SECTION F – TRAUMA, DM, HEME, GI, HTN

Chapter 27: A 55-year-old healthy man with elevated blood pressure

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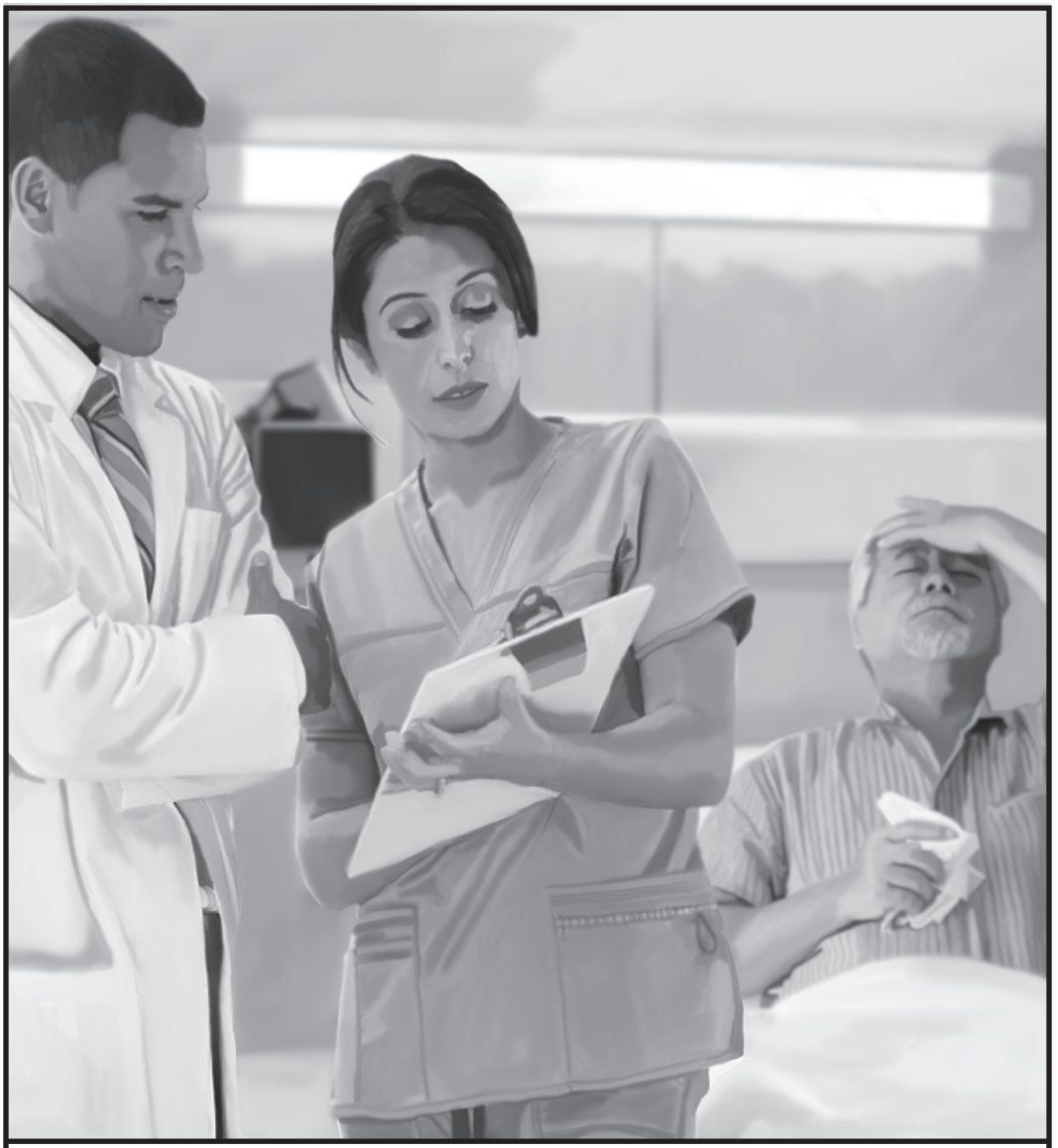

"WE NEED TO GET THE BP DOWN ASAP!"

SECTION F / CHAPTER 27

A 55-year-old healthy man with elevated blood pressure

Hypertension (HTN) is a staggeringly common condition throughout the world, thought to affect a third of Americans and an even greater fraction of patients presenting to the emergency department.[1, 2, 3] An ED patient with an elevated blood pressure (BP) presents a diagnostic and therapeutic challenge, as the spectrum of disease can span from the asymptomatic, chronic patient who needs little more than primary care referral, to the critically ill who require prompt and expert intervention. Furthermore, inconsistent terminology has compounded uncertainty and practice variation. Hypertensive emergency is diagnosed when end-organ injury (i.e., cardiovascular, renal, or neurologic) occurs in the setting of markedly elevated BP.[4]

In the absence of treatment, hypertensive emergency has a very poor prognosis, with 1-year mortality rates reported as high as 79%.[5] Even with prompt treatment the residual risk for end-stage renal disease and cardiovascular complications remains high.[6] In 1939, Keith, et al. found that the mean survival of patients with hypertensive emergency was 10.5 months, with no survivors at 5 years.[7] Appropriate treatment has dramatically reduced immediate deaths and 1-year mortality, with rates ranging between 4% in one European registry and 7–10% in U.S. cohorts.[8, 9, 10]

This case is *real*, including the documentation and medical decision-making. We start with this patient's initial presentation and—yes—the bounceback just one day later.

