Hypertensive Crisis in the Optometry Office

A 59 year-old patient presented to your clinic for an eye examination. During pretesting, your office staff measured the patient’s blood pressure. The automated blood pressure device produced these numbers:

Systolic blood pressure of 202 mmHg
Diastolic blood pressure of 125 mmHg

Is this a scene familiar to you? How would you manage a case like this?

The Centers for Medicare & Medicaid Services (CMS) incentivized routine measurement of blood pressure through the Meaningful Use program. Over the last decade, blood pressure and the understanding of its role in pathogenesis of ocular diseases have evolved, such as the role of ocular perfusion pressure in glaucoma. As blood pressure measurement has become a routine part of the optometrists’ examination, a scenario like this has become a more common clinical encounter in the office.

Hypertensive crises

It is estimated that 32.5% of the adult population in the US is diagnosed with hypertension. Of those, approximately 1% experience severely elevated blood pressure in one’s lifetime. “Severely elevated blood pressure” is generally defined as systolic blood pressure (SBP) higher than 180 mmHg or diastolic blood pressure (DBP) higher than 110 or 120 mmHg.

Severely elevated blood pressure is associated with two crises: emergency and urgency. In the 7th report of the Joint National Committee, hypertensive emergency is defined as a “severe elevation in BP complicated by evidence of impending or progressive target organ dysfunction.” Target-organ-dysfunctions or target-organ-damages (TODs) involve the brain, heart, kidney, and large vessels: stroke, encephalopathy, acute heart failure with pulmonary edema, acute renal failure, and aortic dissection. Patients with hypertensive emergency need to be admitted immediately for antihypertensive treatment.

In contrast to the consensus over a hypersensitive emergency, “hypertensive urgency” is less defined. Patients with hypertensive urgency have severely elevated blood pressure; however, they do not have the signs and symptoms of pending TODs. Clinical dilemma often exists in the management of these individuals because a certain degree of this...
condition can pose an immediate threat while the other spectrum of patients is not at immediate risk for morbidities and mortality.

In order to ensure proper management of hypertensive crises in the optometry office, the following 5 key steps will be discussed:

1. Accuracy check
2. Symptomatology
3. Fundus examination
4. Review of history
5. Timing of referral

#1: Accuracy of blood pressure value

Blood pressure values can vary depending on the choice of the device and technique. Optometry offices are usually equipped with a manual aneroid sphygmomanometer, automated arm device, and/or automated wrist device (Figure 1). Blood pressure measurement is hydrostatic, and its outcome is highly dependent on the cuff location in respect to the right atrium of the heart. While using wrist devices, patients tend to drop the wrist down from the heart level. This would result in a higher blood pressure value than what would be found with proper technique. In order to reduce this tendency, manufacturers have applied position sensors to alert clinicians when the device is mal-positioned. Despite this improvement, automated wrist devices have been considered lacking accuracy and are not endorsed by the American Heart Association for diagnosis and management of hypertension. For this reason, optometrists should not make a critical management decisions based on the values measured with wrist devices.

Proper measurement technique is also essential to ensure accuracy. The following list shows important techniques recommended by the American College of Cardiology Foundation and the American Heart Association.5, 6.

![Blood pressure measurement devices](image_url)
One of the common sources for error is an inappropriate-sized cuff. The diaphragm of the cuff should encircle 80% of the arm circumference.

Despite careful measurement technique, the measured blood pressure could still be inaccurate compared to its true value. A number of physiological factors may influence in-office values: such as the white coat effect, diurnal fluctuation, bladder distension, caffeine intake, emotion, and noise exposure. True BP value can be investigated by using certain techniques, such as 24-hour ambulatory blood pressure monitoring and invasive intra-arterial measurement. However, those methods are unrealistic in the optometry office.

