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The Practical Journal for Emergency Physicians

Emergency Medicine Reports®

Volume 27, Number 15

July 10, 2006

Managing Acute Decompensated Heart Failure in the Emergency Department: Part I

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Introduction

Heart failure (HF) continues to cause substantial morbidity and mortality in the United States, accounting for a higher portion of Medicare costs than any other disease.[1] In fact, this disease is reaching epidemic proportions. HF is the leading cause of hospitalization in patients over the age of 65,[2] and it is the most common reason for re-hospitalization in the same group.[3] The rate of hospitalization for heart failure increased 165% from 1979 to 2000.[2] In 2004 alone, there were more than 1 million heart failure hospitalizations in the United States, and another 1 million in Europe.[3]

To fully understand this epidemic, the impact of comorbid illnesses, patient demographics, and cost need to be explored. Within the general patient population exists a growing number of patients older than age 65. Unfortunately, HF is predominantly a disease of the elderly. The impact of HF is further compounded by improved survival following many previously fatal events, including myocardial infarction and

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Farel (CME question reviewer) owns stock in Johnson & Johnson. Dr. Peacock (author) serves on the speaker's bureau and receives grant/research support from Scios and Biosite; receives research/grant support from and is a retained consultant for Inovise Inc.; and receives grant/research support from Cardiodynamics. Drs. Tallman (author), Sarko (peer reviewer), and LoVecchio (peer reviewer), and Dr. Schneider (editor) report no relationships with companies related to the field of study covered by this CME activity. Dr. Stapczynski (editor) reports that he served as a consultant to Pfizer on the drugs linezolid and dalbavancin. Dr. Bosker (editor in chief) has been compensated for speaking engagements and/or editorial services related to production of peer-reviewed Clinical Consensus Reports under the auspices of unrestricted educational grants for Pfizer, Sanofi-Aventis, Bristol-Myers Squibb, Roche Pharmaceuticals, Bayer, Novartis, Forest Pharmaceuticals, and Schering Plough Corporation. Dr. Bosker also acknowledges that he has received royalties, commissions, and other compensation relating to the sale of textbooks, reprints of articles, and/or other electronic or print materials to the following pharmaceutical companies: Pfizer, Sanofi-Aventis, Bayer, Roche, Forest Laboratories, and Novartis. He is a minor stockholder in Pfizer. Any other stock ownership which he may have in other pharmaceutical companies is managed in blinded fashion by an independent consultant without Dr. Bosker's input or consultation.

chronic disease processes (e.g., hypertension).[2] The demographics have changed and large databases show that clinical trials do *not* accurately identify the true HF population.

A more accurate reflection of the heart failure population is represented by the Acute Decompensated Heart Failure National Registry (ADHERE), a data set that currently contains information on more than 200,000 acute decompensated heart failure (ADHF) hospitalizations and, more importantly, can be used to determine what currently represents the population and standard care of ADHF. Limitations include that it is a self-reporting registry and therefore is not considered the gold standard. Having said this, Table 1 compares clinical trial patients with patients from the ADHERE database. Note that while randomized controlled trials provide data that suggest a therapy or test can work, registry data tell if a therapy or test does work (as real world issues can negate the most effective therapy). A comparison of patients enrolled in ADHF randomized controlled trials vs. ADHERE registry data reveals that in the registry, the average patient is much older, more often female, and more often burdened by co-existent disease.

It is estimated that 80% of patients who present to the emergency department with acute decompensated heart failure (ADHF) will be hospitalized, and repeat admissions are common. In an analysis of 100,000 ADHF hospitalizations from ADHERE, nearly 25% of patients had a history of prior HF hospitalizations in the 6 months preceding their index admission.[2] Information regarding process of care is also provided by this registry. Of the total patients admitted for ADHF, 78% presented through the ED, and 89% reported shortness of breath as the chief complaint. Once hospitalized, 79% of ADHF patients were placed to either telemetry (66%) or the ICU (13%). Although initially small in number, observation unit admissions increased approximately 100% between 2003 and 2004, and are expected to increase as the Center for Medicare and Medicaid Services has identified this as an area to increase.

Altogether, the costs for heart failure hospitalizations approximate $14.7 billion annually, and outpatient costs are estimated to equal this number.[1] Among patients hospitalized with heart failure, almost 80% are receiving Medicare benefits, and these Medicare costs outweigh the costs for patients treated for acute myocardial infarction or any single cancer. Follow-up costs also remain high, and one study estimated that one-year follow-up costs of treating heart failure exceed those of initial hospitalization by 50%.[4]

When considering the progress made regarding treatment and understanding of myocardial infarction, it is unfortunate that no major United States cardiac or emergency medicine professional organization has published guidelines on the treatment of acute decompensated heart failure (ADHF).[3] While many chronic heart failure management guidelines exist and are promoted, the European Society of Cardiology is the only organization to have produced guidelines for acute management. Recommendations for the management of pulmonary congestion in the setting of acute myocardial infarction were published in 2004 by the American College of Cardiology and the American Heart Association.[5] The emergency department is the common gateway to the hospital for many of these patients. Thus, the burden is on the emergency physician to recognize ADHF early and initiate treatment well in advance of disposition to any inpatient hospital setting. An estimated 80% of ED visits for HF result in hospitalizations.

For the current standard of ADHF to represent optimal care, then treatment strategies must be successful in reducing the frequency of hospitalizations and ED visits as well as reducing the days spent in the hospital. In addressing this objective, the greatest success has come with developing a team approach to the patient with recurrent decompensated heart failure and with the advent of observation units.[1] With a balanced team approach, careful discharge planning, and outpatient care, patient outcomes improve and visits to the emergency department decrease.

One measure of successful ED treatment strategies is to evaluate chronic heart failure in the outpatient community setting. The ADHERE Disease Management Quality Initiative for Care Beginning in the Emergency Department Module (ADHERE ED DM) was initiated to provide insight into the treatment patterns and overall quality of disease management (DM) of ADHF in the emergency setting. This registry recorded more specific data on the emergency medicine encounter for ED patients in ADHF.

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by Thomson American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
Editorial Group Head: Glen Harris
Specialty Editor: Shelly Morrow Mark
Marketing Manager: Nan Webb

GST Registration No.: R128870672

Periodicals postage paid at Atlanta, GA. **POSTMASTER:** Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $31. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, $359 each; 10 to 20 additional copies, $319 each.

