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Stroke is the third leading cause of death in the United States, surpassed only by heart diseases and malignant neoplasms.[1] In 2000, stroke accounted for 167,661 deaths, yielding a population death rate of 60.9 per 100,000. Approximately 750,000 new strokes and more than 1 million hospitalizations for stroke occur annually in the United States. Eight percent of stroke victims die within 30 days of the event, and about 30% are dead within a year. Thirty-one percent of stroke survivors require assistance with activities of daily living and 16% require assisted-living institutions. The annual estimated economic impact of stroke on our society, i.e., the costs associated with health care and lost income, is $40.9 billion.[2]

Overall, the incidence of stroke is higher among males than among females; however, in younger age groups, the gender incidence is about the same.[3,4] For people older than age 55, the incidence of stroke more than doubles in each successive decade. Nearly 30% of those who suffer a stroke are younger than age 65.[5] Among the elderly, the number of female stroke victims is higher than the number of male stroke victims, in part because of the greater longevity of females. African-Americans and Hispanics have an increased annual age-adjusted relative risk of ischemic stroke compared with whites.[1,6] Geographically, the southeastern United States has the highest incidence of stroke compared with other regions of the country.[7]

Part 1 of this series will cover the differential diagnosis of stroke, stroke mimics, and risk factors and prevention. Part II will cover the physical examination, laboratory investigations, imaging, and treatment of stroke.

—The Editor

Ischemic Stroke Syndromes: The Challenges of Assessment, Prevention, and Treatment

Part I: Risk Factors, Differential Diagnosis, and Prevention

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Definitions

In 1980, the World Health Organization published what has become the generally accepted definition of stroke: "rapidly developing clinical signs of focal (at times global) disturbance of

cerebral function, lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin."[8] Transient ischemic attacks (TIAs) are neurologic deficits that occur as a result of loss of or decrease in perfusion of the brain, retina, or cochlea. These deficits resolve within 24 hours, most of them within one hour.[9] Focal neurologic deficit lasting more than 24 hours but fewer than three weeks is classified as reversible ischemic neurologic deficit.[10]

Pathophysiology

Ischemic stroke accounts for 80-85% of all cerebrovascular accidents. It can result from thrombosis, embolism, or hypoperfusion (thrombosis is the most common etiology). It occurs as a result of neurons being deprived of oxygen and glucose. The brain does not store these substrates and, thus, requires a constant supply. When the blood supply to the cerebral microvasculature is disrupted, brain energy metabolism is disrupted, leading to loss of aerobic glycolysis, intracellular accumulation of sodium and calcium ions, release of excitotoxic neurotransmitters, elevation of lactate levels with local acidosis, free-radical production, cell swelling, overactivation of lipases and proteases, and cell death. Within 12-15 seconds of complete interruption of cerebral blood flow, electrical activity is suppressed, and within 2-4 minutes, synaptic excitability of cortical neurons is inhibited. After 4-6 minutes, electrical excitability is inhibited. When the cerebral blood flow decreases to about one-third of normal (18 mL/100 gm/min), the brain reaches a threshold for electrical failure but there still is a chance for recovery. This may occur in the area surrounding the core of the infarction, where tissue may be receiving flow from nearby collaterals. This area is known as the ischemic penumbra and is the target for stroke treatments. It is estimated that approximately six hours represents the window of opportunity available before the penumbra is lost and irreversible neurologic damage occurs. When cerebral perfusion further decreases to 8 mL/100 gm/min, as occurs in the core of the infarcted area, the result can be cell death. Between these two thresholds, there may be neurons that are functionally silent but structurally intact and potentially salvageable.[2,11]

Stroke Mimics

Multiple clinical entities of various origins can resemble or be indistinguishable from an ischemic stroke or TIA. In the acute state, considerations of differential diagnosis apply equally to TIA and stroke. It is extremely important to make an accurate diagnosis rapidly, since the treatment of an ischemic stroke is most optimal when initiated within three hours of onset of symptoms. This treatment may be deleterious under other conditions. Several mimics of ischemic stroke and TIA are discussed below. (*See Table 1.*)

Hemorrhage. Intracranial hemorrhage, a mimic of cerebral ischemia, can occur by hypertensive and nonhypertensive mechanisms. Hypertension-related intracranial hemorrhage is caused by rupture of 50- to 300-mm diameter parenchymal perforating arteries damaged by local degenerative effects of hypertension. Focal deficits develop steadily over seconds to minutes. The possibility of intracranial hemorrhage is increased with the presence of coma on arrival, vomiting, severe headache, current warfarin therapy, systolic blood pressure greater than 200 mmHg, or glucose level greater than 170 mg/dL in a nondiabetic patient. The absence of these symptoms decreases the odds of hemorrhage by about one-third.[12]

The putamen is the area of the brain most commonly affected, followed by the cerebral lobes, thalamus, cerebellum, pons, and other parts of the brain. Putaminal hemorrhage is characterized by hemiparesis or hemiplegia (paralysis of half the body) involving the arm, face, or leg, accompanied by a hemisensory syndrome, hemianopsia (loss of vision on the same half of each

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Table 1. Differential Diagnosis of Ischemic Stroke

- Hemorrhage
- Todd's paralysis
- Hypoglycemia
- Migraine headache
- Bell's palsy
- Hyponatremia
- Hyperglycemia
- Meningitis/encephalitis/ abscess
- Hypertensive encephalopathy
- Carotid dissection
- Functional hemiplegia
- Temporal arteritis
- Air embolism
- Multiple sclerosis
- Dementia

visual field), and aphasia (loss or impairment of receptive or expressive language processing) if the dominant hemisphere is affected. Cerebral lobar hemorrhage occurs in the subcortical white matter of the cerebral hemispheres and frontal hemorrhage produces hemiparesis, particularly of the arm, along with behavioral changes and headache. Patients with thalamic hemorrhage present with complete contralateral hemisensory syndrome and capsular hemiparesis or hemiplegia. Cerebellar hemorrhages are sudden in onset, with nausea, vomiting, dizziness, ataxia, and facial and gaze palsy. Rapid worsening of the patient's condition, progressing to coma, is common. Pontine hemorrhages are characterized by quadriplegia, decerebrate rigidity, ophthalmoplegia, and pinpoint pupils. Caudate hemorrhage is characterized by abrupt onset of nausea, vomiting, decreased level of consciousness, and transient hemiparesis or gaze palsy.[13]

Subarachnoid hemorrhage is caused by bleeding from arteries and veins close to the brain surface.[13] Blood accumulates in the basal cisterns and subarachnoid space. The most common cause of subarachnoid hemorrhage is trauma; nontraumatic subarachnoid hemorrhages are caused primarily by rupture of congenital or berry lesions. Thirty percent of patients with subarachnoid hemorrhage present with loss of consciousness, which is associated with a higher mortality. Complaints also may include acute onset of severe ("thunderclap") headache with vision loss, diplopia, or facial pain as a result of cranial nerve involvement. Emotional upset and vigorous exercise have been noted to precipitate some cases of subarachnoid hemorrhage.

A subdural hematoma may form slowly and, thus, its symptoms may emerge gradually.[13] Indeed, the precipitating trauma may have been forgotten since it may have occurred many days before. Patients may demonstrate fluctuating and false localizing signs. Xanthochromia may be noted in the cerebrospinal fluid, and computed tomography (CT) scan may show the hematoma. By the time of presentation, the hematoma may have become isodense with surrounding cerebral tissue. Chronic subdural hemorrhage has been recognized as a cause of stroke and TIA-like symptoms.[14]

Todd's Paralysis. Partial motor or generalized seizures may precipitate postictal weakness, or Todd's paralysis. Patients may present with a focal motor deficit, which may be weakness of an extremity or a complete hemiparesis. The preceding seizure activity may not have been witnessed or apparent, making this diagnosis difficult. The deficits may persist as long as 24 hours.

Hypoglycemia. The brain is particularly vulnerable to hypoglycemia since glucose is the primary energy source for its metabolism. Patients taking oral hypoglycemics or insulin with a resultant hypoglycemia (defined as a blood glucose concentration of ≤ 45 mg/dL) may present with hemiplegia, hemiparesis, or aphasia with or without alteration of mental status. This syndrome also has been well described in alcoholics with hypoglycemia. Patients with a history of stroke may present with exacerbations of previous stroke symptoms upon becoming hypoglycemic or having other electrolyte abnormalities. The diagnosis can be made rapidly by bedside testing, and intravenous glucose can be administered to correct this disorder. The neurologic deficits may resolve immediately or over a number of hours.[15-19]

Migraine Headache. Migraine headaches are a common occurrence, with a higher incidence in females than in males. Acute hemiparesis may follow development of the headache ("complex migraine"). When this neurologic sign occurs in association with migraines, it usually does so in the initial stages of the headache. The diagnosis of migraine probably should not be considered seriously as an explanation for transient hemisphere attacks unless the patient is young, has repeat migraine headaches, experiences classic visual migraine auras at other times, and has a pounding headache contralateral to the sensory or motor symptoms in the hours after the attack.[20] Familial syndromes of hemiplegic migraine are well recognized and related to a mutation of chromosome.[19]

Bell's Palsy. This entity may present with an acute onset of isolated unilateral facial paralysis in the distribution of the seventh cranial nerve.[21] Deficits usually are maximal within five days of onset. Symptoms include inability to fully close the affected eye, sagging of the lower eyelid, diminished lacrimation, or hypersensitivity to sound, gustatory dysfunction, drooling from the affected side of the mouth, and asymmetry of smile. This condition can be distinguished from cortical causes of facial weakness, as it causes ipsilateral paralysis of forehead muscles (cortical causes usually spare the forehead). CT scan of the head usually is normal. Testing for Lyme disease in endemic areas may be warranted. Treatment includes oral corticosteroids and protection of the affected eye.

Hyponatremia. A serum sodium concentration less than 120 mEq/L often is associated with central nervous system symptoms if it occurs acutely. Clinical findings may include mental status changes, focal neurologic deficits, ataxia, and seizures. In patients with hypovolemic hyponatremia, severe neurologic symptoms should be treated immediately with normal or hypertonic saline, depending on the degree of hyponatremia. The goal of treatment is to correct the sodium deficit by 0.5-1.0 mEq/L/hr. More rapid correction is associated with brain edema. Patients with euvolemic or hypervolemic hyponatremia should be treated with water restriction, diuretics, or demeclocycline.[22]

Hyperglycemia. Hyperglycemia with hyperosmolar state may be associated with focal neurologic deficits that mimic a stroke. Neurologic deficits include aphasia, homonymous hemianopsia,

hemisensory deficits, hemiparesis, unilateral hyperreflexia, and the presence of the Babinski sign.[19] The detrimental effect of hyperglycemia may be related to anaerobic glycolysis, leading to tissue acidosis and increased blood-brain barrier permeability.[23]

Meningitis/Encephalitis/Abscess. Patients with these conditions may present with change in mental status, headache, meningismus, and fever. However, encephalitis is more likely to present with focal neurologic deficits and seizures. Both may present with acute transient symptoms, which usually evolve over days or weeks. Seizures often occur before focal signs are evident, which distinguishes meningitis and encephalitis from stroke. CT scan in a patient with ischemic stroke usually is negative initially, but an enhancing mass may be seen on the CT scan of a patient with a cerebral abscess.[20] Spinal fluid obtained after CT scan may be turbid and show a leukocytosis; organisms may be seen on Gram stain.