With an effort to increase the accuracy of in-office measurement, one can utilize the “time” patients spend in the office. It has been identified that blood pressure may significantly drop while the patients sit quietly in the office, and the value may mimic that of ambulatory blood pressure measurement. Grass, et al. applied this concept to 549 patients in the Emergency Department (ED) who presented with severely elevated blood pressure. Patients in this study were all asymptomatic for acute organ damage, and they were instructed to rest and wait in the exam room for 30 minutes. 32% of them showed a spontaneous drop in blood pressure well below SBP of 180mmHg and DBP of 110mHg. Those patients with reduced BP were sent home without antihypertensive treatment. No adverse events occurred.

Key:

- Proper selection of BP device
- Proper technique
- Confirm BP value over 30 minutes

**#2: Symptomatology**

Symptomatology often dictates the urgency of management. Zampaglione and his colleagues tabulated the signs and symptoms of patients with hypertensive emergency and urgency (Table 1). The listed signs and symptoms are consistent with involved end organs. For example, a patient with pulmonary edema from acute heart failure may experience chest pain and difficulty in breathing.

If a patient has any of the signs and symptoms below, optometrists should consider that individual as having a possible hypertensive emergency. The patient should be transported to ED immediately for investigation of target organ damage.

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>Emergencies (%)</th>
<th>Urgencies (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>3.0</td>
<td>22.0%</td>
</tr>
<tr>
<td>Epistaxis</td>
<td>0.0</td>
<td>17.0%</td>
</tr>
<tr>
<td>Sign/Symptom</td>
<td>Value1</td>
<td>Value2</td>
</tr>
<tr>
<td>----------------------</td>
<td>--------</td>
<td>--------</td>
</tr>
<tr>
<td>Chest pain</td>
<td>27.0</td>
<td>9.0</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>22.0</td>
<td>9.0</td>
</tr>
<tr>
<td>Faintness</td>
<td>10.0</td>
<td>10.0</td>
</tr>
<tr>
<td>Psychomotor agitation</td>
<td>0.0</td>
<td>10.0</td>
</tr>
<tr>
<td>Neurological deficit</td>
<td>21.0</td>
<td>3.0</td>
</tr>
<tr>
<td>Vertigo</td>
<td>3.0</td>
<td>7.0</td>
</tr>
<tr>
<td>Paresthesia</td>
<td>8.0</td>
<td>6.0</td>
</tr>
<tr>
<td>Vomitus</td>
<td>3.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>0.0</td>
<td>6.0</td>
</tr>
<tr>
<td>Other</td>
<td>3.0</td>
<td>2.0</td>
</tr>
</tbody>
</table>

Table 1: Signs and symptoms of hypertensive crises*


One exception can be made for a non-specific mild headache. Mild headache in severely elevated blood pressure is relatively common. In the absence of other signs and symptoms of central nervous system involvement, routine neuroimaging is unlikely to reveal CNS involvement.

Key:

- Symptomatic patients should be considered hypertensive emergency until proven otherwise

**#3: Fundus examination**

The fundus provides a tremendous amount of information about the integrity of the microvasculature. Different stages of hypertensive retinopathy represent different pathological processes.

The Keith-Wagener-Barker 4-stage classification has historically been applied to clinical practice. More recently, Wong and Mitchell simplified this classification to 3 stages.

The advantage of this simplified scheme is a linear correlation of retinopathy to target-organ-damages (TODs) that were identified from community studies.
<table>
<thead>
<tr>
<th>Classification</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td><em>Focal or general arteriolar narrowing</em></td>
<td><em>Retinal hemorrhage</em></td>
<td><em>Optic disc edema</em></td>
</tr>
<tr>
<td></td>
<td><em>Copper/silver wiring</em></td>
<td><em>Soft exudate</em></td>
<td><em>Loss of BRB integrity</em></td>
</tr>
<tr>
<td></td>
<td><em>AV nicking</em></td>
<td><em>Hard exudate</em></td>
<td><em>Loss of BRB integrity</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><em>Autoregulation breakthrough</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><em>Death</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td><em>Loss of BRB integrity</em></td>
<td></td>
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<tr>
<td></td>
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<td>4</td>
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</table>

Table 2: Clinical picture of hypertensive retinopathy

Two pathological changes progress as hypertensive retinopathy worsens: a loss of autoregulation ability and a breakdown of Blood-Retinal-Barrier (BRB) integrity.