INITIAL ED VISIT

HPI (21:22): The patient is a very pleasant 53-year-old gentleman with no documented past medical history (nonsubscriber to routine medical care) who presents with complaint of high BP. He reports checking his BP today via a local pharmacy's automated cuff and became alarmed when it read "over 200," and thus he opted to present for emergency evaluation. Otherwise, the patient is without complaint. Specifically, he denies chest pain, shortness of breath, visual changes, or headache. He is well-appearing.

Allergies: NKDA
Social history: Patient denies tobacco abuse, EtOH abuse, or IVDA
Family history: Reviewed and not pertinent
ROS: All other systems reviewed and are negative except as noted. Nursing triage notes were reviewed
PMH: None
PSH: None

PE:

Vitals:				
Temp	**Pulse**	**Resp**	**BP**	**SpO_2**
97.8	**86**	**16**	**202/135**	**99% (RA)**

GA: A&OX3, WA/WN
HEENT: NC/AT, PERRL, TMI, nares patent, trachea midline
CV: RRR s m/r/g/t
RESP: CTAB s wheezes, rales, or rhonchi
ABD: Soft, NT/ND. No palpable midline abdominal mass
BACK: No CVA tenderness
SKIN: No rashes or lesions
NEURO: CN II-XII grossly intact

EKG (21:28): Reviewed and interpreted by me. NSR at 80bpm. LAD, LVH. Normal intervals. No acute ischemic changes, no STEMI.

MDM (21:45): Patient presents with asymptomatic hypertension. There is no evidence of Hypertensive Emergency—no chest pain, SOB, neurologic deficit. Per ACEP guidelines, no indication for further testing. Will refer patient to f/u with PCP.

IMPRESSION (21:52): Hypertension, unspecified

PLAN: F/u with PCP in 48-72h. RTER with worsening symptoms.

At face value, this appears to be a relatively simple and well-documented encounter. It is a complaint we face on a regular basis, so common, in fact, that the American College of Emergency Physicians (ACEP) has developed a clinical policy,[11] which the treating physician even cites in their medical decision-making! Nonetheless, there are multiple areas where the management and documentation can be improved.[7]

Documentation and patient safety issues:

1. **Speed of disposition:** We are often pressured to rapidly evaluate and manage the disposition of patients, but occasionally, the waiting room thins and the department hums along at top efficiency, allowing for rapid management and disposition of patients with lower-acuity complaints. This patient was discharged 30 minutes after arrival. We are held to task for moving too slowly, but moving too quickly may retrospectively be interpreted as being careless.
2. **There's only one set of vital signs recorded:** With the exception of minor complaints, patient vitals should be trended, and nearly always repeated prior to discharge. This is especially true in a patient presenting with a complaint so pertinent to vital signs—an elevated BP! Subsequent BP readings taken in the ED—more often than not—will demonstrate a reduction from triage vitals, which helps to establish a reassuring trend and justifies lack of intervention. If the next BP is 160/90, great… if it is 260/160, maybe our management would be different.

3. **Physical exam:** "CN II-XII grossly intact" is an inadequate neurologic evaluation for this patient in whom the diagnosis is predicated upon an exclusion of focal neurologic deficits. There is no documentation regarding cerebellar motion testing, the presence or absence of papilledema or spontaneous venous pulsations, or the patient's gait.
4. **MDM:** At first glance, citing the ACEP clinical policy regarding asymptomatic elevated BP seems like a good strategy; however any benefit to such charting is nullified (and, indeed, likely destructive) when *this statement is incorrect.* What? We'll discuss in more detail below.
5. **Patient discharge instructions:**
    - It is unclear whether the importance of follow-up for the commencement of antihypertensive therapy was discussed with the patient.
    - There is uncertainty of his ability to obtain timely follow-up. The physician had discussed the patient's non-participation in medical care earlier in the documentation—one would assume that means he doesn't have a primary care provider.
    - Most importantly, the patient is instructed to "RTER with worsening symptoms." What symptoms, exactly? Isn't his whole plan of care predicated upon the fact that this is *asymptomatic* HTN?

Were the decisions regarding the patient's evaluation and management appropriate? For such a common presentation, there is significant practice variation as well as confusion regarding clinical recommendations.

1. What is the role of the emergency clinician in managing patients with asymptomatic HTN?

The majority of patients who present to the ED with elevated BP receive no intervention or instructions regarding their HTN. Recognizing the frequency of ED visits and the paucity of evidence regarding management, the American College of Emergency Physicians (ACEP) released a clinical policy addressing critical issues in the evaluation and management of asymptomatic HTN in 2006 and later amended the document in 2013.[11]

The ACEP clinical policy offers a Level "C" recommendation (i.e., there is an absence of any adequate published literature and is based on panel consensus) that patients with asymptomatic elevated BP do not require routine screening for acute target organ injury, nor do they require routine intervention with antihypertensive medications. It is recommended that patients should be referred for timely outpatient follow-up.[11]

2. What, if anything, changes when the patient is uninsured, has no primary care provider, or is known to be noncompliant with medical care?