Hypertensive Encephalopathy. Hypertensive encephalopathy may occur as a result of an abrupt sustained increase in blood pressure that exceeds the limits of cerebral autoregulation. This occurs at a mean arterial pressure of 150-200 mmHg. At these pressures, cerebral autoregulation no longer can regulate blood flow, and marked vasospasm leads to ischemia, increased vascular permeability, punctate hemorrhages, and brain edema. The onset is acute and patients present with headache, vomiting, drowsiness, confusion, seizures, blindness, focal neurologic deficits, or coma. A careful neurologic examination will help distinguish hypertensive encephalopathy from acute ischemic stroke. Focal deficits in this disorder usually do not follow anatomic patterns. They usually are patchy in distribution or occur on the opposite side of the body. CT scan usually is normal. Hypertensive encephalopathy is a true emergency and is treated with rapid reduction of blood pressure (an action that may be deleterious in acute stroke). Intravenous nitroprusside or labetalol may be used. Symptoms usually resolve rapidly with treatment of blood pressure.[24]

Carotid Dissection. This entity may occur after forced hyperextension or neck trauma. Affected patients may present with focal neurologic deficits. The condition can be diagnosed by magnetic resonance angiography.

Functional Hemiplegia. Functional hemiplegia may be the result of either a conversion disorder or malingering.[19] In a conversion disorder, the disability may be a "cry for help" or the patient may be trying to avoid a certain situation. These patients often appear strangely calm and unconcerned despite their disability. Malingerers usually have secondary gain to their disability, e.g., litigation or avoiding work. Functional hemiplegia can be diagnosed by careful physical examination. The functional deficits rarely occur in an anatomic distribution. The patient's weakness may diminish with coaching, and paralysis will be present in absence of tone or reflex changes. The patient also may demonstrate weakness on foot extension but be able to walk on his/her toes. Functional hemiplegia is a diagnosis of exclusion and must be made only after detailed investigation.

Temporal Arteritis. Temporal arteritis, an uncommon disease entity with an elderly predominance, may be mistaken for a stroke. Patients may present with headache, blurred or loss of vision, and, occasionally, aphasia and hemiparesis. The acute vision loss can be differentiated from visual loss from a posterior cerebral artery occlusion, since the latter gives a homonymous visual field defect that does not violate the vertical midline. Temporal arteritis also presents with scalp tenderness and other systemic symptoms, including malaise, fatigue, arthralgias, and weight loss. The most important sequela of temporal arteritis is permanent blindness; thus, early diagnosis and treatment are critical. A head CT scan will be normal. Definitive diagnosis is by temporal artery biopsy. Laboratory studies may show a sedimentation rate above 100 mm/hr. Treatment is with oral or intravenous steroids, depending on the severity of the symptoms.

Air Embolism. Air embolism should be suspected in patients who have been exposed to acute changes in barometric pressure. The condition also can be caused iatrogenically by inappropriate technique for obtaining venous or arterial access. Apart from the abrupt temporally related onset of focal neurologic symptoms, cardiac auscultation would reveal a harsh new murmur throughout the precordium. These patients should be placed in the left lateral decubitus position to trap the air within the right atrium, where it can be aspirated by Swan-Ganz catheterization. Alternatively, hyperbaric treatment can be used to decrease the size of the bubbles in the cerebral circulation, thus decreasing the area of affected cerebral circulation.[25]

Multiple Sclerosis. The most common presenting symptom of patients subsequently diagnosed with multiple sclerosis is visual or oculomotor disturbances, followed by weakness or a sensory disturbance in one or more limbs.[26] Symptoms generally develop over hours to days, plateau, then decline, but occasionally they may be maximal within seconds to minutes. In this case, vascular causes must be ruled out. Overall, magnetic resonance imaging (MRI) is the most sensitive paraclinical study in the diagnosis of multiple sclerosis. Lesions most frequently are detected with proton density, weighted images, and the fluid attenuated inversion recovery sequence.

Dementia. Posterior cerebral strokes initially present with visual field cuts, an inability to read, and poor ability to manipulate objects and easily could be mistaken for senility in an elderly person. Patients with non-dominant hemispheric lesions initially may experience right-left confusion, leading to disorientation in the home environment. Careful history taking will reveal that these symptoms were of a progressive or insidious onset rather than an abrupt onset.[27]

Diurnal and Seasonal Variations

Cerebral infarction has been positively associated with physical activity, catecholamine levels, blood viscosity, platelet function, coagulability, and fibrinolytic activity. These factors are present with circadian variation. Chronologic patterns of ischemic stroke in the early morning appear to be independent of risk factors and clinical subtypes of stroke. Studies have noted the morning predominance of ischemic stroke between 6 and 10 a.m. and speculate it is correlated with rising changes in heart

Table 2. Modifiable Risk Factors	
• Hypertension	• Cigarette smoking
• Carotid artery disease	• Excessive alcohol use
• Atrial fibrillation	• Physical inactivity
• Diabetes	• Stress
• Hypercholesterolemia	

Table 3. Nonmodifiable Risk Factors for Stroke
• Age
• Race
• Gender
• Genetics and family history

rate and blood pressure, decreased vagal tone, increase in catecholamines, and activation of the renin-angiotensin system, which may make atheroscleotic plaques more likely to rupture and embolize. Another peak in stroke occurrence has been noted between noon and 4 p.m.—the etiology for this is unknown. The fewest stroke events occur between midnight and 4 a.m.[28-31] More strokes are noted during the colder months.

Risk Factors and Prevention

A number of risk factors reliably have been identified as contributing to the development of stroke.[32] Fortunately, some of the factors can be modified by pharmacologic therapy or surgical intervention (*see Table 2*); others cannot be modified (*see Table 3*). Secondary prevention of stroke is aimed at decreasing the proliferation of atherosclerotic disease since ischemic stroke is a cerebrovascular disease.

Modifiable Risk Factors. *Hypertension.* At least 25% of the adult population in the United States has hypertension, making it the single most modifiable risk factor for stroke. Hypertension accelerates atherosclerosis and, thus, increases the risk for stroke four-fold. Blood pressure treatment resulting in a reduction in systolic blood pressure of 10-12 mmHg and diastolic blood pressure of 5-6 mmHg is associated with a 38% reduction of stroke incidence.[33] The Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC-7) recommends lower blood pressure limits than previously recommended. Goals for hypertension treatment now are a blood pressure of less than 140/90 mmHg, or less than 130/80 mmHg in patients with diabetes or chronic kidney disease.[34] The benefit of lower blood pressure is helpful even in hypertensive patients with previous stroke or TIA: A reduction of blood pressure by 9/4 mmHg reduces the risk by one-quarter.[2,35]

Atrial Fibrillation. This dysrhythmia is the most common rhythm disturbance in adults and is associated with more than 120,000 strokes per year in the United States. Patients with atrial fibrillation have a five-fold increase in the risk of stroke and increased mortality compared with patients without atrial fibrillation. The risk of stroke further is increased in patients with recent congestive heart failure, hypertension, high atrial rate, and prior thromboembolism. Atrial high-rate events, defined as an atrial rate more than 220 beats per minute for more than 10 consecutive beats in patients with sinus node dysfunction, is associated with doubling the risk of dying or having a stroke. Anticoagulation in patients with atrial fibrillation decreases the relative risk of suffering a stroke by two-thirds.[2,36]

Asymptomatic Carotid Artery Disease. This term encompasses non-stenosing atherosclerotic plaque and carotid stenosis. About 15% of ischemic strokes are caused by internal carotid artery stenosis. Patients with an asymptomatic carotid bruit have an estimated risk of stroke of 1.5% at the first year and 7.5% at five years. Asymptomatic carotid artery stenosis less than 75% carries a stroke risk of 1.3% annually; with stenosis greater than 75%, the combined TIA and stroke risk is 10% in one year. Carotid endarterectomy of high-grade lesions (70-99%) has been proven to prevent stroke. Absolute risk reduction from carotid endarterectomy is 15% for all strokes.[37,38] The composition of the plaque also is a factor in determining stroke risk. Ulcerated, echolucent, and heterogenous plaques with a soft core, as seen on ultrasound, are at increased risk of arterioarterial embolism.[39]

Previous TIA or Cerebrovascular Accident. Ten to fifteen percent of patients suffer TIAs prior to their cerebrovascular accident. Patients with one or more TIAs have a 10-fold increase in the risk of subsequent CVA. Location of TIAs also is predictive of stroke risk: Hemispheric TIAs carry a greater risk of stroke than retinal TIAs. Previous stroke also is a significant risk for recurrence and for increased morbidity and mortality. This is especially true in the period immediately following the stroke. Recurrent risk varies by stroke subtype, with the greatest risk occurring in patients with atherosclerotic infarction.[32]

Diabetes Mellitus. Diabetic patients have a two- to four-fold increased risk of developing ischemic stroke compared with nondiabetics. Diabetes is associated with development of atherosclerotic disease. In combination with hypertension or hyperlipidemia, the risk of stroke markedly is increased. High insulin levels associated with Type II diabetes increase the risk of atherosclerosis and thus stroke. Diabetics with retinopathy and autonomic neuropathy also appear to be at further increased risk of developing stroke. At this time evidence does not exist that tighter diabetic control decreases the risk of stroke or its recurrence.[2,40]

Hypercholesterolemia. High total cholesterol and low-density lipoprotein levels are associated with atherosclerosis. There is, however, an inconsistent relationship between serum cholesterol levels and death from ischemic stroke; thus, different subtypes of cholesterol confer different levels of risk. Lipid-lowering agents may decrease the development of atherosclerotic plaques and, therefore, possibly cause a regression of plaque formation, leading to a decrease in risk for stroke.[2,41]

Cigarette Smoking. This is associated with a relative risk of brain infarction of 1.7 and also is related to development of subarachnoid hemorrhage in a dose-response fashion. It is a direct risk factor for development of carotid plaque. Smoking cessation always should be encouraged, although more than five

years of cessation may be required before risk is decreased appreciably.[2]

Alcohol Consumption. Heavy alcohol consumption, defined as five or more drinks per day, is associated with increased risk for ischemic stroke. Consumption of up to two drinks per day actually was found to decrease the risk of ischemic stroke (comparing drinkers with non-drinkers). Moderate alcohol intake is associated with an increase in serum high-density lipoproteins. There is, therefore, a J-shaped relationship between alcohol consumption and ischemic stroke.[2,42]

Cocaine and Amphetamine Use. Illicit drug use must be investigated, especially in a young individual presenting with symptoms of stroke. Cocaine is the leading cause of drug-related stroke in the United States. Ischemic stroke can be seen in crack cocaine and amphetamine users, secondary to the drugs' powerful vasoconstrictive effects. In intravenous cocaine users, embolization from infected cardiac valves or injected foreign material can cause cerebral ischemia.

Physical Inactivity. There is a direct relationship between leisure time physical activity and decreased risk of stroke in both sexes and in the Hispanic, Caucasian, and African-American populations. Benefit can be derived from even light physical activity such as walking.[43]

Females and Estrogen. Women who smoke and take high-dose oral contraceptive pills are at increased risk for stroke. The risk of thrombosis increases in the postpartum female, with the highest risk within the first six weeks after delivery.