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Table 1. Randomized Controlled Trials vs. ED Patients

RANDOMIZED CONTROLLED TRIALS	
Age	50-60 years old
Sex	70-80% Men
Co-morbidities	Diabetes 20-25%
Renal insufficiency	Infrequent (mean Cr 1.1-1.3)
Ventricular function	75-80% Systolic Dysfunction (LVEF < 0.40)
PAC use	30-40%
In-hospital mortality	1.5-2.5%

PATIENTS FROM ADHERE DATABASE	
Median age	75 years
Female gender	52%
Race White	72%
Black	20%
History of HF	75%
Ejection fraction < 40%	59%
Hospitalized within 6 months	23%
Chronic renal Insufficiency (creatinine > 1.5 mg/dL)	30%
Systolic BP > 90 mmHg at presentation	98%
Initial BNP	667 pg/mL

The database offers insight into what currently represents the population and "standard of care" of ADHF treatment.[6]

Pathophysiology of Heart Failure

One of the most important challenges in the emergency department is to accurately diagnose heart failure. Patients may experience a period of latent or asymptomatic heart failure prior to ED presentation, and this makes it difficult to determine the etiology of their disease. Furthermore, this is primarily an elderly population with multiple comorbidities, and thus ADHF does not occur as a homogenous ED population. The combination of false-positive and false-negative diagnoses has been reported as high as 18.5% in the emergency setting.[7-9]

Understanding the pathophysiology of this disease is imperative to developing the approach to diagnosis and treatment. Although ADHERE data suggest that a greater number of patients with HF have hypertension than coronary artery disease, the presence of coronary artery disease is the underlying pathology in a significant number of patients.[10,11] Cardiac markers such as troponin and creatinine kinase isoenzymes may be elevated in HF. Such elevation can predict prognosis.[12,13] An increase in cardiac troponins has been associated with a poor prognosis in patients with ADHF and are related to the severity of HF.[14] Thus measures to prevent the development of heart failure also center on resolving the risk factors associated with coronary artery disease, which include hypertension, obesity, smoking, diabetes, and dyslipidemia (though not immediate issues to address in the ED).[15]

ADHF, or cardiogenic pulmonary edema, is characterized by the transudation of excess fluid into the lungs secondary to increased left atrial pressure and subsequently pulmonary venous and capillary pressures. The net result is protein-poor fluid filtering across the pulmonary endothelium into the alveolar spaces. This leads to decreased diffusion capacity, hypoxia, and dyspnea. Cardiogenic pulmonary edema can manifest from either left atrial outflow impairment or left ventricular systolic or diastolic dysfunction. Despite different pathophysiologic initiators, symptoms at ED presentation initially will be identical, making treatment concerns supercede that of ejection fraction determination.

Left atrial outflow impairment can be acute or chronic. In chronically impaired left atrial outflow states (*see Table 2*), pulmonary edema precipitates from decreased left ventricular filling time as a result of rapid heart rate (HR). Acutely impaired left atrial outflow arises from increased left ventricular end-diastolic pressures, which lowers the ability of the left atrium to fill the left ventricle. Less common causes of impaired left atrial outflow include mitral stenosis and left atrial tumors.

Pulmonary edema also can result from left ventricular systolic and diastolic dysfunction,[15] left ventricular outflow obstruction, or left ventricular volume overload. Increased vascular volume can also precipitate pulmonary edema and can occur with pregnancy or increased salt intake. Reduced cardiac output as a result of a contractile deficit is the most common cause of cardiogenic pulmonary edema (CPE). The most common causes for systolic dysfunction are listed in Table 2. The impaired forward flow caused by systolic dysfunction activates the renin-angiotensin-aldosterone (RAAS) and sympathetic nervous system (SNS). This compensation leads to renal sodium and water retention and pulmonary edema.

Diastolic dysfunction is defined by an increase in ventricular stiffness, reduced compliance, and impaired ventricular filling during diastole. It can be induced by both acute and chronic disorders. It is further defined as increased left ventricular end diastolic pressure with a left ventricular ejection fraction greater than or equal to 40%. Diastolic dysfunction can exist with systolic dysfunction or can be an isolated process. Other factors that can lead to pulmonary edema during diastolic dysfunction include concurrent systolic dysfunction, reduced diastolic coronary blood flow, and arrhythmia. Echocardiography can be a useful tool in diagnosing diastolic dysfunction as the cause of an acute event and can identify normal ejection fraction ranges. Left ventricular volume overload can result from ventricular septal rupture, aortic insufficiency, and sodium retention due to renal disease. Acute aortic insufficiency is associated with congenital heart disease or may be the result of infective endocarditis, acute aortic dissection, lupus, or trauma. Left ventricular outflow obstruction can result from critical aortic stenosis, hypertrophic cardiomyopathy, or severe systemic hypertension.

To summarize, systolic dysfunction is due to pump failure, which typically results from ischemic cardiomyopathy, dilated cardiomyopathy (DCM), or valvular disease. Diastolic dysfunction is a factor in approximately one-third of ADHF cases and is associated with hypertension, hypertrophic cardiomyopathy, and infiltrative disease. High output causes of ADHF are less common, and are associated with anemia, hyperthyroidism, and pregnancy.[16]

Neurohormonal Imbalance in ADHF

Left ventricular dysfunction results from myocardial injury.

Table 2. Etiology of Cardiogenic Pulmonary Edema

IMPAIRED LEFT ATRIAL OUTFLOW	SYSTOLIC DYSFUNCTION	DIASTOLIC DYSFUNCTION	LV VOLUME OVERLOAD
Elevated heart rate -V Tach -atrial fibrillation -exertion -fever -stress	Coronary heart disease Hypertension Valvular heart disease	LV hypertrophy Hypertrophic cardiomyopathy Restrictive cardiomyopathy	Ventricular septal rupture Aortic insufficiency Renal disease
Mitral stenosis	Idiopathic dilated cardiomyopathy	Ischemia	LV outflow obstruction
Atrial myxoma		Acute hypertensive crisis	Critical aortic stenosis
Prosthetic valve thrombus		Concurrent systolic dysfunction	Hypertrophic cardiomyopathy
Congenital defects		Reduced diastolic coronary perfusion	Severe systemic hypertension
Mitral insufficiency		Arrhythmia (Afib)	
Mitral regurg. Secondary to ischemic heart disease			

Changes occur in the peripheral circulation that also affect cardiac function and contribute to the symptoms of HF. Vasoconstrictors—those in the sympathetic nervous system and the RAAS, as well as arginine vasopressin and endothelin—become activated to increase afterload and preload by conserving sodium ions and water. Vasodilators—hormones in the natriuretic peptide system, as well as endothelin-derived relaxing factor and prostaglandins—help to unload the left ventricle and promote natriuretic action. In other words, the actions of atrial natriuretic peptides (ANP) and plasma brain natriuretic peptides (BNP) counteract the activation of the RAAS and sympathetic nervous system. Interventions, such as angiotensin-converting enzyme inhibitors (ACEIs) and beta blockers, have been developed to prevent ADHF by blocking the RAAS and SNS. Table 3 illustrates the physiologic effects of neurohormones and the counteraction of the natriuretic peptide system on the RAAS. Figure 1 illustrates the progression of heart failure following acute LV systolic dysfunction.