The ACEP policy makes specific exceptions for patients in whom poor follow-up or medical noncompliance is anticipated with a consensus recommendation that screening for end-organ injury and/or initiation of antihypertensive therapy may reduce the rate of adverse outcomes related to HTN. Several studies have demonstrated higher rates of kidney disease in populations at higher risk for medical noncompliance, suggesting that ED screening and intervention may offer benefit.[12]

3. Should antihypertensive medications be initiated from the ED?

Generally speaking, initiation of antihypertensive therapy from the ED is unnecessary. There are compelling data, however, to suggest that prescription of antihypertensive medications from

the ED is associated with improved BP management at follow-up without any increased risk of adverse events or hypotension, and most experts recommend this approach, particularly in at-risk populations.[13,14]

Putting it all together:
The patient who presents to the emergency department with a chief complaint of "high BP"—that is, the patient with asymptomatic HTN—presents a "fork in the road" for work-up and management. The dearth of high-quality literature examining this population is somewhat reconciled by the ACEP Clinical Policy Statement. HTN represents a chronic issue best managed through primary care resources; however, the unique position of the ED as both the first line for acute care and a safety net for all care, means that the idiosyncrasies of each presentation must be weighed when considering the need for screening or management from the ED. Most patients do not require lab testing, electrocardiograms, or imaging and can be discharged with a plan for appropriate and expedient follow-up. Where social, situational, or other factors might delay or derail that approach, consideration should be given to escalation of intervention in the ED.

Section editor comments (Shiber):

Note that the patient's MAP is ~160. At this rate they are above cerebral vascular autoregulation; the cerebral blood flow (CBF) will be high leading to edema and an elevated intracranial pressure (ICP). A hypertensive emergency WILL happen, it is only a matter of time. Consider that the non-invasive cuff only truly measures a MAP and then uses an algorithm to calculate the SBP and DBP, so that the MAP is actually more accurate. The exam documented the heart exam as RRR s m/r/g/t. Often an S4 is present at this BP indicating a stiff LV as a result of a very high aortic arterload. Finally, I agree with the authors on the minimal neurologic exam; it is very easy to test their gait, as well as other aspects of cerebellar function such as smooth tracking movements and rapid alternating movements.

ED BOUNCEBACK

The patient did not initiate follow-up with primary care. The morning after ED discharge the complained to his wife of headache and had several episodes of vomiting. The following evening his wife found him seizing in a downstairs bathroom, and EMS arrived to find the patient with persistent seizures.

ED VISIT #2

(one day after initial ED visit)

HPI (23:02): The patient is a 53-year-old male who presents BIBA with seizures, no known seizure disorder. He was seen here yesterday evening with asymptomatic HTN and referred to f/u with his primary care doctor. Per the patient's wife, he has been complaining of headache and nausea with vomiting since shortly after discharge. He was found seizing shortly PTA. EMS responded and administered 2mg IV Lorazepam without improvement.

Allergies: NKDA
Social history: Patient denies tobacco abuse, EtOH abuse, or IVDA

Family history: Reviewed and not pertinent
ROS: ROS unobtainable due to severity of condition. Nursing triage notes were reviewed
PMH: None
PSH: None

PE:

Vitals:				
Temp	**Pulse**	**Resp**	**BP**	**SpO2**
99.8	**126**	**8**	**224/140**	**94% (RA)**

GA: Acutely ill. Afebrile
HEENT: NC/AT, pupils 6mm, sluggish.
CV: Tachycardic, no murmur
RESP: CTAB s wheezes, rales, or rhonchi
ABD: Soft, NT/ND. No palpable midline abdominal mass
BACK: No CVA tenderness
SKIN: No rashes or lesions
NEURO: Active tonic-clonic seizure activity.

MDM (23:15): Patient presents with seizures, hypertension, recent complaint of headache. Presumed SAH. Will control seizure activity and proceed immediately to CT.

Author comments (Weinstock):

We are faced with a patient who needs emergent neuroimaging, but the seizures need to be controlled first. While the team establishes IV access and you gather additional history from EMS, we arrive at:

DECISION POINT A – YOU ARE FACED WITH 3 QUESTIONS:

1. **What is the differential diagnosis for the patient presenting with elevated BP and seizure?**
2. **What is the recommended strategy for anti-epileptic therapy?**
3. **In the patient with recalcitrant status epilepticus requiring endotracheal intubation, what is the best approach to medication selection?**

What does the literature say?

Q#1: What is the differential diagnosis for the patient presenting with seizures and HTN?