In early stages of hypertensive retinopathy, arteriolar attenuation first becomes apparent. This is a normal autoregulation response to elevated blood pressure. The innate autoregulation ability of blood vessels allows for changes in its lumen size in an effort to maintain a steady perfusion pressure (Figure 4). When blood pressure rises, vessels become attenuated. If hypertension is controlled at this point, reversal of attenuation will occur. Chronic elevation in blood pressure eventually damages the inner lining of the vessel and causes a pathological process, called hyalinization, and eventually leads to sclerotic change. This morphological change can be clinically seen as copper and more advanced silver wiring (Figure 2). Findings from the Blue Mountain Eye Study\textsuperscript{12} and the Beaver Dam Eye Study\textsuperscript{13} suggest that reversal of attenuation is unlikely to occur once vessels undergo sclerosis even if blood pressure is controlled afterwards.
Another clinical implication of mild hypertensive retinopathy is risks of TODs. The risks are generally linked with longitudinal data rather than cross sectional analysis. For example, the Atherosclerosis Risk in Community (ARIC) Study found a positive correlation between arteriolar attenuation and concurrent left ventricular hypertrophy in the African American population\textsuperscript{14}. Left ventricular hypertrophy is an enlargement of the left ventricle muscle in order to counteract vascular resistance. This condition itself is not considered a disease, but it is a manifestation of chronic stress to the heart. Patients with left ventricular hypertrophy are at a greater risk of eventual heart diseases. Thus, the presence of mild hypertensive retinopathy in patients with severely elevated blood pressure is unlikely to imply an immediate risk of acute organ damages.

The other characteristic pathological change is seen in moderate hypertensive retinopathy patients, the presence of hemorrhages and exudates. This presence of hemorrhages and exudates is a manifestation of a compromised blood-retinal-barrier (BRB). Capillary endothelial cells and tight junctions create a tight seal around the blood vessels forming the BRB. A compromise in the BRB results in the leakage of blood constituents that is clinically evident as hemorrhages and edema. Retinal capillaries share embryological and morphological similarities with those in the brain and kidney. It is no surprise that a compromise in the BRB is suggestive of the same pathological change in the brain as well as the glomeruli of kidney. In the aforementioned ARIC Study, patients with moderate retinopathy had more than 2 fold-increased risks of glomerular filtration dysfunction\textsuperscript{18} and cognitive impairment\textsuperscript{19}. Both conditions are linked with abnormal leakage of protein that should be concealed by the organs’ barrier function. Thus, the presence of moderate retinopathy implies concurrent damage in other organ capillary systems.
Severe hypertensive retinopathy represents a breakdown in both BRB and autoregulation. This stage of retinopathy is characterized by the presence of choroidopathy, macular star, optic disc edema, and focal intraretinal peri-arteriolar transudates (FIPTs). FIPTs are leakage from dilated pre-capillary arterioles. If the retinal vasculature retains autoregulation ability, vessels will maintain a small caliber to cope with high blood pressure. However, their autoregulation ability eventually comes to an end, and vessels cannot remain attenuated and will start dilating. This end process is called “autoregulation breakthrough”, and organ tissues suffer from hyper-perfusion under this condition.

Optic disc edema suggests an even more alarming issue. There are conflicting opinions in its underlying mechanism: encephalopathy, intracranial hypertension, and ischemia in short posterior ciliary arteries. A study showed that patients with severe hypertension suffer from impaired autoregulation in the brain and thus, this stage of retinopathy likely represents an impaired autoregulation in both the retina and the brain. Considering the high mortality rate, patients with severe retinopathy should be considered as a hypertensive emergency and admitted to the ED immediately.