Natriuretic Peptides

Natriuretic peptides (NP) represent a series of proteins released as a result of volume stimulus. First studied in 1981 by Debold it has since become clear that natriuretic proteins exert a counter-balancing influence on many of the deleterious consequences of neurohormonal activation.[16] ANP originates from the cardiac atria and is increased in response to atrial distension. BNP is released in response to increased ventricular stretch or pressure. Its endogenous concentration can be assayed in the forms of BNP and NT–pro BNP. There are multiple assays available for BNP and at least two assays available for the NT–pro BNP. Only BNP is available on a point-of-care testing platform. Physiologically BNP and NT–pro BNP are very different from each other, but both are helpful to confirm the diagnosis of heart failure, especially when the signs and symptoms are ambiguous. The upper limit cutoff of normal varies between the two tests and the assays used.

Once in circulation, BNP binds to natriuretic peptide receptors (NPRs) which are located on endothelial and smooth muscle cells. The physiologic actions of BNP occur when it binds to NPR A and B receptors, which causes an increase in cyclic GMP. In essence, the natriuretic proteins exert a counterbalancing influence on many of the deleterious consequences of neurohormonal activation. BNP is metabolized by three pathways: binding to the NPR-C receptor, cleavage via neutral endopeptidase, and to a lesser extent by renal elimination. NT-pro BNP relies predominantly on renal elimination.

Clinical Features

Presenting Symptoms. Using a symptom-based approach, ADHF is not a homogenous presentation to the ED, but can be divided into two distinct syndromes, defined by the nature of their presentation. The two presentations are manifested as volume overload or hemodynamic mismatch and can be distinguished by the acuity of their symptoms and their response to vasodilation.[3]

In patients with hemodynamic mismatch, function is relatively near their baseline until they suffer a precipitous decompensating event, at which time they present to the ED with severe respiratory distress. The precipitating event leads to a decrease in CO- e.g., MI, dysrhythmia. Fluid overload generally is limited and the response

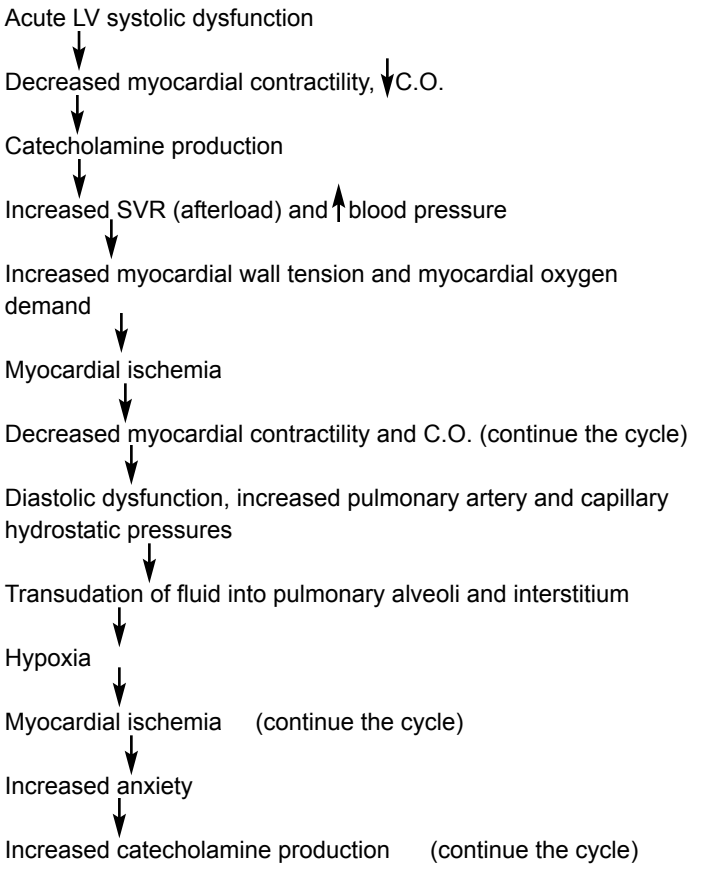
Table 3. Physiologic Effects of Neurohormones

RAAS (RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM)	
Activation of AT_1 receptors by angiotensin	Vasoconstriction Sodium retention Increased aldosterone release Increased cellular growth Increased sympathetic nervous activity
NATRIURETIC PEPTIDE SYSTEM	
ANP, BNP	Vasodilation Sodium excretion Decreased aldosterone levels Inhibition of RAAS Inhibition of sympathetic nervous activity Antiproliferation of vascular smooth muscle cells

Key:
ANP = atrial natriuretic peptide; AT_1 = angiotensin I;
BNP = endogenous B-type natriuretic peptide

Reference: Burrell JC Jr. *J Hypertens* 1999;17(suppl 1):837-843.

Figure 1. Pathophysiology



Adapted from: Mattu A. Heart Failure: Advances in Diagnosis and Treatment, ACEP Scientific Assembly 2005: 2.

to vasodilation is remarkable. They can, in fact, return to being nearly asymptomatic within minutes of treatment with vasodilators despite little volume being removed. The underlying pathophysiology in this cohort is hypothesized to be excessive systemic vascular resistance resulting from diminished cardiac output.[17]

Acute pulmonary edema presents clinically as tachypnea, rales, and respiratory distress due to pulmonary congestion. Dyspnea, especially exertional dyspnea, is the most common symptom of HF.[2] An increase in respiratory rate (> 16) often accompanies dyspnea, but is a nonspecific symptom seen with a wide variety of pulmonary, cardiac, chest wall, or even neurologic disorders. Other manifestations include orthopnea (breathing discomfort precipitated by lying supine) and paroxysmal nocturnal dyspnea or sudden onset of dyspnea at night. Heart failure could also manifest as fatigue, increased frequency of nocturnal dyspnea, and increased systemic venous pressure, which contributes to the GI complaints.

In the volume overload patient, the acuity of presentation is that of moderate respiratory distress, and complaints generally are driven by exertional limitations or the discomfort from excessive volume. Diuretics can remove large volumes from these patients, and they may have partial improvement with vasodilation, but their symptoms only improve with volume removal.