Establishing a focused but thorough differential diagnosis is critical to help direct initial therapy. While we are most suspicious for subarachnoid hemorrhage, alternate possible diagnoses include:

- hypertensive emergencies such as intraparenchymal hemorrhage or posterior reversible encephalopathy syndrome
- an intracranial mass
- head trauma
- an underlying seizure disorder
- alcohol withdrawal seizures and delirium tremens

- severe electrolyte abnormalities (especially hyponatremia or hypoglyecmia)
- drug overdose
- meningoencephalitis

Q#2: What is the recommended strategy for anti-epileptic therapy?

ACEP's clinical policy on seizures[15] is concordant with guidelines by the American Academy of Neurology and Neurocritical Care Society with a recommendation to use a parenteral benzodiazepine as first line therapy, with Level A data supporting the use of lorazepam (0.1mg/kg/dose, max: 4mg/dose) or midazolam (5–10mg).[16] IV lorazepam has classically been employed to reasonable effect; however, lessons from the RAMPART Trial (a sentinel trial regarding EMS management of status epilepticus published in the *New England Journal of Medicine* in 2012), have suggested that intramuscular midazolam may lead to more rapid seizure control in situations where IV access is not already established.[17]

The use of anti-epileptic medications in patients who have failed multiple doses of benzodiazepines is less clear. Emergency physicians may consider intravenous administration of the following medications, which offer similar efficacy and carry Level B recommendations:

- phenytoin
- fosphenytoin
- phenobarbital
- valproate

The use of levetiracetam for patients who have failed initial abortive therapy has grown, and several small studies have suggested that it is likely as effective as more traditional therapies such as phenytoin, with a lower incidence of adverse effects.[18,19]

Q#3: In the patient with recalcitrant status epilepticus requiring endotracheal intubation, what is the best approach to medication selection?

Patients who have received large doses of benzodiazepines, or who continue to seize despite a secondary anti-epileptic agent, often require endotracheal intubation in anticipation of respiratory failure or for airway protection. If further sedation is needed, etomidate (0.3 mg/kg) is a hemodynamically neutral hypnotic agent with rapid onset.[20]

Long-acting paralytics are contraindicated in patients with status epilepticus unless continuous EEG can be expeditiously implemented due to the risk of unrecognized continued seizure activity. There are no data to suggest that the use of depolarizing neuromuscular blockade may precipitate critical hyperkalemia as it is unlikely that the muscle activity induced by neuromuscular blockade will be of any significance in a patient with prolonged seizure activity.

Section editor comments (Shiber):

Rapid sequence intubation (RSI) for any seizure should preferably use succinylcholine so that a clinical seizure can be detected. Additionally, many SZ patients are intubated primarily for airway protection from the sedation due to the benzodiazepines and their postictal state; these patients do not need ongoing sedation but to simply "wake up" in order to be evaluated for extubation.

DECISION POINT A – *What did the actual provider do?*

(23:05): Upon patient arrival, the provider administered a second dose of 2mg IV lorazepam (first dose administered by EMS). Labs including CBC, BMP, UA, Troponin, CK, PT/PTT, and bedside glucose—this was reported by the RN as 86mg/dL. When seizures did not abate, a third dose of lorazepam was given and 1000mg of levetiracetam ordered; however, the patient became hypoxic and the decision was made to intubate for airway protection and further anti-epileptic management.

(23:15): The patient was given etomidate 20mg and succinylcholine 100mg and then intubated via direct laryngoscopy with an 8.0 endotracheal tube. The intubation was uneventful.

The story continues: The patient is now intubated, but the encounter is not over. Sedation and analgesia are required before transfer to the CT scanner. What are the best options? Should this be done with concomitant BP management? We are at:

DECISION POINT B – YOU ARE FACED WITH 2 QUESTIONS:

1. **What is the best approach for post-intubation analgesia and sedation?**
2. **Should the blood pressure be treated prior to imaging?**

What does the literature say?

Q#1: What is the best approach for post-intubation analgesia and sedation?

Many common sedative and amnestic agents possess no analgesic properties, and analgesia should be prioritized in post-intubation care. Fentanyl, a synthetic opioid with rapid titratability and short half-life is a good choice, either as an infusion (0.7–10mcg/kg/h) or in boluses.

A comprehensive review of sedation in the critically ill patient concluded that despite nearly 100 trials examining different sedation regimens, no drug was clearly superior to its comparators.[21] Propofol infusion (5–50mcg/kg/min), is the most commonly used anesthetic for sedation in critically ill patients as well as for general anesthesia in the United States.[22] Its rapid rate of clearance, short duration of action and ease of titration offer distinct advantages in the emergency setting. The use of propofol in patients with status epilepticus is generally regarded as an ideal agent for sedation and further treatment.[23] Propofol is a global CNS depressant and GABA agonist, which has been well-demonstrated to suppress epileptiform activity within seconds after administration.[24] It may cause hypotension due to both vasodilatation and a negative inotropic effect, which may limit application in hypovolemic and hypotensive patients.

Section editor comments (Shiber):

Only if the patient has a concomitant painful condition, as having an ETT and being restrained may not be comfortable but it is not painful; the minute volume (MV) should be adjusted to support the patient comfortably so that you are not simply making them tolerate the ventilator. All opiates suppress spontaneous respirations which is not the goal for our patient nor the majority of patients intubated in the ED. Fentanyl and all opiates will induce sedation, but this is actually a side-effect; a general tenet of medicine is to use a medication for its primary intended effect and not for its side-effects.