The Women's Health Initiative[44] was designed to examine a number of factors affecting the health of postmenopausal women. One arm of this study was a randomized trial that assessed the effect of estrogen plus progestin on ischemic and hemorrhagic stroke. In all examined age groups and categories of baseline stroke risk, the risk of ischemic stroke was increased among women taking estrogen and progestin supplementation.

Hemostatic Factors. Risk for development of ischemic stroke is increased in patients with elevated hemoglobin, hematocrit, and blood viscosity. Patients with increased levels of fibrinogen, factor VIII, Von Willebrand's factor, antithrombin III, and lower mean levels of protein C have an increased risk of stroke. Patients younger than age 50 with increased antiphospholipid antibodies are at elevated risk for stroke, as are patients with anticardiolipin antibodies. The reason is unknown.[11]

Pharmacologic Agents for Prevention of Stroke. *Antihypertensives.* Blood pressure reduction is effective for primary and secondary prevention of stroke. Initially, angiotensin-converting enzyme (ACE) inhibitors and ACE receptor blockers (ARB) were thought to have beneficial effects independent of their direct effects on blood pressure. This effect (possibly related to neuroprotective mechanisms) has not yet been proven definitively. Multiple trials have compared the relative efficacies of different classes and combinations of antihypertensives.[45] Thiazide diuretics, beta-adrenergic antagonists, ACE inhibitors, ARBs, and long-acting calcium channel blockers have been found to reduce the incidence of stroke. The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack trial compared high-risk hypertensive patients randomized to receive ACE inhibitors, calcium channel blockers, or thiazide diuretics. In that trial, thiazide diuretics were found to be the most effective antihypertensive in stroke prevention. The relative inexpensive cost of thiazide diuretics also makes them an attractive option for stroke prevention.[46,47] In the Losartan Intervention for Endpoint Reduction in Hypertension trial, losartan was found to be superior to the beta-adrenergic antagonist atenolol for stroke prevention.[48]

Antilipidemic Agents. Treatment with 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (HMG-CoA reductase inhibitors ["statins"]) has been associated with a 25% reduction in the risk of fatal and nonfatal stroke.

A recent trial evaluated the additive benefits of pravastatin and aspirin to decrease risks of cardiovascular disease. In randomized trials of secondary prevention, pravastatin sodium and aspirin reduced risks of cardiovascular disease. Pravastatin has a predominantly delayed antiatherogenic effect, and aspirin has an immediate antiplatelet effect, raising the possibility of additive clinical benefits. In five randomized trials of secondary prevention with pravastatin (40 mg/d), comprising 73,900 patient-years of observation, aspirin use also was prescribed in varying frequencies, and data were available on a large number of confounding variables. Two large clinical trials (Long-term Intervention With Pravastatin in Ischaemic Disease trial and the Cholesterol and Recurrent Events trial) compared the clinical benefits of combined pravastatin plus aspirin therapy with pravastatin alone. A meta-analysis of these two trials and three smaller angiographic trials was performed. In all analyses, multivariate models were used to adjust for a large number of cardiovascular disease risk factors. Both the individual trials and the meta-analyses demonstrated similar additive benefits of pravastatin and aspirin on cardiovascular disease. In meta-analysis, the relative risk reductions for fatal or nonfatal myocardial infarction were 31% for pravastatin plus aspirin vs. aspirin alone and 26% for pravastatin plus aspirin vs pravastatin alone. For ischemic stroke, the corresponding relative risk reductions were 29% and 31%, respectively. For the composite end point of coronary heart disease death, nonfatal myocardial infarction, coronary artery bypass graft, percutaneous transluminal coronary angioplasty, or ischemic stroke, the relative risk reductions were 24% and 13%. All relative risk reductions were statistically significant. The investigators concluded that more widespread and appropriate combined use of statins and aspirin in secondary prevention of cardiovascular disease will avoid large numbers of premature deaths.[49]

Other lipid-lowering therapies (e.g., dietary measures, resins, fibrates) have not been shown to decrease the risk of stroke.[47] The National Cholesterol Education Program has developed guidelines for the treatment of hyperlipidemia. Patients with diabetes mellitus or atherosclerotic disease require more aggressive lowering of lower-density-lipoprotein (LDL) cholesterol. The goal for therapy in these patients is an LDL cholesterol level of less than 100 mg/dL. In patients with hyperlipidemia, one or no cardiovascular risk factors, and no evidence for atherosclerotic disease, the goal of treatment is 160 mg/dL.[47]

Antiplatelet Agents. Aspirin (acetylsalicylic acid) is the most

widely studied and prescribed antiplatelet drug for patients at high risk of vascular disease. It affects a single pathway in the platelet activation process and provides incomplete protection against cardiovascular events. Adenosine diphosphate receptor antagonists, by blocking an alternate pathway of platelet activation, are slightly more effective than aspirin in reducing serious vascular events in patients at high risk, with similar results for the subset of TIA/ischemic stroke patients. Clopidogrel is an effective and safe alternative in patients who do not tolerate aspirin, in diabetics, in hypercholesterolaemic patients, or in those with a previous history of cardiac surgery. Moreover, antiplatelet combination therapy using agents with different mechanisms of action is an attractive preventive approach.

Aspirin, though beneficial for the primary prevention of myocardial infarction (MI), has not been found to be effective for primary prevention of stroke. In fact, the risk of stroke marginally is increased in patients taking aspirin to prevent first events of stroke. Many studies have demonstrated, however, that aspirin is effective in secondary prevention of stroke.[50] Various doses have been studied. Lower dosages have been found to be better tolerated by the gastrointestinal tract and have fewer bleeding complications while providing a 20-25% relative risk reduction for recurrent stroke. Current Food and Drug Administration guidelines recommend 50-325 mg per day of aspirin for secondary stroke prevention.[48] In the Ticlopidine Aspirin Stroke Study, ticlopidine reduced the rate of occurrence of stroke by 21% at three years.[51] Ticlopidine has the significant side effects of diarrhea, rash, and, rarely, neutropenia, which has limited its use in stroke prevention.

Clopidogrel, which is better tolerated by patients and has a more favorable side effect profile, has emerged as the antiplatelet agent of choice for stroke prevention. When clopidogrel was compared with aspirin in patients with stroke, MI, or peripheral vascular disease (the Clopidogrel vs. Aspirin in Patients at Risk of Ischemic Events [CAPRIE] trial),[52] an 8.7% relative risk reduction was demonstrated. At two years, the absolute risk reduction favoring clopidogrel was only 0.5%.[53]

However, more recent studies have investigated the amplified benefit of clopidogrel over aspirin in patients with a history of ischemic events.[54] The goal of this study was to examine the influence of preexisting symptomatic atherosclerotic disease on subsequent ischemic event rates and compare the efficacy of clopidogrel vs aspirin (acetylsalicylic acid) in patients with such disease. Using the CAPRIE database, a group from the Cleveland Clinic performed multivariate analyses for patients who had symptomatic atherosclerotic disease (ischemic stroke or MI) in their medical history before enrollment in the CAPRIE trial. Two composite end points were used: 1) Ischemic stroke, MI, or vascular death; and 2) Ischemic stroke, MI, or rehospitalization for ischemia. In the CAPRIE population, as would be expected, a previous history of ischemic stroke and MI were historical risk factors that predicted subsequent ischemic events. Compared with the overall population, patients with documented symptomatic atherosclerotic disease had elevated event rates for the end point of ischemic stroke, MI, or vascular death; three-year rates were 20.4% with clopidogrel and 23.8% with aspirin (absolute risk reduction, 3.4%; 95% CI, -0.2 to 7.0; number needed to treat, 29; relative risk reduction, 14.9%; P=0.045). Similar results were obtained for the end point of ischemic stroke, MI, or rehospitalization for ischemia; three-year event rates were 32.7% with clopidogrel and 36.6% with aspirin (absolute risk reduction, 3.9%; 95% CI, -0.4 to 8.1; number needed to treat, 26; relative risk reduction, 12.0%; P = 0.039).

The investigators concluded that patients in the CAPRIE study who had known symptomatic atherosclerotic disease were at higher risk for developing neurovascular ischemic events, and that the absolute benefit of clopidogrel over aspirin seemed to be amplified this high risk subgroup. Other recent clinical trial results with antiplatelet therapy also have yielded important implications in stroke prevention, especially as this relates to combination antiplatelet therapy with aspirin and clopidogrel.[55] This approach is supported scientifically by the observation that ASA and clopidogrel inhibit different pathways, and this dual inhibition may confer additional prophylactic benefits in stroke prevention. The CURE (Clopidogrel in Unstable Angina to Prevent Recurrent Events) study evaluated the efficacy and safety of clopidogrel in addition to acetylsalicylic acid vs standard therapy (including aspirin) in more than 12,000 patients with unstable angina or non-ST-segment elevation MI. In combination with aspirin, clopidogrel reduced the relative risk of the combined atherothrombotic endpoint of cardiovascular death, MI, or stroke by 20% (95% CI 0.72-0.90; $p < 0.001$) and the absolute risk of this composite endpoint by 2.1%. While the study was not powered or designed to demonstrate a reduction in stroke, there was a 14% reduction in stroke risk ($p > 0.05$). This approach was associated with an acceptable 1% increase in the incidence of major bleeding events ($p = 0.001$). In PCI-CURE, a prespecified substudy of patients who underwent percutaneous coronary intervention (PCI) during CURE, the benefits of clopidogrel therapy also was demonstrated.[55]

CREDO (Clopidogrel for Reduction of Events During Observation) was a randomized, double-blind, placebo-controlled trial. In this study of about 2100 patients, continuation of clopidogrel in addition to standard therapy including aspirin for 12 months after PCI was assessed, as was the benefit of a preprocedural clopidogrel loading dose. At one year, there was a 27% reduction in the risk of stroke, MI, or death with long-term clopidogrel therapy ($p = 0.02$). There was a consistent benefit of long duration clopidogrel therapy for each component of the composite endpoint, with a 25.1% relative risk reduction for all-cause stroke. In patients who received clopidogrel six or more hours prior to PCI, there was a 39% reduction in the risk of death, MI, or urgent target-vessel revascularization at 28 days ($p = 0.051$). CREDO data suggests an early loading dose of clopidogrel in patients undergoing stenting may be beneficial, and use of a loading dose followed by long-term continuation of clopidogrel in other high-risk atherothrombotic patients such as those with TIA or ischemic stroke may also be effective in prevention of thrombotic events.[55]

Antithrombotic Agents. In patients with atrial fibrillation, warfarin therapy has been found to be efficacious in preventing

strokes. In patients with nonvalvular atrial fibrillation, warfarin therapy is recommended for patients older than 65 years with or without major risk factors (defined as previous stroke, systemic embolism, TIA, hypertension, poor left ventricular function).[3] Close monitoring is required to keep the patient's international normalized ratio (INR) between 2 and 3. The benefit of treating all patients with valvular atrial fibrillation has been well established. Patients should be screened for risk of bleeding before warfarin therapy is initiated.[50]

Nonmodifiable Risk Factors. *Age.* Age is the most important determinant of the likelihood of stroke. After the age of 55, increments of 10 years are correlated with a doubling of stroke rate in both sexes.[4] As the population ages, the prevalence and economic impact of stroke will increase.