Symptoms of HF can be divided into those due to left- sided or right-sided heart failure. Symptoms of left-sided failure include dyspnea, orthopnea, paroxysmal nocturnal dyspnea (PND), nocturia, fatigue, and altered mental state. Symptoms of right-sided heart failure are the result of elevated systemic venous pressure and include peripheral edema and GI complaints such as anorexia and nausea. A physical finding of jugular venous distention (JVD) may be the most important exam finding for estimating volume status.[2]

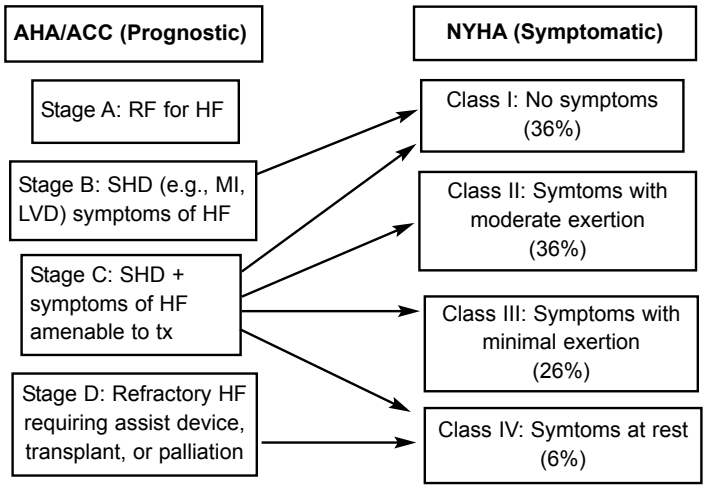
The New York Heart Association (NYHA) class severity for HF patients (*see Figure 2*) represents an historical standard for categorizing clinical severity of HF. NYHA class is directly proportional to survival. There is, however, no direct relationship between NYHA and ejection fraction (EF). New York Heart Association class does suffer from interobserver variability.[18] Brain-type natriuretic peptide assay correlates are provided and likely provide a more objective measure. The mortality rate in NYHA class III patients is 15% to 20% per year. In class IV the mortality rate increases to 50% per year or more.

The American College of Cardiology and the American Heart Association have published a classification system and includes patients who are in asymptomatic initial stages of HF. This system does recognize the fact that earlier intervention has the potential for greater morbidity and mortality benefits as compared to therapy provided in later stages.[19,20]

Differential Diagnoses

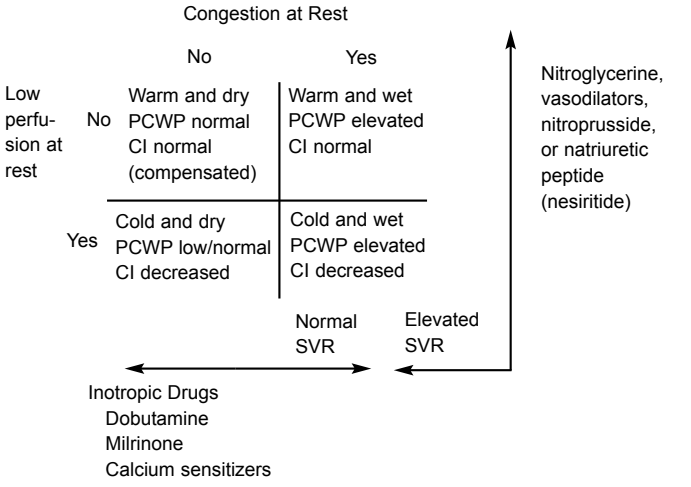
There are a number of clinical conditions that can mimic HF

Figure 2. Heart Failure Classification Systems



Key: RF = risk factor; SHD = structural heart disease
Source: *Pharmacotherapy* 2003;23:997-1020

Figure 3. Hemodynamic Profile Assessment for Patient Selection and Treatment



Signs and Symptoms of Congestion
- Orthopnea/PND
- JVD
- Ascites
- Edema
- Rales

Possible Evidence of Low Perfusion
- Narrow pulse pressure
- Sleepy/obtunded
- Low serum sodium
- Cool extremities
- Hypertension with ACE inhibitor
- Renal dysfunction (one cause)

Table 4. Heart Failure Differential Diagnosis for Dyspneic States

- Chronic obstructive pulmonary disease exacerbation
- Asthma exacerbation
- Pulmonary embolus
- Acute myocardial infarction
- Physical deconditioning
- Obesity
- Pleural effusions
- Pneumonia/pulmonary infection
- Pneumothorax/tension pneumothorax
- Fluid retentive states
- Renal failure/nephritic syndrome
- Liver failure/cirrhosis
- Portal vein thrombosis
- Hypoproteinemia
- Deep venous thrombosis
- Dependent edema
- Low cardiac output states
- Pericardial tamponade
- Sepsis

and the wrong diagnosis can delay optimal response to treatment or cause further decompensation. Shortness of breath is such a common presenting symptom that other conditions causing dyspnea need consideration. Table 4 summarizes the differential diagnosis for dyspnea in the clinical setting. For instance, impending pulmonary embolism (PE) can present with intense dyspnea but in the absence of rales or edema.

It is imperative to be able to distinguish between two diagnoses such as pulmonary edema and PE due to the potential for rapid deterioration. In the patient with pulmonary edema, arterial blood pressure often is elevated and requires immediate therapy with vasodilators. Decreasing preload in a patient with PE may lead to deterioration.

A useful approach to categorizing heart failure in the ED is by hemodynamic classification (*see Figure 3*). Patients can be placed into the appropriate hemodynamic category based on initial history and physical and then appropriate therapy can be selected.[2] For example, the warm and dry patient is well compensated with no congestion at rest. Symptoms typically require minor medication adjustments and follow-up. The warm and wet patient is fluid overloaded and well perfused.

Physical Exam and ADHF Diagnosis

Diagnosis of ADHF begins with a brief history and focused physical exam. A brief history should focus on risk factors, signs or symptoms of myocardial ischemia, arrhythmia, any potential infectious processes and, as appropriate, PE.[21] Questions for the patient or family should include salt intake, medication compliance, and use of alcohol or drugs.

After addressing the airway, breathing, and circulation (ABCs) in treatment, the physical exam should focus on heart and lung sounds, especially murmurs that could indicate valve insufficiency or outflow tract obstruction. Such lesions warrant avoidance of aggressive therapies aimed at prompt reduction of preload and afterload.

Diagnosis of ADHF: How Good is the History and Physical Exam

Auscultation in the ED can be challenging, but should focus on the identification of heart sounds, murmurs, and the presence of rales. Rales may be heard in both lungs and represent the physical finding of pulmonary congestion.