Q#2: Should the blood pressure be treated prior to imaging?

Empiric antihypertensive treatment likely offers little benefit in the undifferentiated hypertensive emergency because the goal BP and medications of choice vary depending on diagnosis; the concept that "minutes count" lacks validity in light of the potential harm of overcorrection. With intracerebral hemorrhage, the best available data suggest that aggressive anti-hypertensive therapy leads to no difference in outcomes of death or disability,[25] and most cohorts consistently demonstrate around a 10% incidence of iatrogenic hypotension following initiation of antihypertensives,[26] suggesting that risk outweighs benefit until a condition amenable to therapy can be identified.

DECISION POINT B – *What did the actual provider do?*

Lorazepam 4mg IV was administered for post-intubation sedation. No antihypertensives were administered prior to imaging.

The story continues:

(23:53): The patient is taken to CT by nursing and respiratory therapy. The treating physician reviews the images in real time and documents "wet read by emergency physician: no large intracranial hemorrhage identified." Initial lab results have returned:

WBC 12.1 (H)
Hgb 11.8gm/dL (L)
Plt 248thou/mcl

Na 147mEq/L (H)
Cl 107mEq/L
K 6.3mEq/L (H)
HCO3 12mEq/L (L)
Creat 4.8mg/dL (H)

Trop 0.05 (H)
CK 4300 (H)

ED course: When patient returns from CT he is noted to have irregular shaking movements. There is debate among the treating physician and resident as to whether this represents seizure activity or agitation and inadequate sedation. Repeat vitals are taken and an ECG ordered which is not able to be interpreted due to patient movement.

T 99.8 **P** 126 R 32 **BP** 228/142 **SpO2** 99%

MDM (00:44): The levetiracetam has arrived we have started the infusion. I ordered propofol infusion to be started at 20mcg/kg/min. CT brain negative for acute intracranial pathology. Patient was noted to have acute renal failure and mild rhabdomyolysis, likely due to prolonged seizure activity.

Author comments (Weinstock):

How should the blood pressure be managed? We are at:

DECISION POINT C – YOU ARE FACED WITH 3 QUESTIONS:

1. **What is the management of hyperkalemia in the setting of acute renal failure?**
2. **What is the patient's presumed diagnosis?**
3. **What is the best approach to emergent antihypertensive therapy?**

What does the literature say?

Q#1: What is the management of hyperkalemia in the setting of acute renal failure?

The emergency treatment of severe hyperkalemia is generally independent of its etiology, and because the therapy is safe, maintain a low threshold for starting treatment.[27] Any ECG manifestations of severe hyperkalemia demand immediate therapy. Electrocardiographic changes are generally progressive and proportional to the rate of rise of potassium; however, ventricular fibrillation may be the first ECG disturbance of hyperkalemia.[28] Treatment should be initiated at 6.5mEq/L, the point at which most patients will begin to manifest ECG changes beginning with peaked T waves, progressing to widening of the QRS complex at potassium levels above 7mEq/L. The risk of sudden cardiac arrest rises significantly at levels above 8mEq/L.

Treatment begins with:

1. IV calcium, usually as 1 amp (1000mg) of calcium gluconate. Calcium directly antagonizes the myocardial effects of hyperkalemia without lowering serum potassium and is beneficial even in patients with normal calcium levels.[29] Calcium should always be given when ECG manifestations of severe hyperkalemia are noted (a widening QRS); however, routine use of calcium based on the absolute potassium values or with isolated peaked T waves is discouraged, as intravenous calcium can lead to cardiac dysrhythmias, tachycardia, and elevated BP.
2. Insulin and dextrose – Efforts to force potassium to redistribute intracellularly should be undertaken, typically with regular insulin (10U IV) (with dextrose administered prior to insulin when blood glucose is < 350mg/dL to support blood glucose levels).
3. Beta-agonists (albuterol) given as nebulization solution (20mg over 10 minutes), and occasionally.
4. Sodium bicarbonate when the pH of the blood is acidotic.

Note that all of the above interventions are temporizing solutions intended to shift potassium intracellularly as kaliuria corrects the systemic potassium overload. Enhanced renal elimination in the non-anuric patient is potentially definitive, and it may be accomplished with loop diuretics. The oliguric or anuric patient may occasionally require emergent hemodialysis. Exchange resins such as sodium polystyrene sulfonate (Kayexalate) have never demonstrated efficacy in decreasing serum potassium levels acutely. Additionally, a serious but infrequent side-effect—the development of colonic necrosis in about 2% of patients receiving SPS—make it a poor choice for hyperkalemia.[29]

Q#2: What is the patient's presumed diagnosis?

