Dear Colleagues:

The EMCREG-International Hypertension Consensus Panel gathered experts from diverse fields, including Emergency Medicine, Cardiology, Neurology, Neurosurgery, Neuro-Critical Care, Obstetrics/Gynecology, Nephrology, Pediatrics, and Hospital Medicine to discuss, analyze, and provide recommendations for treating hypertension. Through this collaboration, virtually any patient presenting with hypertension to an outpatient or emergency department setting will have clear guidelines for treatment and physician follow-up.

Through this EMCREG-International Newsletter, Drs. Judd Hollander and Anna Marie Chang discuss the pathophysiology of hypertension, as well as describe historical and physical examination findings crucial for the evaluation of end organ damage. Descriptions of hypertension associated with the following disease processes: Asymptomatic Hypertension, Acute Coronary Syndrome, Acute Heart Failure Syndromes, Neurological Emergencies, Cocaine or Amphetamine Induced Hypertension, and Aortic Dissection are provided with treatment recommendations. Hopefully the reader of this EMCREG-International Newsletter will also read the recently published EMCREG-International Hypertension Consensus Panel manuscript.

Through collaboration with colleagues from a variety of specialties, patients with hypertension can receive optimal therapy when presenting to any acute care setting. It is our hope you will find this EMCREG-International Newsletter, and the full manuscript of the Hypertension Consensus Panel which forms the basis for this Newsletter, helpful in the care of your patients with hypertension.

Sincerely,

Anna Marie Chang, MD
Department of Emergency Medicine, University of Pennsylvania, Philadelphia, Pennsylvania

Judd E. Hollander, MD
Professor and Clinical Research Director, Department of Emergency Medicine, University of Pennsylvania, Philadelphia, Pennsylvania

Objectives:
1. To discuss the scope of hypertension in the emergency department.
2. To discuss the various emergent presentations of hypertension.
3. Describe recommended treatments for these emergent hypertension related conditions.

Introduction

Hypertension is the most common outpatient diagnosis in the United States, accounting for over 37 million outpatient visits in 2004.¹ Almost 30% of ambulatory visits include a diagnosis of hypertension.² It is more common in older age groups, in males, and in African Americans.²

Hypertension is an independent risk factor for myocardial infarction (MI), heart failure, stroke, and renal disease. Treatment of elevated blood pressure reduces the likelihood of developing these sequelae (Table 1).³ This recognition has led to a recent increase in hypertension control rates, however, the prevalence has stayed the same.

Peer Reviewer for Commercial Bias: Deborah B. Diercks, MD - Associate Professor of Emergency Medicine, Department of Emergency Medicine, University of California, Davis, Sacramento, CA
The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7) divides hypertension into three groupings: pre-hypertension, stage 1 and stage 2 hypertension, and uses these categories to guide therapy (Table 2). These guidelines recommend repeating blood pressure measurements on two separate occasions prior to the diagnosis of hypertension, which is not practical for diagnosis and risk stratification in the emergency department (ED). Over 30% of patients presenting to the ED have an elevated blood pressure (>140/90 mmHg). Studies have shown that 20% to 70% of patients with an increased blood pressure in the ED subsequently have an elevated blood pressure in the outpatient setting. In order to supplement these guidelines, EMCREG-International convened an expert panel to review the literature and develop consensus recommendations for the treatment of hypertensive emergencies in the ED (Table 3). The full proceedings of this symposium have been published in a supplement to the Annals of Emergency Medicine. This monograph highlights some of the top level recommendations by the panel.