Race. African-Americans and Hispanics have a greater stroke incidence than whites across all age groups. There also is an increased mortality from stroke in these minority groups. The increased mortality can be explained only partially by socioeconomic and environmental factors.[4,56,57]

Gender. Overall, the incidence of stroke among males is 30% higher than among females. However, in younger individuals, males and females have an equal incidence of stroke. The increased incidence in young females probably is due to pregnancy and hormonal factors. Males develop ischemic strokes at higher rates than women up to the age of 75 years.

Heredity. A family history of stroke among first-degree relatives is associated with increased risk of cerebral infarction. Parental history is an independent risk factor for stroke. Some inherited diseases can predispose individuals to atherosclerotic and non-atherosclerotic vasculopathies, coagulopathies, and embolisms; examples include Marfan's syndrome, Osler-Weber-Rendu disease, Sturge-Weber syndrome, and Ehlers-Danlos syndrome. Stroke has been reported in patients of all ages with sickle cell disease. Ischemic stroke is more likely in children, and hemorrhagic stroke is more likely in adults, although either type can occur in any age group. Stroke in this population is caused by small or large vessel occlusion, not atherosclerosis. Risk factors for stroke in sickle cell disease are as follows: 1) previous TIA; 2) low steady-state hemoglobin; 3) more frequent or recent acute chest syndrome; and 4) elevated systolic blood pressure. Sixty-seven percent of sickle cell patients with stroke had a recurrent stroke within 12-24 months.[58,59]

TIA Syndromes

TIAs are temporary, focal neurologic deficits of acute onset lasting fewer than 24 hours. Ninety percent of TIAs actually last fewer than 10 minutes. They are a significant warning of ischemic stroke. They can occur from the carotid distribution (90%), the vertebrobasilar distributions (7%), or both (3%).[60]

Carotid TIAs are associated with ischemia to the ipsilateral eye or brain. Symptoms may include any or any combination of the following symptoms:

• Ipsilateral amaurosis fugax: transient blurring or fogging of vision. Only 15% of patients describe the "classic" descending shade over the eye;

Table 4. Stroke Syndromes

- Anterior cerebral artery syndrome
- Middle cerebral artery syndrome
- Posterior cerebral artery syndrome
- Lacunar syndrome
- Vertebrobasilar artery syndrome
- Anterior choroidal artery syndrome

• Contralateral sensory loss or paresthesias;

• Contralateral motor weakness or clumsiness;

• Contralateral homonymous hemianopsia. Patients may have blindness in the visual field contralateral to the ischemic area.

Vertebrobasilar TIAs occur as a result of disrupted blood supply to the brainstem, cerebellum, and visual cortex. Patients will present with the following symptoms:

• Shifting or bilateral motor or sensory loss or paresthesia;

• Bilateral weakness or clumsiness;

• Visual field defects, bilateral or contralateral in homonymous visual fields; or

• Dizziness, diplopia, vertigo, dysphagia, ataxia, or dysarthria.

Stroke Syndromes

Anterior Cerebral Artery Syndrome. (*See Table 4.*)[2,61,62] Clinically recognizable symptoms from anterior cerebral artery occlusion depend on the location of the occlusion and the patency of anastomotic channels. Occlusion proximal to the medial striate artery may produce no discernable symptoms if there is good collateral supply from the opposite anterior cerebral artery. Occlusion proximal to the callosomarginal branch can lead to infarction of a large segment of the frontal lobe, leading to altered mentation, confusion, impaired insight, sucking reflex, and apraxia (inability to perform simple tasks despite full strength in the requisite muscle groups). Paralysis mainly of the opposite leg and mild arm involvement may occur with paralleling sensory deficits. Bowel and bladder incontinence and gait apraxia (wide base of support, short stride, and shuffling gait) also are a part of this syndrome.

Middle Cerebral Artery Syndrome. Middle cerebral artery infarction is one of the most common manifestations of cerebrovascular disease. Symptoms vary depending on the location of the occlusion and the presence of collateral flow. When there is occlusion prior to the lenticulostriate branches, extensive infarctions will occur and involve the internal capsule and the opercular region. Patients experience a dense hemiplegia with paralleling sensory disturbance. Arm and face deficits are worse than those of the leg. Blindness may occur in half the visual field. Right-left confusion, agraphia (loss or impairment of the ability to produce written language), acalculia (impairment of previous ability to perform simple mathematic calculations), aphasia (if the dominant hemisphere is affected), and agnosia (inability to recognize known objects) also can occur.

Posterior Cerebral Artery Syndrome. The posterior cerebral artery supplies portions of the parietal and occipital lobes;

therefore, occlusion leads to vision and thought-processing impairment. Patients may experience blindness in half the visual field, third-nerve paralysis, visual agnosia, altered mental status with impaired memory, and cortical blindness.

Lacunar Syndromes. These occur when small penetrating arteries become occluded. They may be single, multiple, symptomatic, or asymptomatic. Multiple lacunar infarctions are associated with progressive cognitive decline. Five syndromes of lacunar infarcts have been recognized:

• Pure motor hemiparesis—contralateral hemiparesis or hemiplegia, with the face and arm more affected than the leg;

• Pure sensory stroke—characterized by paresthesias, numbness, and a unilateral hemisensory deficit involving the face, arm, trunk, and leg;

• Sensory motor stroke—contralateral unilateral motor deficit with a hemisensory deficit;

• Homolateral ataxia and ataxic hemiparesis—weakness worse in the lower extremity with incoordination of the arm and leg; and

• Dysarthria-clumsy hand—loss of fine motor control of the hand, supranuclear facial weakness, dysarthria, dysphagia, and Babinski's sign are present in this lacunar syndrome.

Vertebrobasilar Artery Syndrome. The posterior inferior cerebellar artery supplies various areas; thus, there are different patterns of infarction. Medial branch territory infarction affecting the vermis and vestibulocerebellum causes vertigo, ataxia, and nystagmus. With lateral cerebellar hemispheric involvement, patients experience vertigo, gait ataxia, nausea, vomiting, gaze palsies, miosis, and dysarthria. With large infarctions, the patient may have altered consciousness or be confused. Patients with anterior inferior cerebellar artery syndrome may have ipsilateral deafness, vertigo, nausea, vomiting, nystagmus, ipsilateral facial hypalgesia (decreased sensitivity to pain), thermoanesthesia, and corneal hypesthesia (increased sensitivity to touch).

Anterior Choroidal Artery Syndrome. This syndrome is rare and can be asymptomatic. However, occlusion of the anterior choroidal artery may produce contralateral hemianopia, hemiplegia, and hemihypalgesia. Because the distribution of this artery is quite variable, symptoms of its disruption show considerable variation. Visual disturbances include a homonymous hemianopsia of variable density with sloping margins. If the posterior limb of the internal capsule is infarcted, contralateral hemimotor or sensory loss may occur. If the retrocapsular sensory and visual radiations are infarcted, a superior homonymous quadrantopia may occur. Middle cerebral artery syndrome may be mimicked by the dysphasia, apraxia, and hemineglect that can be associated with the anterior choroidal artery syndrome.

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CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

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Physician CME Questions

41. Which of the following conditions mimics stroke?
    A. Migraine headache
    B. Bell's palsy
    C. Hyperglycemia
    D. Multiple sclerosis
    E. All of the above

42. Which of the following statements is true?
    A. Ischemic stroke accounts for 80-85% of all cerebrovascular accidents.
    B. Ischemic stroke can result from thrombosis, embolism, or hypoperfusion.
    C. The ischemic penumbra is the target of stroke treatments.
    D. All of the above

43. Which of the following is true of stroke?
    A. Stroke is no longer a significant cause of death in the United States.
    B. The incidence of stroke is higher in females than males.
    C. The relative risk of stroke is increased in African-Americans and Hispanics.
    D. The incidence of stroke is increased in the northeastern United States.

44. The most common cause of subarachnoid hemorrhage is trauma.
    A. True
    B. False

45. Patients may reduce risk of stroke by:
    A. reducing blood pressure to 140/90.
    B. ceasing alcohol use.
    C. carotid endarterectomy of 50% lesion.
    D. tight diabetic control.

46. What is the single most modifiable risk factor for stroke?
    A. Hypercholesterolemia
    B. Hypertension
    C. Diabetes
    D. Alcohol use

47. Which of the following is the most important determinant of the likelihood of stroke?
    A. Gender
    B. Race
    C. Cigarette smoking
    D. Age

48. A patient presents to the emergency department complaining of aphasia, right-sided weakness (arm more than leg), and gaze preference to the left. This patient most likely is suffering from:
    A. an anterior cerebral artery infarct.
    B. a cerebellar infarct.
    C. a middle cerebral artery infarct.
    D. a posterior cerebral artery infarct.

49. The five recognized syndromes of lacunar infarcts are pure motor hemiparesis, pure sensory stroke, sensory motor stroke, homolateral ataxia and ataxic hemiparesis, and dysarthria.
    A. True
    B. False

50. Which of the following statements is true regarding stroke?
    A. Most strokes are hemorrhagic in origin.
    B. Ipsilateral paralysis of forehead muscles is diagnostic for stroke.
    C. Stroke has a morning predominance.
    D. Given the greater longevity of females, stroke has an overall increased incidence in females.

Emergency Medicine Reports CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

In Future Issues: **Stroke, Part II**

CME Answer Key

41. E
42. D
43. C
44. A
45. A
46. B
47. D
48. C
49. A
50. C

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Evaluation and Management of Blunt and Penetrating Thoracic Trauma

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Trauma to the thoracic cavity is responsible for approximately 10-25% of all trauma-related deaths,[1,2] with the majority of these deaths occurring after arrival at the emergency department (ED). The mortality for isolated chest injury is relatively low (less than 5%); however, with multiple organ system involvement, the mortality approaches 30%.[1]

This article dissects the critical aspects of thoracic trauma and highlights acute care management strategies.

— The Editor

Introduction

Thoracic trauma may be either blunt or penetrating. Compression, deceleration forces, and direct impact are mechanisms that result in the injury patterns seen in thoracic trauma. Unlike blunt trauma, penetrating trauma results from extrinsic violation of the integrity of the thoracic cavity. These injury mechanisms may result in pneumothorax, hemothorax, pulmonary contusion, or injuries to the mediastinal structures. With the proliferation of firearms, penetrating chest injuries are becoming more prevalent, and nearly all result in development of a pneumothorax, with hemothorax occurring in almost 75% of the cases.[1] The use of scoring scales such as injury severity scale, abbreviated injury severity scale, or the thoracic trauma severity score may assist the ED physician and trauma team in algorithmic decision-making. Although controversies abound regarding management of traumatic arrest patients, the decision to perform thoracotomy should be individualized. Moreover, fewer than 10% of blunt chest injuries and 15-30% of penetrating injuries require thoracotomy.[2]

Morbidity and mortality from chest injuries results from the injury pattern itself and physiologic derangements such as hypoxia, hypercarbia, and acidosis. The pathophysiology of these clinical entities arises from inadequate tissue perfusion from hypovolemia (secondary to blood loss), ventilation and perfusion mismatch from pulmonary contusion, or change in intrathoracic pressure from either tension or open pneumothorax.[2]

The presence of severe respiratory distress is associated with a high mortality, with 10% of these patients requiring intubation at the scene or immediately on arrival in the ED.[1] The most common associated risk factors for respiratory distress include multiple rib fractures, shock, pneumothorax, hemopneumothorax, and coma.[1]

Thoracic Injuries

Thoracic cage injuries result from the direct effect of trauma to the chest wall or the thoracic cage. They include flail chest and rib, sternal, thoracic spine, and scapular fractures.