In the presence of normal imaging and the patient's history of headache, seizure, and altered mental status, the lack of compelling evidence for infection, and his severely elevated BP, the patient most likely has Posterior Reversible Encephalopathy Syndrome, or PRES. Once termed hypertensive encephalopathy, that nomenclature has been eschewed since a landmark paper in 1996 married the clinical diagnosis of hypertensive encephalopathy with the radiologic syndrome of posterior leukoencephalopathy to arrive at PRES.[30] PRES is a disorder of reversible subcortical vasogenic edema in patients with acute neurologic symptoms and characteristic radiographic features. Clinical manifestations include:

- non-localized headache unresponsive to analgesics
- visual disturbances
- altered mental status
- seizures in the setting of acute HTN or cytotoxic therapies[31]

Aside from ICU admission and critical care consultation, our patient would benefit from specialist evaluation by neurology for the presumed diagnosis of PRES as well as nephrology for his acute renal failure.

Q#3: What is the best approach to emergent antihypertensive therapy?

The short-term goal for treatment of PRES is to reduce the systolic BP by 10–15% within the first hour and by 25% within the following several hours.[32] Some sources recommend a target of less than 160/100mmHg; however, no randomized clinical trials have been undertaken assessing therapeutic interventions or BP goals.[33] As such, the choice of antihypertensive agent is left to the discretion of the treating physician. Nicardipine (at a starting dose of 2.5–5mg/h, max dose: 15mg/h) is commonly used, though Clevidipine, another dihydropyridine calcium channel blocker, has gained traction due to its rapid action and ease of titration.[34]

Pronounced fluctuations of BP should be avoided, as they may exacerbate cerebral edema and ischemia. Note that hypertensive states are thought to induce an abnormal pressure natriuresis mechanism, which in the setting of acute BP elevation and hypertensive emergencies may contribute to a fluid-contracted state.[35] Initiation of medications to decrease systemic vascular resistance may lead to overshoot in BP targets as a "clamped down" but hypovolemic circulatory system is forced to suddenly relax. Administration of a small volume (500–1000mL) of isotonic fluid prior to the antihypertensives may mitigate this drop.[36]

DECISION POINT C – *What did the actual provider do?*

MDM (01:14): The patient was administered 1g of calcium gluconate, as well as 50mEq sodium bicarbonate, 10U regular insulin, and 25mg of dextrose. Nephrology was consulted and advised commencement of isotonic bicarbonate infusion, 150mEq of sodium bicarbonate in 1000mL of 0.9% NS infused at 125mL/hr.

MDM (01:49): Patient with presumed hypertensive encephalopathy. I have started antihypertensive therapy with nicardipine and paged neurology as well as the ICU team for admission. The patient was started on a nicardipine infusion at 5mg/hr. The dose was steadily increased by the bedside nurse to 15mg/hr. Repeat BPs were documented in the treatment record:

2302—224/140
2312—212/138
2324—238/148
2350—225/139
0028—228/142
0044—230/138
0110—244/140
0145—236/130
0155—232/126
0205—228/126
0224—136/74
0226—119/68

Consulting editor comments (Weingart):

Though there was an adverse outcome, I maintain that nicardipine is the agent of choice for arterial vasodilation. For other patients such aortic catastrophes where an agent is needed to block the heart rate and inotropy; use esmolol. But the vasodilator of choice for everything else is nicardipine, meaning that we only need to learn about one drug. But we need to use it right, which means we can't leave the patient at the same dose that decreased their BP initially. Nicardipine should be given as a push which is repeated, if necessary. And then when the blood pressure's at goal, we decrease the dose to 3mg/hour, and work from there.

If we leave the nicardipine at the initial dose, we are bound to fail. For example, everyone's called away for a cardiac arrest and then return 1 hour later and the patient's blood pressure is 60/40. This can't happen.

Regarding the goal BP, we need to separate out those diseases which need rapid BP lowering, such as the hypertensive patient with aortic dissection or abdominal aortic aneurysm; we'll bring those patients immediately down to 120, 110, systolic without a second thought. Versus, the neuro hypertensive emergencies where we shoot for 25 or 30% decrease in BP. With these patients—hypertensive emergency, stroke, intercranial hemorrhage, or a PRES patient—we need a balance; not too high, but not too low. For these patients a 25–30% reduction, or less than the 180mmHg is a very reasonable goal.

Author comments (Weinstock):

In the "olden days" we used to pinprick a nifedipine capsule and squirt it under the tongue to decrease the BP... until we found out that this was causing more harm than good. This case strikes to the heart of "first do no harm." We "won the BP battle," but lost the war. There are not many patients who require bedside attendance or every 5 minute checks, but management of the hypertensive emergency/posterior reversible encephalopathy syndrome (PRES) patient does. We have taken him from 224/140 to 119/68. We are at:

DECISION POINT D – YOU ARE FACED WITH 2 QUESTIONS:

1. **What immediate corrective actions can be undertaken when patients suffer iatrogenic *hypo*tension?**
2. **What is the role of push-dose pressors in immediate BP control?**

What does the literature say?

Q#1: What immediate corrective actions can be undertaken when patients suffer iatrogenic *hypo*tension?