### Table 1. End Organ Damage Associated with Hypertensive Emergencies

<table>
<thead>
<tr>
<th>End Organ Damage Type</th>
<th>Cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral Infarction</td>
<td>24.5</td>
</tr>
<tr>
<td>Hypertensive Encephalopathy</td>
<td>16.3</td>
</tr>
<tr>
<td>Acute Heart Failure Syndrome</td>
<td>14.3</td>
</tr>
<tr>
<td>Acute Coronary Syndrome</td>
<td>12.0</td>
</tr>
<tr>
<td>Intracerebral or Subarachnoid Hemorrhage</td>
<td>4.5</td>
</tr>
<tr>
<td>Aortic Dissection</td>
<td>2.0</td>
</tr>
</tbody>
</table>


### Table 2. JNC-7 Classification of Hypertension

<table>
<thead>
<tr>
<th>Class</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
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<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Pre-hypertension</td>
<td>120-139</td>
<td>80-89</td>
</tr>
<tr>
<td>Stage 1</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
<td>Stage 2</td>
<td>160 or higher</td>
<td>100 or higher</td>
</tr>
</tbody>
</table>

Pathophysiology

Blood pressure is regulated by the cardiovascular, renal, endocrine, and central nervous systems. In turn, blood pressure also affects these systems. Blood pressure is the primary determinant of tensile stress in arteries. Chronic elevation of blood pressure leads to vascular remodeling with resultant thickening and sclerosis of vessel walls. The sclerotic changes enhance the shearing force in the vessel, further contributing to vessel damage. In addition, the endothelium secretes nitric oxide, prostacyclin, and endothelin, which modulate vascular tone. Prolonged vasoconstriction leads to endothelial dysfunction, loss of nitric oxide production, and irreversible rise in peripheral arterial resistance. Inflammatory cytokine release causes an increase in endothelial permeability, decreases fibrinolysis, and increases coagulation.11

The renin-angiotensin-aldosterone system contributes to blood pressure regulation. Renin is released from the kidneys in response to underperfusion of the kidney and low sodium intake. This causes activation of angiotensin II, a potent vasoconstrictor, and subsequently increases blood pressure. Angiotensin II also causes aldosterone release, which further increases blood pressure.11,12 Persistent stimulation of the renin-angiotensin-aldosterone axis may result in a rise in creatinine, hypokalemic metabolic alkalosis, hematuria and proteinuria. With renal end organ damage due to hypertension, the kidneys will demonstrate glomerular ischemia, proliferative endarteritis with capillary and arteriolar necrosis.13

Table 3. EMCREG-International Hypertension Consensus Panel

<table>
<thead>
<tr>
<th>Panel Member</th>
<th>Institution</th>
<th>Specialty</th>
<th>Topic</th>
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</thead>
<tbody>
<tr>
<td>James W. Hoekstra, MD</td>
<td>Wake Forest</td>
<td>EM</td>
<td>Co-Chair</td>
</tr>
<tr>
<td>Adnan I. Qureshi, MD</td>
<td>U Minnesota</td>
<td>Neurology</td>
<td>Co-Chair</td>
</tr>
<tr>
<td>Alpesh N. Amin, MD</td>
<td>U California-Irvine</td>
<td>Hospital Medicine</td>
<td>Parenteral Medications</td>
</tr>
<tr>
<td>John R. Barton, MD</td>
<td>U Kentucky</td>
<td>Ob/Gyn</td>
<td>Pre/Eclampsia</td>
</tr>
<tr>
<td>Albert T. Cheung, MD</td>
<td>U Pennsylvania</td>
<td>Anesthesiology</td>
<td>Anesthesia</td>
</tr>
<tr>
<td>David M. Cline, MD</td>
<td>Wake Forest</td>
<td>EM</td>
<td>Epidemiology</td>
</tr>
<tr>
<td>Deborah B. Diercks, MD</td>
<td>UC Davis</td>
<td>EM</td>
<td>Heart Failure/ACS</td>
</tr>
<tr>
<td>Katherine L. Heilpern, MD</td>
<td>Emory</td>
<td>EM</td>
<td>Pathophysiology</td>
</tr>
<tr>
<td>Robert W. Hobson, MD</td>
<td>U New Jersey</td>
<td>Surgery</td>
<td>Vascular Surgery</td>
</tr>
<tr>
<td>Judd E. Hollander, MD</td>
<td>U Pennsylvania</td>
<td>EM</td>
<td>Cocaine/Other Drugs</td>
</tr>
<tr>
<td>Raj K. Narayan, MD</td>
<td>U Cincinnati</td>
<td>Neurosurgery</td>
<td>Head Trauma</td>
</tr>
<tr>
<td>E. Magnus Ohman, MD</td>
<td>Duke U</td>
<td>Cardiology</td>
<td>Heart Failure/ACS</td>
</tr>
<tr>
<td>Arthur M. Pancioli, MD</td>
<td>U Cincinnati</td>
<td>EM</td>
<td>Neuro Emergencies</td>
</tr>
<tr>
<td>Alluru S. Reddi, MD</td>
<td>UMDNJ</td>
<td>Nephrology</td>
<td>Essential HTN</td>
</tr>
<tr>
<td>Lori A. Shutter, MD</td>
<td>U Cincinnati</td>
<td>Neuro/Crit</td>
<td>Head Trauma</td>
</tr>
<tr>
<td>Corey M. Slovis, MD</td>
<td>Vanderbilt U</td>
<td>EM</td>
<td>Essential HTN</td>
</tr>
</tbody>
</table>