Rib Fractures and Flail Chest. The ribs are the most commonly injured structures in the thorax.[2] This injury should be suspected in patients with localized chest wall pain or contusion over one or more rib segments after trauma. The upper ribs, first through third, are well protected by the bony framework of the upper limb; protective structures include the scapula, humerus, and clavicle, along with their muscular attachments.[2] Fracture of the first, second, or third rib requires a significant amount of force and should lead to high index of suspicion for injuries involving the head, neck, spinal cord, tracheobronchial tree, lungs, or the great vessels.[1,2] The mortality associated with these fractures is approximately 15-30%.[1,2]

The middle ribs, four through nine, are most commonly affected by blunt trauma.[2] A direct force striking anterior-to-posterior on the thoracic cage fractures these ribs along their shafts and tends to drive the ends of the bones into the thoracic cavity. This subsequently leads to intrathoracic injuries such as pneumothorax or lung contusion.[2] Furthermore, fracture of the lower ribs (ribs 10-12) should heighten suspicion for intra-abdominal injuries involving the liver and spleen.

A flail chest results when fractures occur in two or more ribs, or a fracture involves multiple segments of a rib. This results in a lack of continuity between the fractured segments and the rest of the thoracic cage, resulting in the disruption of normal chest wall movement. The morbidity from flail chest is not from the paradoxical chest wall movement produced, but rather from the underlying injury to the lung parenchyma (pulmonary contusion), and persistent splinting that results from severe pain. The pulmonary contusion leads to ventilation perfusion mismatch, leading to hypoxia. *(See Figure 1; see more on pulmonary contusion in the section dedicated to this entity.)*

Elderly and Pediatric Patients. The presence of rib fractures in older individuals carries significant morbidity and mortality. An elderly patient with a rib fracture resulting from blunt chest trauma has twice the morbidity and mortality of younger patients.[4,5] For each additional rib fracture, mortality increases by 19%.[4] Similarly, pediatric patients who present with rib fractures should alert the practitioner to possible child abuse. Although a rib fracture could occur in a pediatric patient who sustains blunt chest trauma, it is very rare. Its presence portends the possibility of concomitant severe injuries. Thus, geriatric and pediatric patients with multiple rib fractures warrant extreme vigilance.

The initial evaluation of a patient with a rib fracture involves palpation for localized pain, crepitus, subcutaneous emphysema, and deformities. In simple rib fractures, chest radiography alone will suffice. The purpose of the study is not to visualize the rib per se, but to evaluate for possible coexisting complications such as pneumothorax. Patients who sustain rib fractures from significant trauma to the thoracic cage, in addition to chest radiography, require computed tomography (CT) of the chest to qualify the extent of intrathoracic organ involvement (e.g., hemopneumothorax or aortic, bronchial, esophageal, cardiac, or diaphragmatic injuries).

Initial therapeutic intervention for an isolated rib fracture focuses on adequate ventilation and analgesia for pain control. Many therapeutic regimens are available for pain management. These modalities include opioid analgesics, transcutaneous electric nerve stimulation, non-steroidal anti-inflammatory drugs, and regional nerve blocks (intercostals, epidural, interpleural, and thoracic paravertebral).[6,7]

In patients with simple flail chest and minimal or no lung contusion, analgesia or intercostal nerve blocks with chest physiotherapy may be adequate. In patients with flail chest and moderate to severe pulmonary contusion, evidence of hypoxia, and signs of shock, mechanical ventilation should be initiated. Mechanical ventilation should be considered in individuals with flail chest and shock, three or more associated injuries, severe head injury, underlying pulmonary disease, fracture of eight or more ribs, or age older than 65 years.[1] Bergeron et al showed that after adjusting for severity, co-morbidity, multiple rib fractures, and age, patients older than 65 years had five times the odds of dying when compared to those younger than 65 years.[8] In patients with flail chest and two or more injuries, early mechani-

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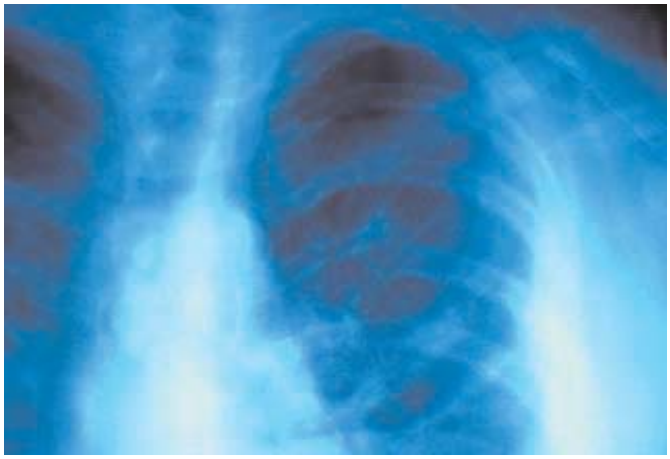
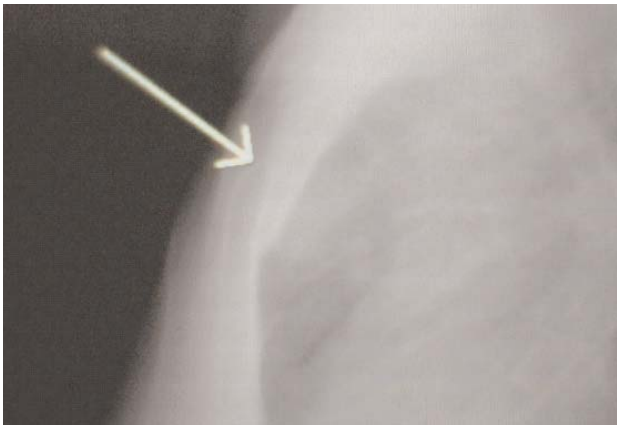
Figure 1. Rib Fractures with Pulmonary Contusion



The above image demonstrates multiple rib fractures and a pulmonary contusion.

Figure 2. Sternal Fracture with Posterior Displacement



A 78-year-old man who sustained a seatbelt injury in a motor vehicle accident. The arrow shows a sternal frature with posterior displacement. (Image courtesy of A. Adewale, MD.)

cal ventilatory support has been shown to reduce the mortality from 69% to 7%.[1]

The disposition of a patient with a rib fracture resulting from blunt chest trauma should be individualized. Patients with isolated rib fractures may be discharged home safely with good analgesia and an incentive spirometer. In patients with multiple rib fractures, admission often is recommended for pain control and to minimize the potential associated morbidities. All geriatric patients with multiple rib fractures require admission for management.[4,5,8]

Sternal Fracture. Sternal fractures commonly result from a direct blow to the chest wall during motor vehicular accidents or from falls, and account for approximately 8% of thoracic injuries.[9] The majority of these injuries result from direct contact with the steering wheel or from seatbelt compression. The presence of a sternal injury should alert practitioners to the possibility of an underlying cardiac or pulmonary injury. However, in the absence of concomitant injuries and other comorbidities, the incidence of morbidity is only about 5.4%,[9] and mortality is approximately 0.7-0.8%.[1,9] Previously, fractures of the sternum were believed to be harbingers of underlying cardiac injury; however, multiple recent studies[9-12] have proven otherwise. It is prudent to remember anecdotal cases of ventricular wall rupture associated with a sternal fracture.[13] *(See Figure 2.)*

The initial diagnostic modalities for sternal fracture are posterior-anterior and lateral chest radiographs. Although some studies have reported better diagnostic yield with sternal ultrasound and bone scintigraphy,[14,15] chest radiography still remains the initial diagnostic aid.

The extent of the evaluation and management should be guided by the severity of the injury and co-morbid factors. Most studies recommend a baseline electrocardiogram (ECG) and creatinine kinase (CK-MB),[1,11,12] with a 2-D echocardiogram reserved for those patients whose clinical presentation warrants the diagnostic test. Althought there are significant variations in institutional management of sternal fractures, the disposition of these patients should be individualized. Most patients with an isolated sternal fracture and no significant underlying medical problems may be discharged safely with appropriate analgesia.[10-12]

Thoracic Spine Injury. The thoracic spine supports the posterior segment of the thoracic cage. The first 10 vertebrae are fixed owing to their articulation with the thoracic cage.[1] Thoracic spine fractures often result from a fall from a height, a motor vehicle collision (especially with ejection), or a motorcycle accident.[16] Motorcycle accidents are the most common mechanisms because of the forced hyperflexion of the thoracic spine.[17,18] Fractures of the thoracic spine usually result from an excessive force, and an anatomically narrow thoracic spinal canal leads to a high incidence of associated neurologic complications.[1,2] Thoracic spinal fractures include anterior wedge compression, burst, chance, and fracture dislocation injuries. *(See Figure 3.)*

The initial evaluation of a patient with a possible spinal injury involves palpation of the spine for step off or subluxation, deformity, or midline tenderness. In most minor blunt chest trauma patients, an ordinary thoracic spine radiograph is adequate for evaluation. However, in severe multi-trauma patients with decreased levels of consciousness, severe alcohol intoxication, or respiratory distress requiring mechanical ventilation, it may be difficult to adequately assess the thoracic spine with routine radiographs. In these patients, CT of the spine has been shown to have better sensitivity, specificity, and negative and positive predictive values when compared to routine thoracic spine radiography.[19-21] Hence, in severe multi-trauma patients, CT scan of the thoracic spine should be the diagnostic study of choice.

The initial therapeutic management of a potential thoracic spinal injury associated with chest trauma involves spinal immobilization until radiographic clearance. However, because of the high incidence of spinal cord involvement associated with thoracic spine injury, the early utility of steroids is very controver-

Figure 3. Thoracic Spinal Fracture

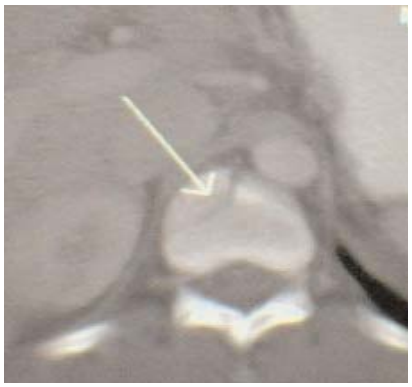
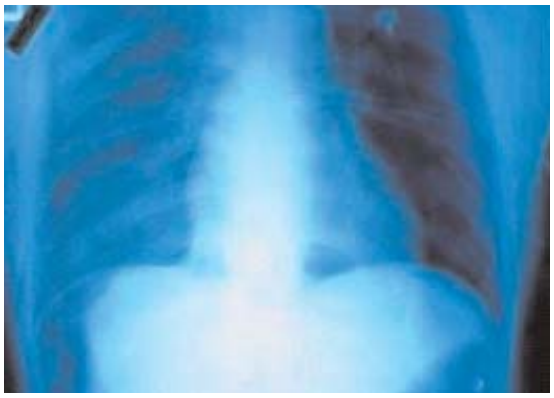


Image courtesy of A. Adewale, MD.

Figure 4. Hemopneumothorax



The chest radiograph above demonstrates a hemopneumothorax in a 49-year-old male involved in a motor vehicle collision.

sial. While some studies support the utilization of corticosteroids,[22,23] others do not consider it as a mandated standard of care, but rather as a treatment option.[24,25] Currently, the National Acute Spinal Cord Injury Studies (NASCIS) advocates the early use of steroids.