A third heart sound, if present, may be difficult to auscultate

Table 5. Sensitivity of History and Physical Findings for an ADHF Diagnosis

VARIABLE	SENSITIVITY	SPECIFICITY	ACCURACY
Hx of HF	62	94	80
Dyspnea	56	53	54
Orthopnea	47	88	72
Rale	56	80	70
S3	20	99	66
JVD	39	94	72
Edema	67	68	68

Table 6. Findings of Physical Examination

	SYSTOLIC DYSFUNCTION	DIASTOLIC DYSFUNCTION
Third heart sound	Frequent	Rare
Fourth heart sound	Rare	Frequent
Rales	Occasional	Occasional
Peripheral edema	Frequent	Rare
Jugular venous distention	Frequent	Rare
Cardiomegaly	Usual	Rare

Modified from Braunwald E. Heart disease. Philadelphia; WB Saunders, 1997.

in the ED setting. It is due to decreased compliance of the left ventricle and most commonly is heard in patients with volume overload and tachycardia. An S_3, when detected, is specific for elevated left ventricular pressure,[22] low ejection fraction,[23] and worse outcomes.[24-26] Pulsus alternans may be present in severe cases and if identified is virtually diagnostic of severe advanced heart failure.[27] (*See Tables 5 and 6.*)

Hepatojugular reflux can occur in patients with mild heart failure and is manifested as JVD while compressing the liver. Right-sided heart failure leads to raised systemic venous pressure, resulting in JVD, peripheral edema, and ascites. JVD may be the most important exam finding for estimating volume status.[2]

Look for peripheral edema to accumulate in dependent parts of the body such as feet, ankles, and the pretibial regions of legs in ambulatory people. Presacral edema is more common in non-ambulatory patients. Ascites is a result of elevated pressure in hepatic veins and transudation of fluid into the peritoneal cavity. Keep in mind that misdiagnoses can be attributed to obesity, deconditioning, or another dyspneic condition. When limited to using only the history and physical exam, the diagnosis of HF frequently is in error.

Ancillary Testing

Objective tools can improve diagnostic accuracy. A right heart catheterization can yield great diagnostic value, but is not routinely performed in the ED. Echocardiography, the gold standard to measure ejection fraction and wall motion, also is not readily available, but reports of previous echocardiograms (ECGs) may be found in prior records.

Table 7. Chest Radiograph

	Sensitivity ↑ PRELOAD (%)	Sensitivity ↓ EF (%)	INTEROBSERVER AGREEMENT
Cardiomegaly	44	51	Moderate
Redistribution	65	37	Fair to moderate
Intensified edema	45	18	Moderate to almost perfect

Chest Radiography. A chest x-ray (CXR) should be obtained in all suspected ADHF patients but use caution in the interpretation. The key findings on the CXR indicative of HF are cardiomegaly and evidence of redistribution (cephalization) but, due to limited sensitivity, these findings alone cannot exclude or confirm HF or elevated filling pressure. Though incapable of excluding LV dysfunction, the CXR can help confirm other diagnoses, such as pneumonia. The key findings on the CXR indicative of HF are (in order of descending frequency): dilated upper lobe vessels, cardiomegaly, interstitial edema, enlarged pulmonary artery, pleural effusion, alveolar edema, prominent superior vena cava, and Kerley lines. The ED physician often encounters chronic HF patients in which the CXR does not identify patients with high pulmonary capillary wedge pressure (PCWP).[28]

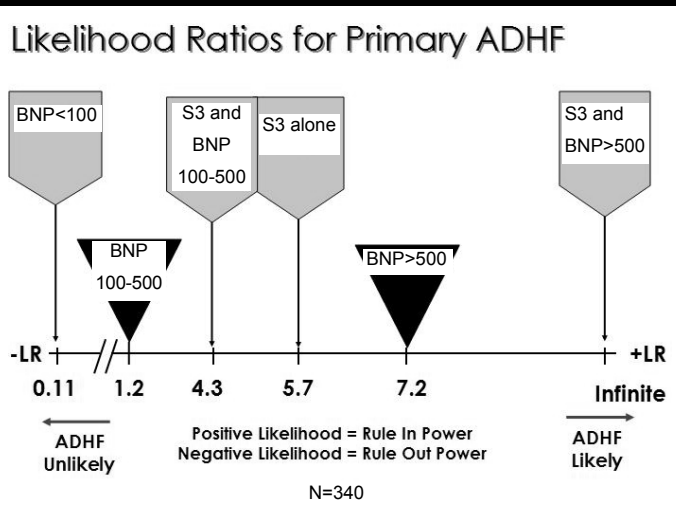
Cardiomegaly can suggest an HF diagnosis, and a cardiothoracic ratio greater than 60% correlates with increased five-year mortality.[28] However, the CXR has poor sensitivity for cardiomegaly (20% of cardiomegaly seen on echocardiography is missed on CXR).[29] Pleural effusions are common in HF but often missed on CXR. A portable CXR done with the patient in the supine position further degrades the diagnostic accuracy to detect pleural effusions. Detection of pleural effusion if supine is 67% sensitive, 70% specific, and 67% accurate, as demonstrated in one study of 34 patients with pleural effusions proven by decubitus CXRs.[30]

Sensitivity is poor for HF findings with a portable radiograph. (*See Table 7.*) In cases of mild HF, only dilated upper lobe vessels were found in greater than 60% of patients.[3] With severe HF, CXR findings occurred in at least 66% of patients, thus the frequency of CXR findings that confirm HF increases with severity of HF.

Electrocardiography. Though of limited diagnostic value, an ECG should be performed on every patient who presents with dyspnea and suspected HF. The ECG is a useful diagnostic tool in the assessment of HF patients because it can identify causal abnormalities, such as acute MI and acute ischemia, both of which would require CCU admission as well as a specific approach to treatment. An ECG also can assess cardiac rhythm, identify possible electrolyte disturbance (e.g., hyperkalemia), and may indicate a potential drug toxicity (e.g., junctional bradycardia due to digoxin toxicity).

The ECG also offers prognostic information. In dilated cardiomyopathy, the presence of abnormal Q waves, QRS duration greater than 0.12 ms, or left bundle-branch-block predict an increased five-year mortality rate.[31] The resting ECG is relatively

Figure 4. Sensitivity, Specificity, and +LR for an S3 in Predicting HF in Challenging Subgroups



insensitive and nonspecific in identifying severe underlying coronary disease (e.g., presence or absence of old Q waves).

A period of cardiac monitoring is essential for patients with HF. Continuous monitoring may be indicated by the clinical presentation (e.g., recent palpitations or near-syncope). With aggressive treatment such as repetitive diuretic dosing, an electrolyte abnormality may increase the possibility of arrhythmias. Initially, ECG monitoring may exclude cardiac arrhythmias as the cause of the current exacerbation.[31-33]

Heart Failure and Digital Heart Sound Detection for Rapid Diagnosis and Initial Treatment

Phonocardiography is the measurement of heart sounds and can be used to help detect an S_3 or an S_4 that easily can be missed in the noisy ED environment. The third (S_3) heart sound may be a normal finding in patients younger than 40 years, but in the patient at risk for HF, the S_3 is 93-99% specific for positive diagnosis of ADHF. The presence of an S_3 is highly correlated to LV systolic dysfunction, reduced ejection fraction, and elevated LV end diastolic pressure. In acute myocardial infarction (AMI), S_3 usually indicates concomitant HF. In dialysis patients, S_3 usually indicates fluid overload and elevated left ventricular end diastolic pressure (LVEDP). A third heart sound and JVD together carry a poor prognosis. Figure 4 illustrates likelihood ratios for primary ADHF.