Antihypertensive agents should be discontinued immediately upon recognition of *hypo*tension, as supratherapeutic dosing is a common complication of infusion of vasoactives with longer half-lives. Sedative infusions should also be lowered significantly or halted, as most sedating agents cause a decrease in systemic vascular resistance and are associated with vasoplegia. IV fluids should be administered to support the BP.

Section editor comments (Shiber):

Yes! Stopping sedatives is something that many forget to do in the ED and simply start or increase vasopressor infusions while the propofol drip continues!

Q#2: What is the role of push-dose pressors in immediate BP control?

Bolus administration of intravenous vasopressors and inotropics, or push-dose pressors, is a technique that has been used for decades in anesthesia environments and has gained recent traction in the emergency medicine community.[37] Phenylephrine, for example, is a pure alpha-1 agonist with a broad safety profile which can rapidly correct hypotension due to iatrogenic vasoplegia. While literature surrounding this technique continues to emerge, it appears to be a safe and effective intervention which can be lifesaving in temporizing critically low BPs.[38]

DECISION POINT D – *What did the actual provider do?*

ED course: The patient suffered a profound drop in BP as a consequence of the propofol and nicardipine infusions. The patient's nurse noted the decline and stopped all medications immediately before informing the physician, who ordered 2L of IV fluid to be administered. BP slowly recovered.

0231—128/74
0240—145/90
0252—166/92
0300—169/90

(03:04): The patient was transferred to the ICU.

What was the patient's outcome?

BP was controlled with nicardipine and the patient was eventually transitioned to enteral antihypertensive therapy. MRI later demonstrated hyperintensity and restricted diffusion of the bilateral frontoparietal region consistent with watershed infarction, as well as subcortical vasogenic edema in the bilateral parieto-occipital region consistent with PRES.

The patient was unable to be weaned from mechanical ventilation due to depressed mental status and ataxic respirations and ultimately underwent tracheostomy and PEG placement prior to transfer to a long-term acute care facility for prolonged ventilator management.

Chapter author debrief: This was a difficult case—an unexpected and rapid deterioration of a seemingly benign complaint that was further complicated and compounded by the consequences of intervention. Certainly, the treating physician did a commendable job in rapid control of the patient's presenting status epilepticus, expert control of the airway, and expedient diagnosis. The proper interventions were ordered and implemented within minutes of the patient's arrival to the ED, and a complex case full of critical care decision points was skillfully dispositioned. Nonetheless, the ED environment is defined by the high risk of serious disease and advanced intervention. While reduction of the patient's BP was the correct and necessary intervention, aggressive up-titration of antihypertensives and rapid BP lowering adds complexity and danger without increased benefit. Caution should be the rule when medications are used to manipulate patients' BP, and slow and deliberate reduction of MAP by no more than 15% can ensure efficacy without compromising safety.

Chapter summary:

- Management of asymptomatic high BP is nuanced, requiring a patient-specific approach.
- Utilization of clinical practice guidelines demands a thorough understanding and proper application (especially before citing them in the MDM).
- Posterior Reversible Encephalopathy Syndrome (PRES) is the new term for hypertensive encephalopathy where there is a neurologic consequence of end-organ damage in hypertensive emergency.
- Hypertensive emergencies are often fluid-depleted states, and initiation of parenteral antihypertensives can cause rapid and dangerous reductions in BP. Consider administration of IV fluid prior to vasoactive therapy in the patient with evidence of volume contraction and target MAP reduction between 10–20%.

References:

1. Nwankwo T, Yoon SS, Burt V, et al. Hypertension among adults in the United States: National health and nutrition examination survey, 2011–2012. NCHS Data Brief 2013;133:1–8.
2. Anchala R, Kannuri NK, Pant H, et al. Hypertension in India: A systematic review and meta-analysis of prevalence, awareness, and control of hypertension. J Hypertens 2014;32(6):1170–7.
3. Wang J, Zhang L, Wang F, et al. Prevalence, awareness, treatment, and control of hypertension in China: Results from a national survey. Am J Hypertens 2014;27(11):1355–61.
4. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure: The JNC 7 report. JAMA 2003;289(19):2560–72.
5. Rodriguez MA, Kumar SK, De Caro M. Hypertensive crisis. Cardiol Rev 2010;18(2):102–7.
6. Cremer A, Amraoui F, Lip GYH, et al. From malignant hypertension to hypertension-MOD: A modern definition for an old but still dangerous emergency. J Hum Hypertens 2016;30(8):463–6.

