Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Virus access to CNS from nasal mucosa to olfactory bulb and olfactory tracts	Preceding URI		Nasopharyngeal swab Throat culture
CV	Autonomic dysfunction Neurogenic stunned Myocardium	Transient myocardial dysfunction	Labile BP, HR	ECG, troponin, CK, ECHO, left ventricular angio
HEME	Increased or normal WBC			CBC, WBC differential, serum antibody titers
RENAL	SIADH	Water intoxication Anorexia N/V Personality disorders Neurologic abnormality	No evidence of volume depletion Normal skin turgor Normal BP Mental status changes from lethargy to coma	Serum Na^+ and osmolality Urine Na^+ and osmolality BUN, Cr
CNS	Focal, global neurologic disturbances	Fever Headache Seizure Personality change Memory loss Confusion Weakness Sleep/awake abnormality Hearing, speech, visual changes	Focal neurologic deficits, altered mentation, papilledema, anisocoria; if spinal cord involvement: flaccid paraplegia, increased DTRs	CSF: Cell count (increased WBC, lymphocyte predominance), protein (increased), Gram stain, viral and bacterial culture, antibodies, antigens, viral PCR, viral DNA sequencing, MRI (temporal lobe involvement, hemorrhagic lesions, ±mass effect), EEG, CT

Key References: Tunkel AR, Glaser CA, Bloch KC, et al.: The management of encephalitis: clinical practice guidelines by the Infectious Diseases Society of America, *Clin Infect Dis* 47(3):303–327, 2008; Pryzbylkowski PG, Dunkman WJ, Liu R, et al.: Case report: anti-*N*-methyl-D-aspartate receptor encephalitis and its anesthetic implications, *Anesth Analg* 113(5):1188–1191, 2011.

Preoperative Implications

Preoperative Preparation

- Document neurologic exam.
- Elicit Hx of increased ICP or seizure.
- If suspicion of anti-NMDA receptor encephalitis:
  - Concern for anesthetic interaction with a dysregulated NMDA receptor (i.e., propofol, midazolam, methadone, N_2O).
- If SIADH present, correct electrolyte and free water abnormality.
  - Sodium administration or fluid restriction depending on severity of hyponatremia.
  - Beware of central pontine myelinolysis with rapid correction of hyponatremia.

Monitoring

- Standard ASA monitors
- Invasive monitors when indicated; arterial line if ICP is an issue
- If EVD in use, continue monitoring ICP in the OR
- Lytes

Airway

- None

Induction

- Potential for hyperkalemic response to depolarizing NMBs if myopathy, paralysis, or prolonged immobilization; prefer use of nondepolarizing NMBs
- Autonomic instability and labile hemodynamics

Maintenance

- If pt is receiving seizure prophylaxis (e.g., phenytoin [Dilantin], carbamazepine [Tegretol], phenobarbital [Luminal], primodone [Mysoline], valproic acid [Depakote]) be aware of potentiation of sedative effects and alteration of hepatic metabolism of anesthetics and muscle relaxants.

Extubation

- Delayed awakening
- Seizures on emergence

Postoperative Period

- Delirium
- Other neurologic deterioration, including clinical or subclinical seizures

Anticipated Problems/Concerns

- Delayed emergence.
- SIADH, careful selection of replacement fluid.
- Hyperkalemic response to succinylcholine.
- Universal precautions for contact with infected materials; sterilization of reusable instruments.
- Use disposable instruments, specifically with JC virus disease.

Encephalopathy, Hypertensive

Shane V. Cherry | Christian Diez

Risk

- Chronic Htn, renal disease (particularly end-stage renal disease), malignancy, sympathomimetic drugs, and a history of transplantation and immunosuppressive therapies

Perioperative Risks

- Increased risk of myocardial ischemia, ventricular dysrhythmias, HF, aortic dissection, cerebral hemorrhage, coma, long-term neurologic disability, renal failure, or sudden death

Worry About

- Myocardial ischemia or infarction
- Aortic dissection
- HF
- Pulm edema
- Cerebral infarction (ischemic or hemorrhagic) or intracranial hemorrhage
- Acute renal failure
- Eclampsia in at-risk parturients
- Microangiopathic hemolytic anemia

Overview

- The most common clinical presentations of hypertensive emergencies are cerebral infarction (24.5%), pulm edema (22.5%), hypertensive encephalopathy (16.3%), and HF (12%).
- Hypertensive encephalopathy is by definition a hypertensive emergency and has recently come to fall under the umbrella term PRES.
- Hypertensive encephalopathy is a relatively rapidly evolving syndrome of severe Htn in association with (most commonly) seizures, headache, visual disturbances, altered mental status, vomiting, ataxia, and focal neurologic deficits that may become rapidly fatal.
- Occurs when the systemic BP is elevated beyond the cerebral autoregulatory threshold of MAP, typically greater than 160 mm Hg ("autoregulation breakthrough").
- Differential Dx: Ischemic or hemorrhagic stroke (particularly posterior circulation stroke), toxicology syndrome from drugs of abuse (e.g., cocaine), encephalitis, and venous sinus thrombosis.
- It is critically important to distinguish between ischemic stroke and hypertensive encephalopathy because the treatment for hypertensive encephalopathy is lowering of BP, whereas outcomes are improved with higher BPs after acute ischemic stroke and therefore antihypertensives are generally not recommended.
- Hypertensive encephalopathy can develop in pts with or without chronic Htn. However, because the cerebral autoregulation curve is shifted to the right in chronically hypertensive pts, it may take significantly higher BPs for these pts to develop signs of encephalopathy.
- As the name implies, PRES is usually reversible if diagnosed early and treated appropriately but can quickly become irreversible and fatal.
- Diagnostic test of choice is MRI, which will reveal symmetric reversible T2 high signal intensities located in the occipital and parietal lobes as a result of subcortical vasogenic edema. CT is not sensitive for the lesions of PRES and will often be normal.