The fourth (S_4) heart sound is an uncommon finding in young healthy subjects, but is nonspecific in the elderly, which is the population with HF. This highly correlates to LV dysfunction, elevated LVEDP and reduced LV compliance. An S_4 may be present in ADHF but may be present in other conditions, such as valvular disorders, LVH, and hypertrophic obstructive cardiomyopathy. New devices allow digital detection of extra heart sounds, taken at the same time as the ECG via two acoustical sensors in the V_3/V_4 positions. Algorithms extract ECG and acoustical information simultaneously.

Laboratory Testing. Asymptomatic cardiac ischemia can precipitate acute HF or produce decompensation in previously stable HF patients. Troponin and creatinine kinase isoenzyme levels are predictors of adverse prognosis and risk in patients with HF.[8,9] Because of their kinetics, a positive result may diagnose cardiac ischemia, but a negative result may not exclude it. As compared to AMI in non-HF patients, elevated cardiac necrosis markers in HF may not necessarily represent epicardial coronary artery occlusion.[34] However, cardiac marker elevation is associated with an increased risk of adverse outcome.[35-40] Perna et al. concluded that a cardiac troponin T (cTnT) level of 0.1 ng/mL or greater was associated with poor long-term survival and emerged as a powerful independent predictor of mortality in patients with acute cardiogenic pulmonary edema.[41]

Additional laboratory tests to consider include serum electrolytes. Serum magnesium levels should be ordered when either arrhythmias or severe hypokalemia is present.

Both sodium and potassium can be measurably altered by the activation of the RAAS or by diuretic treatment. Low serum sodium is not uncommon and is due to dilutional hyponatremia from expansion of extracellular fluid volume and correlates with a worse prognosis. The same can be said for worsening renal function. Elevated creatinine and liver function tests also are predictors of poor outcome. Inflammatory markers (e.g., C-reactive protein) appear to be elevated in CHF patients; however, their prognostic role requires further study.[32]

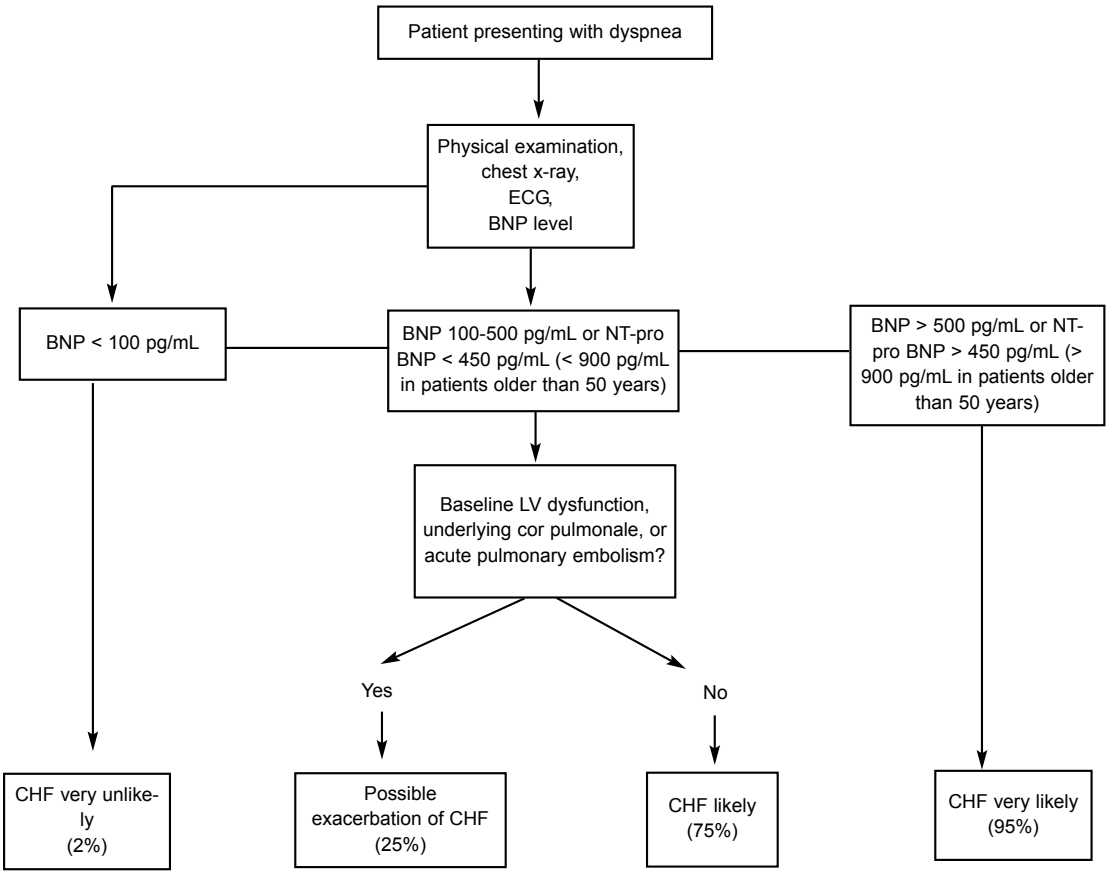
Natriuretic Peptides as a Diagnostic Tool

Brain-type natriuretic peptide levels correlate well with function and clinical presentation. In a study examining patients with and without heart failure, mean BNP levels were 38 in normal patients and 1076 pg/mL in those with heart failure.[42] The extent of the BNP elevation also corresponds well to functional class. The NYHA classification is used to estimate severity of heart failure, with higher scores being directly proportional to increased mortality. When heart failure is graded by NYHA class, the BNP levels vary directly with severity.[43,44]

Endogenous BNP has a half-life of only 22 minutes.[42] In comparison NT–pro BNP has a half-life of 1-2 hours. In the ED, BNP or NT-pro BNP can be used to diagnose heart failure. It is most helpful in those patients that are difficult to diagnose, such as in the combination of chronic obstructive pulmonary disease (COPD) and heart failure. In dyspnea resulting from isolated COPD, BNP generally is less than 100 pg/mL as compared to those whose symptoms are the result of heart failure where levels may exceed 1000 pg/mL.[42]

BNP is able to predict clinical events in patients presenting to the emergency department. Levels greater than 480 pg/mL predicted a markedly increased risk of death or re-hospitalization from heart failure in the following one to six months, as compared to those patients with levels less than 230 pg/mL.[43] Mortality also is predicted by BNP. Mortality in patients with BNPs greater than 73 pg/mL is markedly increased in the next 10-12 months, as compared to those with lower BNPs. Research indicates that admission status also correlates with BNP levels. In

Figure 5. Algorithm for Using BNP Levels in the ED to Diagnose CHF[37,38]



one study, heart failure patients required hospital admission with BNP levels greater than 700 pg/mL, while those able to be treated as an outpatients had BNPs less than 254 pg/mL.[42-44]

Figure 5 shows the recommended cutpoints of BNP as well as the general BNP reference guidelines. Current (BNP consensus panel)[45] recommendations suggest the laboratory should perform BNP testing on a continuous 24-hour basis with a turn-around time of 60 minutes or less. If the BNP is less than 100 pg/mL, then HF is highly unlikely (negative predictive value is 90%). If the BNP is greater than 500 pg/mL, then HF is highly likely (PPV = 90%). If the BNP is 100-500 pg/mL, consider that the baseline BNP may be elevated due to a stable underlying dysfunction, which could include right ventricular failure from core pulmonale, acute pulmonary embolism, or renal failure. Brain-type natriuretic peptide also rises with age and is affected by gender, comorbidity, and drug use, and should not be used as the sole diagnostic tool in assessing patients for heart failure. It should be considered in addition to the clinical context presented by the patient history and physical examination, chest x-ray, ECG, and other laboratory assessments.