Patients with chronic hypertension may experience decreased cerebral blood flow and subsequent cerebral ischemia if blood pressure is lowered rapidly.
An imbalance in sympathetic tone and parasympathetic response can result in increased cardiac output and arterial vasoconstriction. Patients with chronic hypertension may experience decreased cerebral blood flow and subsequent cerebral ischemia if blood pressure is lowered rapidly. Conversely, a rapid rise in blood pressure can cause the classic triad of hypertensive encephalopathy: hypertension, altered mental status and papilledema. Chronic hypertension also affects the heart by increasing left ventricular mass, which may result in decreased coronary perfusion pressure, increased myocardial oxygen consumption, and subsequent myocardial ischemia.

History and Physical Examination

The history and physical examination should be focused on evaluation of possible end organ damage. The history should include medication use as well as any recreational drug use which may be a secondary cause of hypertension. Complaints of chest pain should lead to evaluation for possible acute coronary syndrome (ACS) and aortic dissection. Sudden onset of headache may suggest subarachnoid hemorrhage. Shortness of breath should lead to evaluation of heart failure. The physical examination should include auscultation of the lungs for evidence of pulmonary edema, and the heart for murmurs or gallops. The neurological examination should focus on lateralizing signs which may suggest a stroke. In the setting of severe hypertension, there is the potential for disruption of the blood-retina barrier, necrosis, retinal ischemia and optic disk edema such as hemorrhage, ‘cotton wool spots’ and papilledema on funduscopic examination. Retinopathy suggests concurrent vascular disease in the cerebral arterioles, thereby increasing the risk of all vascular complications.

Asymptomatic Hypertension

Most patients with hypertension in the ED will be asymptomatic. However, hypertension is a risk factor for many urgent or emergent conditions including subarachnoid hemorrhage, ischemic and hemorrhagic stroke, aortic dissection, heart failure, and ACS. Unless there are signs of acute end organ damage, or the patient is known to already carry the diagnosis of hypertension, ACEP Practice Guidelines do not recommend the routine initiation of anti-hypertensive medications in the ED. Asymptomatic patients with pre-hypertension or stage 1 hypertension and a normal physical examination should be referred for a blood pressure re-check. ACEP guidelines also point out that other association guidelines similarly do not recommend using intravenous agents to acutely lower blood pressure, and the goal should not be to normalize blood pressure during the ED visit.

The EMCREG-International consensus conference recommends a stepped approach to referral time period and treatment initiation (Table 4). The recommended referral time for repeat evaluation is 1-2 months unless the systolic blood pressure (SBP) is ≥180 mmHg or the diastolic pressure (DBP) is ≥110 mmHg. For blood pressure above these levels, it is recommended the patient be advised to arrange follow-up within one week. Above a SBP of 210 mmHg or DBP of 120 mmHg, it is reasonable to initiate oral anti-hypertensive medications and recommend earlier follow-up (Table 5). If a patient needs to be started on an anti-hypertensive, the physician should obtain electrolytes and a creatinine prior to starting medication and refer the patient for timely outpatient follow-up.