Intrathoracic Injuries

Pneumothorax. Pneumothorax results when air enters the potential space between the parietal and visceral pleura. This injury can be caused by either penetrating or blunt trauma.[2] The most common cause of pneumothorax in blunt trauma is a lung laceration with air leak.[2] It is a common complication of chest trauma, and a recognizable cause of preventable death.[26,27] About 6.7% occur without rib fractures, while the incidence increases with number of rib fractures.[28]

For the normal functioning of the pulmonary apparatus, the thorax is completely filled by the lungs, which are held to the chest wall by surface tension between the pleural surfaces. The presence of air in the pleural space leads to eventual collapse of the lungs. This collapse results in a ventilation/perfusion (V/Q) mismatch, because the blood perfusing the non-ventilated area is not oxygenated.[2] This ultimately results in respiratory distress and hypoxia. Early in the course of a pneumothorax or a small pneumothorax, the patient may be asymptomatic. However, as the intrathoracic pressure increases because of air in the pleural space, a simple asymptomatic pneumothorax may become a life-threatening tension pneumothorax.

The diagnosis of pneumothorax often is accomplished by clinical examination and chest radiography. However, in a severely traumatized patient, supine chest radiography may miss a pneumothorax.[29] Adjunctive diagnostic aids, such as ultrasonography and CT scan, have been shown to have both high sensitivity and specificity in diagnosing pneumothorax.[29,30]

The management of simple pneumothorax is expectant. However, when mechanical ventilation is indicated, prophylactic chest tube thoracostomy should be performed to prevent tension pneumothorax. In acutely dyspneic patients with hemodynamic instability after chest trauma, rapid needle decompression followed by tube thoracostomy is the standard of care.

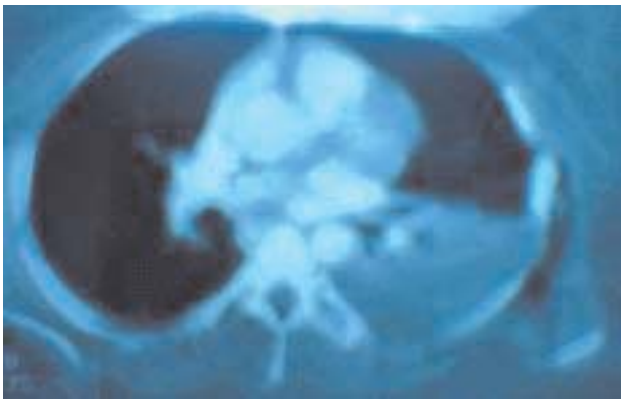
Hemothorax. Hemothorax commonly results from lacerations to the lungs, intercostal vessels, or the internal mammary artery due to either blunt or penetrating trauma.[1,2] Patients with large hemothoraces often are dyspneic, with some degree of respiratory distress because of restricted ventilation. *(See Figure 4.)*

The diagnosis of hemothorax should be made promptly. Although the majority of these injuries are diagnosed during the initial phase of assessment and management of the patient, there have been reported cases of delayed manifestation up to eight hours after initial presentation.[31] The initial diagnostic method is the chest radiograph, and a hemothorax that is large enough to be apparent on chest radiograph should be evacuated. However, in a supine trauma patient, up to 1000 mL of blood could be missed on a radiograph.[1] Studies showing the utility of ultrasound have produced mixed results.[32,33] The use of CT scans has been shown to be the most sensitive and specific for diagnosing and accurately assessing hemothoraces in chest trauma patients.[34-36] CT adequately quantifies the extent of the injury complex and the underlying complications.

The initial management of a hemothorax involves the insertion of a large-caliber chest tube for drainage, or open thoracotomy. In the majority of patients with hemothoraces, tube thoracostomy alone is adequate and is effective in more than 80% of the cases.[37] In the presence of persistent bleeding, videothoracoscopic evaluation and treatment have been shown to be as effective as open throracotomy, and minimize the complications that accompany thoracotomy.[37] The presence of more than 1500 mL of blood in the initial chest tube drainage, drainage of more than 200 mL an hour for 2-4 hours, or ongoing transfusion requirements mandate surgical exploration with open thoracotomy.[2] *(See Figure 5.)*

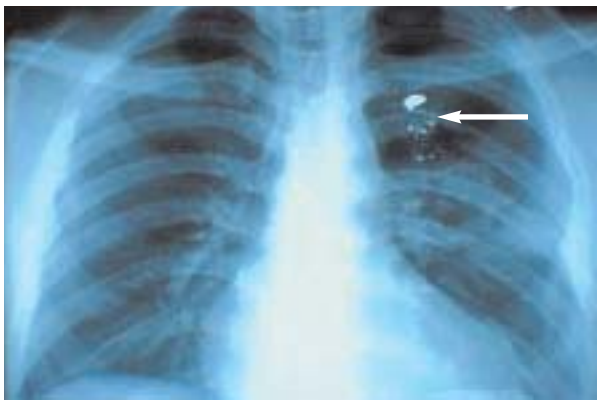
Pulmonary Contusion. Contusion to the lung parenchyma is a significant cause of morbidity and mortality resulting from thoracic trauma. It is the most common cause of lethal chest injury.[2] Usually a result of direct force to the chest wall, the injury results

Figure 5. Hemothorax and Pulmonary Contusion



A CT scan of the chest that demonstrates a hemothorax and pulmonary contusion. (Images courtesy of A. Adewale, MD.)

Figure 6. Rib Fracture from Gunshot Wound, with Pulmonary Contusion



An isolated rib fracture from a gunshot wound to the chest with a pulmonary contusion. The arrow indicates a bullet fragment.

from a coup and countrecoup effect. The transmitted force produces direct damage to the lung parenchyma and associated hemorrhage and edema. In the majority of cases, respiratory failure develops over days rather than instantaneously.[2]

The pathophysiology of pulmonary contusion is poorly understood, and only minimal advances have been made in the past 20 years.[38] The primary injury to the lung results in increased blood flow to the uninjured lung and parenchyma due to reflex decrease in pulmonary vascular resistance. This results in the extravasation of fluid into the alveoli and the interstitial spaces. With aggressive resuscitation, the level of the edema and blood in the lung parenchyma and interstitial spaces increases, thus producing a V/Q mismatch that is manifested as worsening hypoxia, hypercarbia, and acidosis.[1]

The diagnosis of pulmonary contusion is dependent on the extent of the lung parenchyma involved. Initial radiographic manifestations may be minimal or significant, depending on the individual patient. Even so, the chest radiograph should be the initial diagnostic aid, and for the majority of patients will show diffuse opacification of the involved lung parenchyma. *(See Figure 6.)* CT scan of the chest may be used in patients to adequately identify the exact lung segment involved, quantify the contusion volume, and act as a prognosticator of morbidity and mortality.[39,40]

The management of pulmonary contusion depends on the severity, associated injuries, and co-morbid conditions of the patient. The physiologic parameters that determine the severity of a pulmonary contusion in a chest trauma patient include: oxygen saturation less than 90% that is not responding to routine oxygen supplementation (nasal canula, bag-valve-mask, or facemask); PaO_2 less than 65 mmHg; persistent tachypnea or FiO_2/PaO_2 less than 300.[1,2,41]

The primary management goal is to maintain adequate ventilation. The severity of the injury determines the modality of mechanical ventilation utilized. The need for intubation should be individualized, since the majority may be managed non-invasively.[42] In a patient with a contused lung, the plateau airway pressure is increased, while static compliance and $ETCO_2$ is decreased.[43] Independent lung ventilation (ILV) now is a commonly used modality for managing pulmonary contusion, because it is effective in reducing V/Q mismatch, improving oxygenation with a setting of tidal volume (TV) and positive end-expiratory pressures (PEEP) to keep pulmonary plateau pressure below safe thresholds for barotrauma.[42,43] Presently, the use of lung protective ventilatory strategy with low TV and high PEEP has become a standard practice.[43,44] In most severe cases, placing the patient in a decubitus position (involved lung dependent position), use of nitrous oxide (because of its less dense characteristics), utility of pressure-control ventilation with paralysis, and the use of high-frequency oscillator have been shown to improve survival.

Blunt Myocardial Injury. Blunt myocardial injury (BMI) commonly is caused by high-speed motor vehicle collisions, although injuries resulting from low-velocity collisions have been reported.[1] Due to different criteria used in diagnosing BMI, the incidence is difficult to determine. However, the prevalence ranges from 3-56%, depending on the study. The mechanisms of this injury include direct blows, athletic trauma, industrial crush, blasts, rapid deceleration, or falls from a height.[1]

A direct blow to the precordium (such as the chest wall striking the steering wheel in a motor vehicle collision, or the handlebar or a guardrail in a motorcycle collision, or being struck by a high-velocity object such as a baseball) creates a force that compresses the myocardium against the spine. *(See Figure 7.)* Because of the relatively free anterior-posterior movement of the heart, the momentum generated from a rapid deceleration accident maintains the heart in a uniform, straight-line motion, resulting in a direct strike against the internal sternum.[1,2] BMI includes injuries such as cardiac contusion, cardiac concussion (commotio cordis), cardiac chamber rupture, or valvular disruption.

The manifestations of BMI originate from a direct injury to the myocardium that results in sub-endocardial hemorrhage, in turn leading to the formation of localized edema and the mobi-

lization of the inflammatory response system. The resulting inflammatory changes cause a redistribution of coronary flow that may manifest as ischemic type chest pain.[1] The severity of the presentation is contingent upon the underlying coronary artery disease, since transient coronary redistribution can produce a total or near-total occlusion of coronary vessels that already are stenosed. The worsening stenosis may lead to signs of acute coronary syndrome or acute myocardial infarction (AMI).

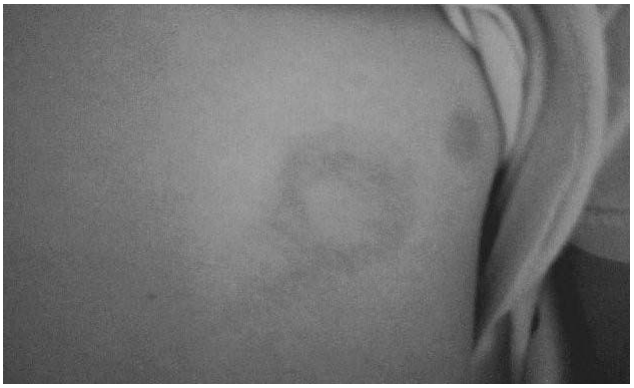
The clinical manifestations of this injury, in addition to ischemic or ischemic-like chest pain, include rhythm and conduction disturbances (e.g., multiple premature ventricular contractions and premature atrial contractions, atrial fibrillation, ventricular tachycardia, etc.), tachycardia out of proportion to volume loss or pain, right bundle-branch block (RBBB), and heart failure.[1,2] Also, the clinician should be suspicious of BMI in the presence of increased central venous pressure (CVP) and the absence of obvious cause.