7. Keith NM, Wagener HP, Barker NW. Some different types of essential hypertension: Their course and prognosis. Am J Med Sci 1974;268(6):336–45.
8. Monnet X, Marik PE. What's new with hypertensive crises? Intensive Care Med 2015;41(1):127–30.
9. Leiba A, Cohen-Arazi O, Mendel L, et al. Incidence, aetiology and mortality secondary to hypertensive emergencies in a large-scale referral centre in Israel (1991-2010). J Hum Hypertens 2016;30(8):498–02.
10. González-Pacheco H, Morales-Victorino N, Núñez-Urquiza JP, et al. Patients with hypertensive crises who are admitted to a coronary care unit: Clinical characteristics and outcomes. J Clin Hypertens (Greenwich) 2013;15(3):210–4.
11. Wolf SJ, Lo B, Shih RD, et al. (2013). Clinical policy: critical issues in the evaluation and management of adult patients in the emergency department with asymptomatic elevated blood pressure. Ann Emerg Med 2013;62(1):59–68.
12. Nishijima DK, Paladino L, Sinert R. Routine testing in patients with asymptomatic elevated BP in the ED. Am J Emerg Med 2010;28(2):235–42.
13. Brody A, Rahman T, Reed B, et al. Safety and efficacy of antihypertensive prescription at emergency department discharge. Acad Emerg Med 2015;22(5):632–5.
14. Karras DJ, Kruus LK, Cienki JJ, et al. Evaluation and treatment of patients with severely elevated blood pressure in academic emergency departments: a multicenter study. Ann Emerg Med 2006; 47(3):230–36.
15. Huff SJ, Melnick E, Tomaszewski CA, et al. Clinical policy: Critical issues in the evaluation and management of adult patients presenting to the emergency department with seizures. Ann Emerg Med 2014;63(4):437–47.
16. Brophy, Gretchen M., et al. Guidelines for the evaluation and management of status epilepticus. Neurocrit Care 2012;17(1):3–23.
17. Silbergleit R, Durkalski V, Lowenstein D, et al. Intramuscular versus intravenous therapy for prehospital status epilepticus. N Engl J Med 2012;366(7):591–600.
18. Mundlamuri RC, Sinha S, Subbakrishna DK, et al. Management of generalised convulsive status epilepticus (SE): A prospective randomised controlled study of combined treatment with intravenous lorazepam with either phenytoin, sodium valproate or levetiracetam–pilot study. Epilepsy Res 2015;114:52–8..
19. O'Connor J, Levy P, Wilson SS. Levetiracetam is as effective as phenytoin for status epilepticus with less adverse events in the ED. Crit Care Med 2015;43.12(Suppl.):6.
20. Oglesby AJ. Should etomidate be the induction agent of choice for rapid sequence intubation in the emergency department? Emerg Med J 2004;21(6):655–9.
21. Reade MC, Finfer S. Sedation and delirium in the intensive care unit. N Engl J Med 2014;370(5):444–54.
22. Makii JM, Mirski MA, Lewin JJ 3rd. Sedation and analgesia in critically ill neurologic patients. J Pharm Pract 2010;23(5):455–69.
23. Carley S, Crawford I. Towards evidence based emergency medicine: Best BETs from the Manchester Royal Infirmary. Propofol for resistant status epilepticus. Emerg Med J 2002;19(2):143–4.
24. Brown LA, Levin GM. Role of propofol in refractory status epilepticus. Ann Pharmacother 1998;32(10):1053–9.
25. Qureshi AI, Palesch YY, Barsan WG, et al. Intensive blood-pressure lowering in patients

with acute cerebral hemorrhage. N Engl J Med 2016;375(11):1033–43.
26. Peacock WF, Varon J, Baumann BM, et al. CLUE: A randomized comparative effectiveness trial of IV nicardipine versus labetalol use in the emergency department. Crit Care 2011;15(3):R157.
27. Weisberg LS. Management of severe hyperkalemia. Crit Care Med 2008;36(12):3246–51.
28. Dodge HT, Grant RP, Seavey PW. The effect of induced hyperkalemia on the normal and abnormal electrocardiogram. Am Heart J 1953;45(5):725–40.
29. Chamberlain MJ. Emergency treatment of hyperkalaemia. Lancet 1964;1(7331):464–7.
30. Hinchey J, Chaves C, Appignani B, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med 1996;334(8):494–500.
31. Min Z, Gnann JW Jr. Reversible posterior leukoencephalopathy syndrome. Intern Emerg Med 2014;9(3):341–2.
32. Fugate JE, Claassen DO, Cloft HJ, et al. Posterior reversible encephalopathy syndrome: Associated clinical and radiologic findings. Mayo Clin Proc 2010;85(5):427-32.
33. Liao PY, Lee CC, Chen CY. Hypertensive brain stem encephalopathy. Am J Emerg Med 2015;33(1):131.e5–7.
34. Finger JR, Kurczewski LM, Brophy GM. Clevidipine versus nicardipine for acute blood pressure reduction in a neuroscience intensive care population. Neurocrit Care 2016;26(2):167–73.
35. Hall JE, Mizelle HL, Hildebrandt DA, et al. Abnormal pressure natriuresis. A cause or a consequence of hypertension? Hypertension 1990;15(6 Pt 1):547–59.
36. Haas AR, Marik PE. Critical care issues for the nephrologist: Current diagnosis and management of hypertensive emergency. Semin Dial 2006;19(6):502–12.
37. Holden D, Ramich J, Timm E, et al. Safety considerations and guideline-based safe use recommendations for "bolus-dose" vasopressors in the emergency department. Ann Emerg Med 2018;71(1):83–92.
38. Weingart S. Push-dose pressors for immediate blood pressure control. Clin Exp Emerg Med 2015; 2(2):131–2.