### Table 4. EMCREG-International Consensus Panel Recommendations for Treatment of Asymptomatic Hypertension without End Organ Damage

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Recommended Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>140-159/90-99</td>
<td>Observe and confirm within 2 months</td>
</tr>
<tr>
<td>160-179/100-109</td>
<td>Confirm and treat within 1 month</td>
</tr>
<tr>
<td>180-209/110-119</td>
<td>Confirm and treat within 1 week</td>
</tr>
<tr>
<td>210+/120+</td>
<td>Confirm, evaluate, initiate oral outpatient treatment, and refer for follow-up</td>
</tr>
</tbody>
</table>
The EMCREG-International Hypertension Consensus Panel: Management of Hypertensive Emergencies

Table 5. Therapeutic Options for Individual Drugs Based on the Underlying Condition of the Patient (JNC 7 Report)

<table>
<thead>
<tr>
<th>Concurrent conditions</th>
<th>Initial therapy options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Failure</td>
<td>THIAZ, BB, ACEI, ARB, ALDO ANT</td>
</tr>
<tr>
<td>Post-myocardial infarction</td>
<td>BB, ACEI, ALDO ANT</td>
</tr>
<tr>
<td>High CVD risk</td>
<td>THIAZ, BB, ACEI, CCD</td>
</tr>
<tr>
<td>Diabetes</td>
<td>THIAZ, BB, ACEI, ARB, CCD</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>ACEI, ARB</td>
</tr>
</tbody>
</table>

Abbreviations: ACEI, angiotensin converting enzyme inhibitor; ALDO ANT, aldosterone antagonist; ARB, angiotensin receptor blocker; BB, β-blocker; CCB, calcium channel blocker; THIAZ, thiazide. Adapted with permission from Chobanian AV, et al. JAMA 2003;289(19):2560-2572.

Acute Coronary Syndrome

The objective in the treatment of hypertension in patients with ACS is reduction of ischemic symptoms. The treatment of hypertension in the setting of ACS may require multiple agents. In the ACC/AHA guidelines for ST-segment elevation MI, both nitroglycerin and beta-blockers are a Class I recommendation. In patients with ongoing symptoms, calcium channel blockers are a Class IIa recommendation. In the ACC/AHA guidelines for unstable angina/non-ST-segment elevation MI, several medications receive a Class I recommendation for treatment. Nitroglycerin is the first line agent which lowers blood pressure and reduces ischemic symptoms. For continuing symptoms, the guidelines recommend a beta-blocker, followed by a nondihydropyridine calcium antagonist, if necessary. An ACE inhibitor is recommended in patients with ongoing symptoms despite initial treatment if the patient has known left ventricular dysfunction, heart failure, or diabetes.

It should be noted that in patients with an ST-segment elevation MI, the use of fibrinolytics is contraindicated if the blood pressure is greater than 185/100 mmHg.

Acute Heart Failure Syndrome

Patients with acute heart failure syndrome (AHFS) often present with elevated blood pressure. Patients with acute symptoms should be stratified according to their initial blood pressure and treatment should be based upon this stratification. Treatment for patients with elevated systolic blood pressure (>140 mmHg) has focused on the use of vasodilators. The European Society of Cardiology (ESC) recommends nitroglycerin or other vasodilators as the first line treatment for AHFS (Class I, Level B). Nitroprusside or intravenous angiotensin converting enzyme inhibitors are also options. Blood pressures should not be normalized, but instead, lowered by 30 mmHg. If this cannot be achieved through diuretics and vasodilators, the ESC recommends a calcium channel blocker. Research is currently underway to define the role of agents such as vasopressin antagonists, adenosine antagonists, and endothelin antagonists, as potential treatment options for this group of patients.

