal medulla.
- Aldosterone promotes restoration of circulating volume by correcting water and sodium depletion.
- Diagnosis is suggested by increases in both plasma renin (>2 ng/mL) and aldosterone, but the ratio of plasma aldosterone concentration to renin activity is <10 ng/dL per ng/mL/h (ratio >35 strongly suggests primary hyperaldosteronism).
- In some situations, such as pregnancy and chronic renal disease, increased aldosterone is an adaptive response and is not necessarily deleterious.

Etiology
- Any pathophysiologic process that causes a chronic and relative decrease of perfusion pressure in the juxtaglomerular apparatus has the potential to cause secondary hyperaldosteronism. Examples include:
  - Increased central venous pressure and therefore increased capillary hydrostatic pressure.
  - Decreased cardiac output.
  - Vasodilation.
  - Impaired plasma protein synthesis and excess loss.
- Common causes:
  - Heart failure (increased venous pressure and reduced cardiac output).
  - Liver disease with cirrhosis (peripheral and splanchnic vasodilation and reduced albumin synthesis).
  - Nephrotic disease (protein loss).
- Renovascular (hyper-reninemic) Htn: Related to atherosclerosis (renal artery stenosis) or fibromuscular dysplasia.
- Renovascular disease causing decreased renal perfusion pressure independent of systemic pressure: Renal artery stenosis, aortic coarctation.
- Renin-secreting tumors (e.g., tumor of the juxtaglomerular apparatus or renal cell carcinoma).
- Pregnancy.