Presently, there is no gold standard for diagnosing cardiac contusion. The true diagnosis only can be ascertained through direct visualization of the injured myocardium. Currently utilized diagnostic aids include:

- Chest radiography—The presence of pulmonary contusion or sternal fracture on chest x-ray should lead to a high index of suspicion for BMI;
- ECG—Its utility is controversial. However, recent studies have shown its advantages as a stratification and medical decision-making tool. Velmahos et al show that "the combination of normal ECG and cardiac TnI at admission and 8 hours later rules out the diagnosis of significant blunt cardiac injury."[45]
- Cardiac enzymes—Its utility also is controversial. However, multiple recent studies[46,49-52] are beginning to show its usefulness in determining if a patient has sustained significant BMI. Mori et al showed that "abnormal titers of cardiac TnI suggesting myocardial contusion may be found in more than half of the patients with blunt chest trauma."[46]
- Echocardiography—This is a very good screening and adjunctive test for evaluating BMI. Because of the low yield and lack of specificity of transthoracic echocardiography (TTE), transesophageal echocardiography (TEE) is the modality of choice when BMI is suspected. Chirillo et al showed that TTE "has a low diagnostic yield in severe blunt chest trauma, while transesophageal echocardiography provides accurate diagnosis in a short time at the bedside."[47]
- Magnetic resonance imaging (MRI)—The ability of cardiac MRI to adequately visualize the entire myocardium and the adjoining great vessels might increase its utility in chest trauma patients. Descat et al reported two cases of myocardial contusion diagnosed using MRI.[48]

The management of BMI depends on the clinical manifestations. Although the majority of the cases are benign, some may present with AMI,[53,54] persistent hypotension, or cardiogenic shock that warrants acute intervention. Recent literature proposes the use of ECG and TnI as the initial screening tools. If the initial ECG and TnI (four hours after the injury) are normal in the absence of concurrent injuries, the patient safely may be discharged from the ED.[52] However, if the ECG or the TnI is abnormal, further workup is indicated. The patient should be admitted to a monitored unit with serial cardiac enzymes and a TEE to adequately evaluate the cardiac apparatus. In the absence of abnormalities on TEE and the normalization of cTnI, the patient should be reassessed, and the clinical parameters should determine the disposition.

Figure 7. Blunt Myocardial Injury



A 7-year-old boy who presented with dizziness and chest pain after being struck on the chest by a baseball. (Image courtesy of A. Adewale, MD.)

Commotio Cordis. Commotio cordis, or cardiac concussion, is the most common cause of traumatic death in an athlete.[55-57] It causes sudden or near cardiac death in the absence of structural abnormalities, and results from an object directly striking the chest wall at a phase of ventricular depolarization.[56] Ventricular fibrillation is the most commonly induced arrhythmia. The survival rate is very low when the condition is not recognized. With the popularity of automatic electric defibrillators in public places, the survival from this phenomenon could be improved, since the ventricular fibrillation may respond to rapid defibrillation.

Esophageal Injury. Traumatic esophageal injury is very uncommon.[2,57] When it occurs, injury to the esophagus most commonly results from penetrating trauma; however, it also can result from blunt injury to the lower thoracic cavity. Upper esophageal injury may accompany lower cervical or upper thoracic spine injuries, while distal injuries are rarely caused by blunt chest trauma.[57,58] The mechanism of this injury is the forceful expulsion of gastric contents into the esophagus caused by a severe blow to the lower chest wall or upper abdomen. The resulting increase in intragastric pressure being transmitted to the esophagus results in a linear tear of the lower esophagus. This tear leads to the leakage of gastric contents into the mediastinum.[2]

The clinical presentation of the disease is similar to that of an esophageal tear secondary to persistent, profuse retching. In a severely traumatized patient, the diagnosis initially may be missed, and delayed diagnosis may lead to increased morbidity and mortality.

Esophageal rupture should be suspected in a patient with blunt trauma and one of the following features:

- Left hemothorax or pneumothorax without rib fracture;
- A significant blow to the lower sternum or epigastria with pain out of proportion to the apparent injury;
- Presence of particulate matter in chest tube drainage; or
- Presence of pneumomediastinum.

The diagnosis of esophageal perforation is very challenging. If suspected, a non-ionic contrast esophagogram should be performed.[1] However, this study carries a high false-negative rate, and in patients with a high degree of suspicion for this injury, flexible esophagoscopy in conjunction with esophagogram may increase diagnostic yield.

The management of esophageal perforation is very controversial and is guided by the location of the perforation (hypopharynx, cervical, thoracic, or distal). While some authors propose surgical management for all perforations,[59-63] some propose conservative management based on Cameron's criteria (i.e., minimal signs of sepsis, disruption contained within the mediastinum, drainage of cavity back into the esophagus, and minimal symptoms).[64] According to Rios et al, "Conservative treatment should be considered for patients meeting Cameron's criteria, since their evolution is favorable with low morbidity and mortality."[64]

Despite technical and nutritional advances, the mortality rate for esophageal injuries ranges from 5-25% for those treated within 12 hours, to 25-66% for those treated after 24 hours.[1] Regardless of the management approach, esophageal perforation is a life-threatening condition that requires early diagnosis and management to minimize the morbidity and mortality.

Diaphragmatic Injury. Blunt injury to the diaphragm is uncommon, occurring in about 0.8-8% of hospitalized chest trauma patients.[65] The incidence of the laterality of this injury varies depending on the study. However, most studies report the incidence of left-side injury to be between 60-90%.[1,66-68] This probably is because of the protection provided by the liver on the right and the possible left posterior lateral weakness of the diaphragm.[1,2] Because of the significant contribution of the diaphragm to normal ventilation, injury to this structure may lead to significant respiratory compromise.

This injury often initially is missed unless the defect is large enough to cause acute herniation of the abdominal viscera into the thoracic cavity. In this instance, the chest radiograph will show the gastrium in the thoracic cavity or a coiled nasogastric tube in the thoracic cavity. Smaller defects may cause a delay in diagnosis until the abdominal viscera slowly migrates into the thoracic cavity, causing compression of the adjacent lung leading to either bowel strangulation or tension pneumothorax.[1]

Diaphragmatic injuries create a diagnostic dilemma. The chest radiograph is diagnostic in most cases of large ruptures; however, in the small defects, the injury often is missed or the chest radiograph is misinterpreted as showing an elevated diaphragm, acute gastric dilatation, a loculated pneumohemothorax, or a sub-pulmonary hematoma.[2] The general consensus for the modality of choice for diagnosis is the helical CT; multiple studies have shown helical CT scan with sagittal and coronal reconstruction to be very sensitive in diagnosing diaphragmatic injury.[65,69,70] In one study, sensitivity approaches 84%, with specificity of 77%.[70] The most accurate modality for diagnosis is MRI. MRI is capable of directly acquiring both coronal and sagittal images, and allows the evaluation of the entire diaphragm, and shows the exact site and size of rupture in all cases as reported in one study.[69] However, its usefulness is limited in the acutely traumatized patient. Its utility is beneficial in otherwise stable patients with blunt chest trauma with a high index of suspicion for isolated diaphragmatic injury. With technological advances in the field of thoracic surgery, videothoracoscopic evaluation of the diaphragm in the hand of an experienced surgeon is emerging as a diagnostic modality.[71,72]

The management of diaphragmatic injury mostly is surgical. Most trauma patients with suspected diaphragmatic injury undergoing exploratory laparotomy for any intra-abdominal injury should have the diaphragm evaluated for possible defect. However, with the advances in laparoscopic techniques, thoracolaparoscopic repair is becoming the modality of choice for repair of an isolated diaphragmatic injury.[71] Isolated diaphragmatic injury in the absence of other surgical injuries carries low mortality, and Bergeron et al showed that operative repair can be deferred without appreciable increase in mortality if no other indications mandate surgery.[72]

Blunt Aortic Injury. Blunt traumatic thoracic aortic injury (BAI) is a rare, but very lethal, condition. It often occurs as a result of a decelerating injury from high-speed motor vehicle collision (low-speed in older population) or a fall from a height.[1] The incidence of BAI is about 6.8 per 100,000 motor vehicle occupants, with a steady increase with increasing age.[73] Approximately 80-90% of the patients die at the scene,[1,73] and up to 50% of the remaining patients die within 24 hours if not promptly diagnosed and treated.[1] Of the survivors who make it to the hospital, the ultimate survival rate is lower for patients who are older than 60 years.[73]

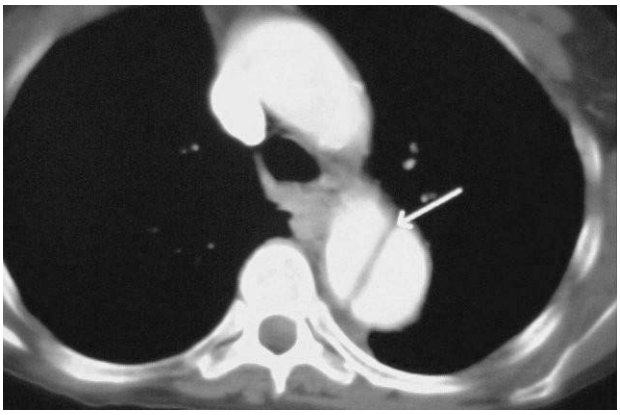
Three mechanical factors contribute to aortic rupture. These factors include shearing stress, bending stress, and torsion stress.[1] As deceleration occurs, a gradient is created between the mobile aortic arch and the immobile descending aorta. This gradient places the aortic isthmus under tension, and the resultant shearing stress can lead to rupture or tear opposite the fixation site.[1] Bending stress results from the hyperflexion of the aortic arch produced by the downward traction exerted by the heart, and torsion stress results from anterior posterior compression of the chest, with the heart displaced to the left combined with an intravascular pressure wave transmitted to the aorta.[1] When combined, these three forces produce maximum stress to the inner surface of the aorta at the ligamentum arteriosum, which is the point of greatest fixation.[1]

The clinical presentation of BAI is vague, since specific signs and symptoms often are absent.[2] Most patients with free rupture, as elucidated earlier, die at the scene. However, patients with contained rupture who make it to the hospital may exhibit transient hypotension, dysphagia, hoarseness, or acute dyspnea.[1,74] Although clinical factors (e.g., multiple rib fractures, flail chest,

Table 1. Radiographic Findings Suggestive of Aortic Injury[1,2]

- Widened mediastinum
- Aortic knob obliteration
- Rightward deviation of the trachea
- Obliteration of the aorto-pulmonary window
- Left mainstem bronchus depression
- Rightward deviation of the esophagus
- Paratracheal stripe widening
- Presence of apical cap
- Left hemothorax

Figure 8. Thoracic Aortic Dissection



CT-scan of a 78-year-old female involved in a motor vehicle accident. The arrow shows a thoracic aortic dissection. (Image courtesy of A. Adewale, MD.)

pulse deficits, or hoarseness without laryngeal injury) may be suggestive of BAI, about one-third of patients with this injury have no external signs of thoracic injury on initial physical examination.[1]

The diagnosis of BAI requires a high index of suspicion. A chest radiograph remains the most appropriate initial screening modality, with a negative predictive value approaching 95%.[75] The supine chest x-ray may not show the classic findings; however, the presence of a widened mediastinum mandates further investigation. On chest x-ray, the presence of a widened mediastinum and a hemothorax in a patient with transient hypotension should increase the suspicion of aortic injury.[74] *(See Table 1.)*

The radiographic algorithm utilized after the initial chest x-ray should be dictated by the patient's clinical condition. Although aortography still is the recognized gold standard, fast spiral helical CT scan has emerged as a diagnostic study that potentially will supplant aortography.[76] Multiple studies[77-80] have shown spiral CT angiography to have a sensitivity of 96-100%, specificity of 98-100%, and accuracy of 99-100%. In the hemodynamically stable patient, contrast-enhanced helical CT has a critical role in the exclusion of BAI and prevents unnecessary thoracic aortography.[80]

The role of TEE in the evaluation of BAI has been well documented.[75,77] Since the specificity and sensitivity of TEE are similar to those of helical CT, the indication for TEE is for the hemodynamically unstable patient with suspected BAI.[75,77] Although TEE and CT have similar diagnostic accuracy, TEE allows for the diagnosis of associated cardiac injuries, and is more sensitive than CT for the identification of intimal or medial lesions of the thoracic aorta.[77] *(See Figure 8.)*

While aortography still is the recognized gold standard for diagnosing BAI, in the case of equivocal aortographic findings, intravascular ultrasound (IVUS), with sensitivity approaching 92%, and specificity of 100%, could be used as an adjunctive diagnostic aid.[81] The role of MRI in the evaluation of trauma patients remains indeterminate. Despite sensitivity and specificity approaching 100% for aortic injury, MRI's utility in trauma patients is not feasible logistically because of the requirements of a metal-free environment and the long period of time that the patient must lie in isolation in a quiet room.[1]

The management of BAI typically is surgical following the initial resuscitation using the American College of Surgeons Advanced Trauma Life Support protocol. Emergent surgical intervention is the accepted standard of care. However, in some selected cases, such as patients deemed to be at high operative risk because of associated injuries or pre-existing medical conditions, or in stable patients for whom conditions for surgery are not ideal, delayed surgical intervention may be warranted.[1] The surgical approach utilized is institutionally dependent.