A number of clinical scenarios can alter BNP levels. BNP is altered with chronic renal insufficiency. As chronic kidney disease advances, a higher BNP cut-off point may be required. An upper limit of normal of approximately 200 pg/mL is reasonable for those with an estimated glomular filtration rate (GFR) less than 60 mL /min,[42] In other non-heart failure cardiopulmonary diseases, BNP values may be increased. In approximately 20% of patients with pulmonary disease, BNP is elevated, which implies combined heart failure and lung disease, or cor pulmonale. In the setting of pulmonary embolism, BNP is elevated in one-third of cases and is associated with right ventricular pressure overload and higher mortality. Brain-type natriuretic peptide is not diagnostic for acute PE, however, and BNP levels can range from 100 to 500 pg/mL in cases of pulmonary hypertension and right ventricular pressure volume overload. Finally, BNP has been studied as a marker in patients with acute coronary syndromes. When used together, BNP and cardiac troponin levels provided a more effective tool for identifying patients at an increased risk for clinically important cardiac events related to acute coronary syndrome (ACS) and HF.[46-48]

There are situations when BNP levels are lower than expected clinically. In cases of preserved systolic function heart failure or diastolic myocardial dysfunction, BNP may be found to be approximately half as high as when compared to cases of systolic dysfunction. Therefore, BNP may or may not detect patients with diastolic dysfunction. Obese patients tend to have lower levels of BNP when measured for any given severity of heart failure. Physiologically, adipose tissue is related to the natriuretic clearance receptor, and obesity can interfere with the usual diagnostic approach to heart failure. In a study by Mehra, nearly 40% of obese patients had BNPs less than 100 pg/mL.[49] Lastly, unexpectedly low levels can occur during the first one to two hours of

Table 8. Special Circumstances in Interpreting BNP Levels

FACTORS OTHER THAN CHF THAT CAN ACCOUNT FOR HIGH BNP LEVELS

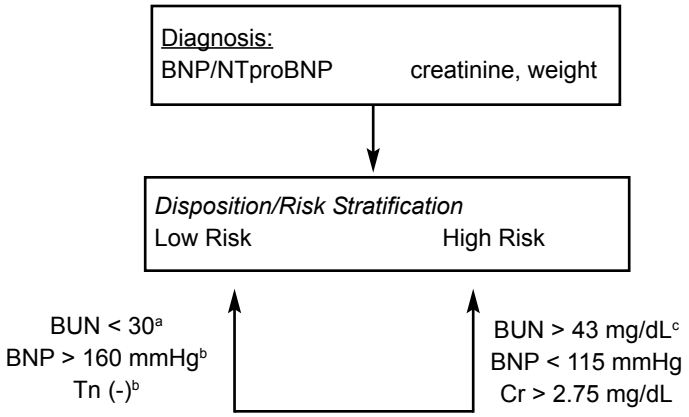
- Advanced age
- Renal failure
- Myocardial infarction
- Acute coronary syndrome
- Lung disease with right-sided failure
- Acute large pulmonary embolism
- High-output states (e.g., cirrhosis)

FACTORS THAT CAN ACCOUNT FOR LOWER-THAN-EXPECTED BNP LEVELS WHEN CHF IS PRESENT

- Flash pulmonary edema
- Obesity
- CHF secondary to causes upstream from the left ventricle
  - Acute mitral regurgitation
  - Mitral stenosis
  - Atrial myxoma
- Stable NYHA class I patients with low ejection fractions

Key:
BNP = B-type natriuretic peptide; CHF = congestive heart failure; NYHA = New York Heart Association.

Figure 6. Risk Stratification of Emergency Department Patients with Acute Dyspnea



a) Burkardt, *AJEM*, 2005; b) Diercks, *HFSA*, 2004;c) Fonorow, *JAMA*, 2005

flash pulmonary edema.[50,51] Table 8 illustrates special circumstances in interpreting BNP levels. Figure 5 illustrates an algorithm for using BNP levels in the ED to diagnose ADHF. There is less literature on the use of NT-pro BNP. Elevation of NT-pro BNP above 450 pg/mL in patients younger than 50 years, or elevations above 900 pg/mL in patients older than 50 years are sensitive and specific for the diagnosis of HF.[52]

Risk Stratification

This is a process well known in the setting of suspected acute coronary syndrome but is less well defined for ADHF. Risk stratification for ADHF can be divided into low- and high-risk predictors for adverse outcomes. Patients with a low risk of adverse outcomes can be considered for possible 23-hour observation admission. Longer hospitalization and aggressive therapy would be warranted for patients at high risk for an adverse event.

The low-risk population was evaluated in two studies. One study[53] evaluated predictors of successful therapy in nearly 500 patients. Low risk was defined as discharge within 24 hours, and no death or rehospitalization within 30 days. Parameters predicting low risk include an initial negative troponin and systolic blood pressure (SBP) greater than 160 mmHg. Burkhardt[54] used similar methods and was able to identify predictors of short stay failure. A measured BUN greater than 30 mg/dL at ED presentation was associated with an increased probability of hospitalization in excess of 24 hours and, therefore, identified patients inappropriate for observation unit therapy.

A high-risk population also can be identified. An analysis of more than 80,000 patients from the ADHERE registry revealed a BUN exceeding 43 mg/dL to be the single greatest acute mortality predictor.[55] The mortality risk of nearly 10% compared to only 2.8% if the BUN was less than 43 mg/dL. The next most powerful mortality predictor was an SBP below 115 mmHg. Combined with a high BUN, mortality increased to approximately 15%. If serum creatinine is also more than 2.75 mg/dL, in-hospital mortality is greater than 22%. (*See Figure 6.*)

These parameters help identify patients appropriate for a 23-hour observation unit vs. those requiring more aggressive care and admission to a more critical bed. Patients directed to an observation unit can receive rapid emergency care for HF symptoms with close follow-up arranged at the time of discharge.