Neurological Emergencies

In the setting of acute ischemic stroke, aggressive lowering of blood pressure can reduce perfusion which expands the zone of infarction. This is well documented with sublingual nifedipine, which should never be used to rapidly reduce blood pressure. The only clear indications for blood pressure reduction in stroke are for patients with hemorrhagic stroke and patients being treated with fibrinolysis for ischemic stroke. In these populations, elevated blood pressure significantly increases the risk of intracerebral hemorrhage. Fibrinolytic therapy should not be given to patients who have a SBP >185 mmHg or a DBP >110 mmHg at the time of treatment. When treated with a fibrinolytic, the blood pressure must be maintained <180/105 mmHg for 24 hours.
In intracerebral hemorrhage (ICH), blood pressure should be controlled to maintain cerebral perfusion pressure. It is reasonable to maintain mean arterial pressure (MAP) below 130 mmHg or systolic blood pressure below 180 mmHg for the first 24 hours following symptom onset. In patients with elevation in intracranial pressure, mean arterial pressure should be maintained below 110 mmHg, or SBP below 160 mmHg, for the first 24 hours following symptom onset. It is recommended in patients who have undergone a craniotomy, the MAP should be maintained under 100 mmHg. Hopefully these patients will not still be in the ED. In all cases, MAP should be maintained above 90 mmHg, and the cerebral perfusion pressure should be maintained above 70 mmHg. When the clinician determines blood pressure lowering is required for ICH, the preferred agents are esmolol, labetolol, and nicardipine. Nitroprusside and hydralazine should not be used in the setting of neurologic emergencies. These agents can cause adverse consequences on intracranial pressure in patients with elevated intracranial pressure.

Patients with an ischemic stroke who have concurrent evidence of end organ damage such as acute myocardial infarction, aortic dissection, hypertensive encephalopathy, acute renal failure, and acute pulmonary edema, or patients with extremes of blood pressure the clinician feels compelled to treat, would be best treated with labetolol or nicardipine.

Blood pressure control is vital in subarachnoid hemorrhage (SAH). There is a linear relationship between early rebleeding and increasing SBP above 160 mmHg. Currently, most physicians caring for aneurysmal SAH treat elevated blood pressure when the patients MAP is above 130 and try to maintain the SBP below 160 mmHg. Oral nimodipine is used in patients with aneurysmal SAH to prevent delayed ischemic neurological deficits. Nimodipine may have a hypotensive effect but is not the preferred agent for treatment of hypertension. Prior to treatment with any antihypertensive agent, pain control and sedation should be addressed and blood pressure should then be reassessed. When blood pressure lowering is required for SAH, the preferred agents are esmolol, labetolol, and nicardipine.

**Cocaine or Amphetamine Induced Hypertension**

Sympathomimetic agents can induce hypertension. The ED approach to the patient with cocaine intoxication is analogous to that of the patient with hypertension with some important differences. Asymptomatic patients do not require treatment because the half-life of the sympathomimetic agents is short and the stimulus for hypertension will resolve in several hours. When patients are agitated or require treatment for potential end organ damage, benzodiazepines are recommended as they will usually reduce the blood pressure and heart rate. When sedation is unsuccessful and the patient has hypertension that warrants treatment, management with sublingual or intravenous nitroglycerin or intravenous phentolamine is indicated. Beta blockade is contraindicated because it results in an unopposed alpha-adrenergic effect, and can lead to vasoconstriction and a paradoxical increase in blood pressure.

**Aortic Dissection**

Chronic hypertension affects arterial wall composition causing thickening, calcification, fibrosis and fatty acid deposition. The vessel wall becomes vulnerable to pulsatile forces that can cause an intimal tear creating a false lumen. Because acute aortic dissection involving the ascending thoracic aorta or aortic arch (Stanford type A) is also a surgical emergency, the treatment of hypertension in these patients must take into account surgical considerations. A recent review recommended blood pressure control...
with a target SBP of 110 mmHg achieved using a combination
of narcotic analgesics, intravenous beta-blockers, and vasodilating
drugs such as sodium nitroprusside.\textsuperscript{40} This antihypertensive regimen
is consistent with the recommendations of the ESC Task Force on
Aortic Dissection which recommended pain relief such as morphine
sulfate, reduction of systolic blood pressure using beta-blockers
including intravenous esmolol, labetolol, metoprolol, or propranolol,
and the addition of vasodilators such as sodium nitroprusside
to achieve a SBP in the range of 100 mmHg to 120 mmHg.\textsuperscript{41}
Verapamil or diltiazem are recommended as alternatives in patients
with potential intolerance to beta blockers.\textsuperscript{40,41} The EMCREG-
International Hypertension Consensus Panel recommendations for
these disease processes are provided in Table 6.