Tracheobronchial Injury. Injury to the tracheobronchial area occurs very rarely and often is associated with other injuries.[84] Although this injury potentially is fatal, it often is overlooked on initial assessment.[2] The reported mortality for tracheobronchial injury (TBI) has fallen from 36% in the 1950s to less than 9% in the 1970s.[85] The mechanism of this injury results from the effect of rapid deceleration on a relatively mobile bronchial structure and its fixed proximal segments.[1] The majority of these injuries occur within 2 cm of the carina, or at the origin of the lobar bronchi.[1,86]

The common clinical presentations of TBI are signs of respiratory distress (dyspnea, stridor, or hemoptysis), subcutaneous emphysema, and sternal tenderness.[1,87,88] The presence of pneumomediastinum, pneumothorax, widened mediastinum, or deep cervical emphysema on chest radiograph may suggest TBI.[1,2,84,88]

The diagnosis of TBI requires a high index of suspicion. The morbidity and mortality increases if not diagnosed early.[87] The initial screening modality is the chest radiograph, which often demonstrates the signs suggestive of TBI (as described in the paragraph above). According to one study, the CT scan of the chest demonstrated similar findings to the chest radiograph, but failed to confirm the diagnosis.[89] The presence of the "fallen lung" sign (a collapsed lung in a dependent position hanging on the hilum by its vascular attachment) on CT scan is highly suggestive of TBI.[84] Tracheobronchoscopy is the definitive diagnostic modality of choice.[1,2,84,88] On bronchoscopy, the injury pattern visualized is a transverse tear in the main bronchus involved or a disruption at the origin of an upper lung bronchus, while the trachea shows a vertical tear in the membranous portion near its attachment to the tracheal cartilages.[1]

Since most patients with TBI present with pneumothorax or tension pneumothorax, initial needle decompression with subsequent thoracostomy tube placement is required. The presence of persistent air leak with proper chest tube placement and drainage is highly suggestive of TBI until proven otherwise.[1,2,89]

The presence of persistent hypoxia despite intubation and chest tube placement mandates the use of temporizing opposite main stem bronchus intubation to provide adequate oxygenation,[2] and also minimizes the effect of the ventilation and perfusion mismatch. In some instances, blind endotracheal intubation may be difficult, owing to anatomic distortion from pharyngeal injuries, paratracheal hematoma, or the TBI itself. For these patients, immediate operative intervention is required.[2] However, in the more stable patients, acute surgical intervention could be delayed until inflammation and edema resolve.[2,90] The definitive surgical treatment involve the reestablishment of the anatomic continuity of the tracheobronchial tree if the lesion affects more than one-third of the circumference.[86]

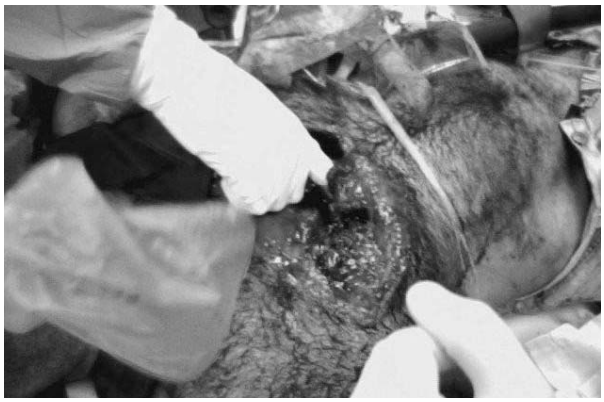
Finally, independent of mechanism and anatomic location of injury, delay in diagnosis is the single most important factor influencing outcome.[91]

Penetrating Chest Injury. Penetrating chest injuries (PCIs) are more common in urban medical centers. Most of these injuries involve firearms and knives. Injuries involving the cardiac, vascular, and transmediastinal structures are the most lethal, with prehospital mortality approaching 86% for cardiac injuries, and 92% for extrapericardial vasculatures.[92] Of patients who make it to the hospital alive, only about 5-15% require thoracotomy.[1] Of those who survive to the hospital, the mortality for those with cardiac injury is 21.9%, and 1.5% for those without cardiac injury.[93] Survival rates from stab wounds generally are much higher than those from gunshot wounds.[1,93]

The injury pattern in PCI may include extensive lung laceration *(See Figure 9),* a sucking chest wound, or mediastinal traversing injuries. A sucking chest wound acts as a one-way valve that allows air to enter the pleural cavity during inspiration and none to leave during expiration. This eventually leads to an expanding or tension pneumothorax.[1] In the prehospital setting, it is imperative that the wound be covered with occlusive dressings on only three sides. This allows air to escape the pleural cavity during expiration and, thus, prevents development of a tension pneumothorax. On arrival to the ED, the wound should be examined and covered completely with occlusive dressing, with simultaneous insertion of a chest tube at a site other than the initial injury location.[1,2]

Wounds that traverse the mediastinum may involve the great vessels, heart, tracheobronchial tree, or the esophagus.[2] The overall mortality for these injuries approaches 20%.[2] The evaluation of these injuries in hemodynamically stable patients can be performed non-operatively.[94] Trauma ultrasound may be used for assessing pericardial tamponade, spiral CT for evaluating transmediastinal injuries, and organ-specific studies (e.g., esophagogram, aortography, bronchoscopy, thoracolaparoscopic evacuation of retained hemothorax, or repair of diaphragmatic injury) are minimally invasive management techniques for stable PCI patients.[94]

Figure 9. Penetrating Chest Injury



This penetrating chest injury was sustained by a motorcyclist who struck an embankment in a high-speed collision (Image courtesy of A. Adewale, MD.).

In hemodynamically unstable patients, there should be a high index of suspicion for exsanguinating thoracic hemorrhage, tension pneumothorax, or pericardial tamponade.[1,2] In this situation, a bilateral chest tube thoracostomy is warranted to decompress possible hemopneumothorax and document volume of blood in chest tube drainage.[2] The performance of ED thoracotomy is mainly for evacuation of pericardial blood, direct control of exsanguinating hemorrhage, open cardiac massage, or cross-clamping the descending aorta to slow blood loss below the diaphragm and increase perfusion to the brain and heart.[1,2] With a bleak survival report for emergent ED thoracotomy, each facility should develop a uniform guideline for performance of this procedure. *(See* Trauma Reports *2003;4:1-12 for a thorough discussion of ED thoracotomy.)* Recent studies[95,96] show that the presence of signs of life on arrival to the hospital, in addition to the mechanism of injury and location of major injury, should be the determinants of the indications for emergent thoracotomy.

Conclusion

Thoracic cavity trauma carries significant morbidity and mortality because of the vital structures it involves. With technologic advances, most of these injuries now can be evaluated with minimally invasive diagnostic aids. The evaluation and management of injuries involving this cavity should be individualized, with special consideration for the pediatric and geriatric population.

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CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

CE/CME Questions

1. The most commonly injured structure(s) in the thorax is/are:
   A. the sternum.
   B. the clavicle.
   C. the ribs.
   D. the lungs.

2. The indication for surgical exploration or videothoroscopic evaluation of hemothorax in a chest trauma patient is:
   A. initial chest tube drainage of 500 mL.
   B. the drainage of 100 mL/hr of blood for 2-4 hrs.
   C. spontaneous resolution of bleeding.
   D. initial chest tube drainage of more than 1500 mL.

3. The "fallen lung sign" is suggestive of which injury?
   A. Tension pneumothorax
   B. Esophageal tear
   C. Tracheobronchial injury
   D. Hemopneumothorax

4. One of the physiologic parameters that determine the severity of pulmonary contusion is:
   A. initial oxygen saturation less than 95%.
   B. initial PaO_2 less than 65 mmHg.
   C. initial PaO_2/FiO_2 less than 400.
   D. initial respiratory rate of 42.

5. The gold standard for diagnosing cardiac contusion is:
   A. electrocardiogram.
   B. cardiac enzymes.
   C. echocardiogram (TEE or TTE).
   D. There is no gold standard for diagnosing cardiac contusion.

6. The most commonly induced arrythmia in commotio cordis (cardiac concussion) is:
   A. ventricular tachycardia.
   B. asystole.
   C. ventricular fibrillation.
   D. supraventricular tachycardia.

7. Esophaphageal rupture should be suspected in blunt trauma patients with which of the following features?
   A. Multiple rib fractures
   B. Left hemothorax or pneumothorax without rib fractures
   C. Lack of particulate matter in chest tube drainage
   D. Persistent retching

8. A coiled nasogastric tube in the thoracic cavity is indicative of which of the following injuries?
   A. Blunt aortic injury
   B. Tracheobronchial injury
   C. Diaphragmatic injury
   D. Pulmonary contusion

9. Regarding the diagnosis of blunt aortic injury, which of the following statements is *not* true?
   A. Aortography is still the gold standard.
   B. CT scan angiography is as specific and sensitive as aortography.
   C. Magnetic resonance imaging is the most specific and has replaced aortography as the gold standard.
   D. Transesophageal echocardiography is quick, sensitive, specific, and enables evaluation of the cardiac apparatus.

10. Which of the following statements regarding transmediastinal injuries (stab or gunshot) is true?
    A. A gunshot wound to the heart has a better survival rate.
    B. A stab wound to the heart has a better survival rate.
    C. Survival rates for stab wounds and gunshot wounds to the chest are the same.

CME Objectives

Upon completing this program, the participants will be able to:

a.) Recognize or increase index of suspicion for thoracic trauma;

b.) Identify how to correctly and quickly stabilize and manage thoracic trauma;

c.) Employ various diagnostic modalities for thoracic trauma; and

d.) Recognize both likely and rare complications that may occur.

Answer Key

1. C	6. C
2. D	7. B
3. C	8. C
4. B	9. C
5. D	10. B

In Future Issues: **Trauma Ultrasound**