Key:

- Progressively increased risk of TODs with worsening hypertensive retinopathy.

#4: Review of history
If the patient is asymptomatic and the fundus shows no retinopathy or only mild retinopathy, is he/she a non-urgent patient? To address this question, a careful review of their history is critical.

Experts suggest that true hypertensive urgency should be distinguished from “severe uncontrolled hypertension” among asymptomatic patients based on the presence of risk factors for progressive organ damages\(^3,10\). These risk factors include, but are not limited to, the established history of cardiovascular, cerebral and renal diseases such as congestive heart failure, unstable angina, coronary artery disease, renal insufficiency, transient ischemic attack, or stroke\(^10\). Patients with such histories should be given greater attention and should be considered “hypertensive urgency” even if they are asymptomatic and have no clear signs of damages in the BRB or autoregulation.

Attention should also be paid to how rapidly the blood pressure rose to this severe level. Autoregulation has an amazing adaptability of adjusting itself to chronically elevated blood pressure to provide a steady perfusion pressure to protect organs (Figure 4). Its effect is prominent in certain organs and vessels, such as the brain, coronary artery, kidney, aorta, and retina. However, this shift takes months and years to occur. Therefore, patients who experience sudden pressure rise are more likely to fall outside the protective range of autoregulation. High-risk profiles include younger individuals, pregnancy, use of recreational drugs, and no prior diagnosis of hypertension.

![Figure 4: Autoregulation](image_url)

Overall, the risk assessment should be made based on a global analysis of age, chronicity of hypertension, rapidity of blood pressure increase, and preexisting TODs rather than the actual severity of hypertension\(^10\).

**#5: Timing of referral**

How urgently should patients with severely elevated pressure be managed?
The appropriate timeframe for a suspected case of hypertensive emergency is clear and simple: immediate referral to the ED. The case warrants immediate workup for acute TODs. Upon confirmation, the patient will be admitted to the intensive care unit and receive intravenous antihypertensive treatment to lower BP by 20~25% depending on the underlying condition.

Patients with hypertensive urgency are not manifesting signs and symptoms of acute organ damages. However, these patients possess certain risks for progressive organ damage. The risks may be apparent in their fundus or in their medical histories. At this time, it is still unclear when these patients will convert to hypertensive emergency. Therefore, a timely referral should be made without delay within 24~48 hours \(^3,10\). Depending on the circumstance, referral to the ED may be appropriate in these cases.

What about asymptomatic patients with zero to minimal risks? Should they still be sent to the ED?

In their recent clinical policy, the American College of Emergency Physicians reviewed the evidence and expert opinions on how to manage asymptomatic patients with markedly elevated blood pressure in the emergency department \(^{24}\). This review did not find sufficient evidence to support routine work-ups (ex: serum creatinine, urinalysis, ECG) and the practice of lowering blood pressure at the ED. It is noteworthy that experts did recommend that care should be given to patients with poor follow-up, limited health care access, older age, or an African American racial background. This recommendation is not based on a patient’s blood pressure value, rather on a patient’s profile.

You may wonder why a more aggressive treatment approach was not recommended in this review. The key is in autoregulation. When the patient has no overt signs, including in the fundus, and symptoms of organ damage from severe hypertension, it is very likely that the patient’s autoregulation has shifted to a rightward direction maintaining a steady perfusion pressure to vital organs even at a higher mean arterial pressure (Figure 4). This “right” shift ironically makes patients more vulnerable for a risk of hypo-perfusion by an abrupt drop in blood pressure in an attempt to normalize it. These patients may suffer ischemia in vital organs, such as myocardial ischemia and infarction, stroke, and death from treatment itself \(^{25}\). To minimize this risk while achieving a substantial anti-hypertensive effect, long-acting medications should be administered by a provider who can monitor their progress regularly. This allows a gradual shift in autoregulation to a leftward direction. Thus, patients with low risks are more likely to achieve long-term success in the hands of their own primary care physicians.