Etiology

- The critical event is failure of cerebral autoregulation (for any reason) leading to cerebrovascular endothelial dysfunction and vasogenic edema that renders the pt encephalopathic.
- Leading theory of pathophysiology is that in the presence of increased cerebral perfusion pressure, the increased capillary hydrostatic pressure leads to vasogenic edema and may even disrupt the blood-brain barrier.
- Chemokines and cytotoxic agents may play a role in the endothelial dysfunction, as evidenced by the existence of PRES in normotensive and even hypotensive pts (e.g., undergoing chemotherapy or septic shock).
- Nonhypertensive causes and associations of PRES include:
  - Endocrine disorders: Pheochromocytoma, renin-secreting tumor, Cushing disease, and Conn syndrome.
  - Drug induced: Immunosuppressive therapy, chemotherapy, erythropoietin, MAOIs, abrupt discontinuation of antihypertensive drugs, sympathomimetic drugs, and drugs of abuse (e.g., cocaine, amphetamines, LSD).
  - AIDS.
  - Thrombotic thrombocytopenic purpura.
  - Status post CEA (CEA hyperperfusion syndrome).
  - Preeclampsia and eclampsia.
  - Acute intermittent porphyria.
  - Autonomic hyperreactivity, as with spinal cord lesions.

Usual Treatment

- Placement of arterial cath for beat-to-beat BP monitoring.
- Reduce MAP by 20% within the first h or to a target diastolic BP of 100–110 mm Hg, whichever value is greater.
- Parenteral antihypertensive agents: Most commonly used agent is sodium nitroprusside. Other common alternatives include labetalol, fenoldopam, nicardipine, enalaprilat, or hydralazine. Nitroglycerin has been reported to exacerbate symptoms and is contraindicated.
- In general, vasodilators, such as sodium nitroprusside, have a lesser effect on the cerebral circulation versus other vascular beds, making them primary choices for pts with hypertensive encephalopathy. Clonidine should be avoided because of its CNS depressant effects.
- Anticonvulsant therapy: Preferentially use agents that can be rapidly loaded, such as benzodiazepines or phenytoin/phenobarbital.
- Withdrawal of exacerbating factors (e.g., corticosteroids, immunosuppressive drugs).
- In eclampsia, delivery of the fetus and the placenta, as well as parenteral magnesium, are the mainstay of therapy.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Htn Myocardial ischemia HF Aortic dissection	Htn Angina CHF Pain radiating to the back Preeclampsia	S_3 S_4 gallop JVD	ECG CXR BMP, CBC with peripheral smear Arteriogram
RESP	Pulm edema Decreased lung compliance Decreased FEV, FVC	Dyspnea Frothy sputum Orthopnea	Rales	CXR O_2 sat
GI	Vomiting	Abdominal pain	Abd mass	CT Plasma metanephrine levels
RENAL	Renal failure	Anuria		UA Measurement of UO
CNS	Cerebral infarction or intracranial hemorrhage Seizures Severe headache Visual disturbances Focal deficits Stupor, coma	Mental status exam	Altered mental status Retinal arteriolar spasm on ophthalmoscopy Papilledema May have a normal fundoscopic exam Neck stiffness Pronator drift	MRI

Key References: Granata G, Greco A, Iannella G, et al.: Posterior reversible encephalopathy syndrome—insight into pathogenesis, clinical variants and treatment approaches, *Autoimmun Rev* 14(9):830–836, 2015; Vaughan CJ, Delanty N: Hypertensive emergencies, *Lancet* 356(9227):411–417, 2000.

Preoperative Implications

Preinduction

- Determine home medications, compliance with antihypertensive regimens, and adequacy of BP control.
- Evaluate for end-organ damage.

Monitoring

- Arterial cath.
- Central venous cath or PA cath may be used if extensive surgery is planned or there is evidence of other end-organ damage (e.g., left ventricular dysfunction, renal failure).

General Anesthesia

- Volatile anesthetics are useful in attenuating sympathetic nervous system pressor responses; there is no evidence to suggest one volatile agent over another for control of intraop Htn.
- Nitrous oxide-opioid technique can be used be used for maintenance of anesthesia in pts with labile pressure while under GA; a volatile agent may be needed during periods of abrupt changes in surgical stimulation.
- Antihypertensive agent by continuous infusion is frequently a more effective means of controlling BP when compared with titrating volatile agent to BP.

Induction

- Induction of anesthesia may produce an exaggerated decrease in BP, particularly in the presence of diastolic Htn (intravascular volume depletion).
- Direct laryngoscopy and tracheal intubation can produce significant Htn; limit the duration of laryngoscopy and consider the use of opioids, lidocaine, beta blockers, and vasodilators to blunt the autonomic response.

Maintenance

- Control BP and minimize wide fluctuations because overly aggressive treatment of Htn may worsen other end-organ function.
- Monitor for myocardial ischemia.

Regional Anesthesia

- Epinephrine-containing solutions may place the hypertensive pt at risk or worsen an existent hypertensive crisis (e.g., epidural in a preeclamptic parturient).

Postoperative Period

- Extubate with careful BP control.
- Continue parenteral antihypertensive therapy and monitoring of invasive BP and mental status during transport and recovery.
- Maintain monitoring for other end-organ morbidity, such as myocardial ischemia, cardiac dysrhythmias, HF, stroke, and bleeding.

Anticipated Problems/Concerns

- Particular caution is necessary with the elderly, as well as pts with chronic Htn; overaggressive reduction in BP may worsen mental status and cause stroke.