**Conclusion**

Patients will often present to the emergency department with
hypertension. In asymptomatic patients, it is usually not
necessary to treat but to recommend follow-up for the patient.
Hypertension is a risk factor for many of the urgent conditions
seen in the ED, including SAH, ischemic and hemorrhagic
stroke, aortic dissection, heart failure, and ACS, and these
patients may need aggressive management of their blood
pressure. The appropriate therapeutic approach depends
on the clinical presentation and condition of the patient.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Therapeutic Goal</th>
<th>Suggested Medication</th>
<th>Other information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Coronary Syndrome</td>
<td>Reduction of blood pressure and reduction of myocardial oxygen demand</td>
<td>Nitroglycerin, IV β-blocker</td>
<td>β-blockade also reduces mortality associated with ventricular dysrhythmias</td>
</tr>
<tr>
<td>Acute Heart Failure Syndromes</td>
<td>Diuresis, reduction of blood pressure by vasodilation</td>
<td>IV nitroglycerin, morphine, diuretic, possible IV ACE-I</td>
<td>Diuretics and ACE-I can worsen renal function</td>
</tr>
<tr>
<td>Ischemic Stroke, Subarachnoid Hemorrhage</td>
<td>Avoid hypoperfusion, do not exceed 20% reduction in blood pressure</td>
<td>IV esmolol, labetolol, and nicardipine</td>
<td>BP control is essential if considering tPA, otherwise, there is no clear benefit of intensive blood pressure control in the setting of stroke</td>
</tr>
<tr>
<td>Acute Cocaine Intoxication</td>
<td>Reduction of sympathomimetic drive</td>
<td>Benzodiazepines, IV nitroglycerin, phentolamine</td>
<td>Measure core temperature and treat hyperthermia; consider multi-drug intoxication</td>
</tr>
<tr>
<td>Aortic Dissection</td>
<td>Reduction of shear forces and tachycardia, maintain SBP 120-140 and HR 60</td>
<td>IV labetolol, β-blocker, nitroprusside</td>
<td>Nitroprusside is extremely potent and can become toxic; avoid volume depletion for possible OR</td>
</tr>
</tbody>
</table>
References


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CME Post Test

After you have read the monograph carefully, record your answers by circling the appropriate letter answer for each question.

1. At which of the following diastolic blood pressures should you administer parenteral antihypertensive medications in an asymptomatic patient?
   a. 70  d. 110
   b. 90  e. None of the above
   c. 100

2. The most common end organ affected by hypertensive emergencies is
   a. Brain  c. Kidney
   b. Heart  d. Liver

3. The initial anti-hypertensive medication for treatment of aortic dissection is preferentially
   a. A beta antagonist  b. A thiazide diuretic
   c. An angiotensin converting enzyme inhibitor  d. Nitroglycerin

4. Which of the following is contraindicated in a patient intoxicated with cocaine and having chest pain with hypertension?
   a. A beta antagonist  b. A thiazide diuretic
   c. An angiotensin converting enzyme inhibitor  d. Nitroglycerin
   e. A benzodiazepine

5. For a patients with a blood pressure of 170/105, ACEP Practice Guidelines recommend initiation of antihypertensive medications in the ED only when there are signs of acute end organ damage, or the patient is known to already carry the diagnosis of hypertension.
   a. True  b. False

6. According to the EMCREG-International consensus recommendations, which of the following are true:
   a. If the blood pressure is < 180/110 mmHg, the patient should follow up within 1-2 months.
   b. For a blood pressure > 180/110 mmHg, the patient should follow up within 1 week.
   c. For a blood pressure > 210/120 mmHg the patient should have early follow up and it is reasonable to begin oral therapy.
   d. All of the above

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What topics would be of interest to you for future CME programs?

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Management of Hypertensive Emergencies
February 2008, Volume 2