**Summary**

Management of patients with severely elevated blood pressure brings a number of dilemmas to optometrists. Inappropriate management of this condition could result in significant disability and mortality. While some patients need immediate care at the ED, others do not necessarily benefit from ED referral. Studies have noted a disappointing follow-up rate at the primary care physician’s office after an ED visit \(^{26,27}\). Meanwhile, budgetary constraints have recently made the CMS focus on reducing the cost for non-emergent ED visits. Optometrists need a strategy.

Optometrists are unique health care providers who are capable of making accurate assessments on the integrity of the microvasculature. By carefully examining a patient’s symptoms, fundus, and medical histories (Table 3), optometrists can triage this challenging condition and guide the patients to long-term success in their lives.

<p>| Severe uncontrolled HTN | Hypertensive urgency | Hypertensive emergency |</p>
<table>
<thead>
<tr>
<th>Symptomatology</th>
<th>Asymptomatic or non-specific mild headache</th>
<th>Asymptomatic or non-specific mild headache</th>
<th>Symptomatic of acute TODs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fundus</td>
<td>No or mild retinopathy</td>
<td>Moderate retinopathy</td>
<td>Severe retinopathy</td>
</tr>
<tr>
<td>Medical History</td>
<td>No pertinent history</td>
<td>History of preexisting organ injury</td>
<td>May or may not have pertinent history</td>
</tr>
<tr>
<td>Timing of referral</td>
<td>Within 1 week to PCP</td>
<td>Within 24~48 hours to PCP or ED</td>
<td>Immediate referral to ED</td>
</tr>
</tbody>
</table>

Table 3: Management strategy for patients with severely elevated blood pressure

Acknowledgment: I would like to thank Sean Umamoto, Ph.D. for his insightful comments on this manuscript.

For comments and questions, please contact Dr. Kanai at kunikanai@berkeley.edu

Reference

1. Which of the following devices is the least ideal to determine blood pressure in patients with suspected hypertensive crises?
   a. Manual aneroid sphygmomanometer
   b. Automated arm device
   c. Automated wrist device
   d. All above is equally ideal

2. Which of the following techniques likely results in a faulty blood pressure value?
   a. Placement of a cuff at the heart level
   b. Deflation of a cuff at the rate of 2mmHg per second
   c. Having a patient seated in a chair with no back support
   d. The diaphragm of a cuff encircles 80% of the arm circumference

3. Which of the organs/vessels can hypertensive emergency involve?
   a. Brain
   b. Kidney
   c. Heart
   d. Aorta
   e. All above

4. True or False: a patient with systolic blood pressure of 200mmHg and diastolic blood pressure of 120mmHg is at the immediate risk of acute organ failure regardless of their symptoms, fundus appearance, and medical history.

5. Which of the following symptoms is suggestive of progressive target-organ-damages?
   a. Shortness of breath
   b. Severe headache
   c. Chest pain
   d. Speech slur
   e. All above

6. True or False: the retinal vasculature exhibits autoregulation response to a variety of blood pressure.

7. True or False: mild hypertensive retinopathy exemplifies “autoregulation breakthrough”.

8. True or False: moderate hypertensive retinopathy is suggestive of a compromise in the glomerular filtration barrier of the kidney.

9. True or False: when blood pressure is severely elevated, a patient with known history of unstable angina should be given extra caution in management.

10. Which of the following statement is true in regards to autoregulation?
    a. In chronic hypertension, autoregulation gradually shifts to a rightward direction.
    b. If blood pressure rises acutely, the organ may undergo hyper-perfusion.
    c. If blood pressure is abruptly reduced, the organ may undergo hypo-perfusion.
    d. Slow anti-hypertensive treatment allows autoregulation to shift gradually to a leftward direction.
    e. All above
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