References

1. Peacock WF, Young J, Collins S, et al. Heart failure observation units: Optimizing care. Cardiology/Review Article. *Ann Emerg Med* 2006;47:22-33.
2. Peacock WF, Tiffany B. Cardiac Emergencies. *Congestive Heart Failure: Demographics and Economics*. The McGraw-Hill Companies, Inc. 2006;297.
3. Peacock WF. Heart failure in the emergency department. *Critical Pathways in Emergency Medicine*: *A journal of evidence-based medicine*. 2003;2:207-220.
4. Ansari M, Massie BM. Heart failure: How big is the problem? Who are the patients? What does the future hold? *Am Heart J* 2003;146:1-4.
5. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction). *Circulation* 2004;110:588.
6. Summers RL. Disease management of acute decompensated heart failure: The ADHERE emergency medicine module. Emergency diagnosis and treatment of ADHF. CME Monograph from ACEP 2005 Spring Congress Satellite Symposium. Mar 4, 2005; 33-38.

7. Peacock WF, Emerman CL. Preventing readmission in heart failure. *Emerg Med* October 2003. Available at: http://www.emedmag.com/html/pre/cov/covers/101503.asp. Accessed March 16, 2005.
8. Freda BJ, Peacock WF, Lindwell CJ, et al. Outcomes in heart failure patients with elevated cardiac markers of ischemia [abstract]. *J Card Fail* 2002; 8(4suppl):570.
9. Peacock WF. Acute emergency department management of heart failure. *Heart Fail Rev* 2003;8:335-338.
10. Dao Q, Krishnaswamy P, Kazanegra R, et al. Utility of B-type natriuretic peptide in the diagosis of congestive heart failure in an urgent care setting. *J Am Coll Cardiol* 2001;37:379-385.
11. Maisel AS, Krishnaswamy P, Nowak RM, et al. Breathing Not Properly Multinational Study Investigators: Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. *N Engl J Med* 2003;347: 161-167.
12. Hut SA, Baker DW, Chin MH, et al. ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult; executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (committee to revise the 1995 Guidelines for the Evaluation and Management of Heart Failure). *J Am Coll Cardiol* 2001;38:2101-2113.
13. Dei Cas L, Metra M, Nodaic S, et al. Prevention and management of heart failure in patients at risk. *Am J Cardiol* 2003;91(suppl 9A):10F-17F.
14. Potturi S, Ventura HO, Mulumundi M, et al. Cardiac troponin levels in heart failure. *Cardiology in Review* 2004;12:21-25.
15. Black HR. The burden of cardiovascular disease: Following the link from hypertension to myocardial infarction and heart failure. *Am J Hypertens* 2003;16(suppl 1):4S-6S.
16. Sharis PJ, Connor CP. *Evidence Based Cardiology*. Philadelphia: Lippincott Williams and Wilkins; 2003:290-291.
17. Maisel AS, Cairns CB, Kosowsky JM. Urgent management of acute decompensated heart failure: The role of the emergency physician. *Clinician* Vol.23, No. 2.
18. de Groote P, et al. Right ventricular ejection fraction is an independent predictor of survival in patients with moderate heart failure. *J Am Coll Cardiol* 1998;32:948-954.
19. Missov E, Mais J. A novel biochemical approach to congestive heart failure: Cardiac troponin T. *Am Heart J* 1999;138:95-99.
20. Glover DR, Littler WA. Factors influencing survival and mode of death in severe chronic ischemic cardiac failure. *Br Heart J* 1987;57:125-132.
21. Rame JE, Dries DL, Drazner MH. The prognostic value of the physical examination in patients with chronic heart failure. *Congest Heart Fail* 2003;9:170-175,178.
22. Patel R, Bushnell DL, Sobotka PA. Implications of an audible third heart sound I evaluation cardiac function. *West J Med* 1993;158:606-609.
23. Pina IL. The epidemiology, pathophysiology, and diagnosis of heart failure. *Cardiology Review* 2005;(22, suppl 8):4-5.
24. Drazner MH, Rome JE, Stevenson LW, et al. Prognostic importance of elevated jugular venous pressure and a third heart sound in patients with heart failure. *N Engl J Med* 2001;345:574-587.
25. Peacock WF, Freda BJ. The clinical challenge of heart failure: Comprehensive evidence-based management of the hospitalized patient with acute myocardial decompensation—diagnosis, risk stratification, and outcome effective treatment. *Emerg Med Report* 2002;23:81-92.
26. Spodick D, Quary V. Prevalence of the fourth heart sound by phonocardiography in the absence of cardiac disease. *Am Heart J* 1974;87:11-14.
27. Lee YC, Sutton FJ. Pulsus alternans in patients with congestive cardiomyopathy. *Circulation* 1982;65:1533-1534.
28. Chakko S, Woska D, Marinez H, et al. Clinical, radiographic, and hemodynamic correlations in chronic congestive heart failure: Conflicting results may lead to inappropriate care. *Am J Med* 1991;90:353-359.
29. Kono T, Suwa M, Ngnada H, et al. Clinical significance of normal cardiac silhouette in dilated cardiomyopathy—evaluation based upon echocardiography and MRI. *Japanese Circulation Journal* 1992;56:359-365.
30. Ruskin RA, Gurney JW, Thorsen MK, et al. Detection of pleural effusions on supine chest radiographs. *AJR Am J Roentgenol* 1987;148:681-683.
31. Koga Y, Wada T, Toshima H, et al. Prognostic significance of ECG findings in patients with dilated cardiomyopathy. *Heart Ves* 1993;8:37-41.
32. DeFerrari GM, Tavazzi L. The role of arrhythmias in the progression of heart failure. *Eur J Heart Fail* 1999;1:33-40.
33. Mathew J, Hunsberger S, Fleg J, et al. Incidence, predictive factors, and prognostic significance of supraventricular tachyarrhythmia in congestive heart failure. *Chest* 2000;118:914-922.
34. Del Carlo CH, O'Connor CM. Cardiac troponins in congestive heart failure. *Am Heart J* 1999;138:646-653.
35. Sato Y, Kita T, Takatsu Y, et al. Biochemical markers of myocyte injury in heart failure. *Heart* 2004;90:1110-1113.
36. Missou E, Calzolari C, Pau B. Circulating cardiac troponin I in severe congestive heart failure. *Circulation* 1997;96:2953-2958.
37. Setsuta K, Seino Y, Takahashi N, et al. Clinical significance of elevated levels of cardiac troponin T in patients with chronic heart failure. *Am J Cardiol* 1997; 84:608-611.
38. LaVecchia L, Mezzena G, Onetto R, et al. Detectable serum troponin I in patients with heart failure or non-myocardial ischemic origin. *Am J Cardiol* 1997;80:88-89.
39. Healey JS, Smith ST, Davies RF, et al. Prognostic value of serum troponin T and troponin I in patients with heart failure. *J Card Fail* 2000;6:234.
40. Perna ER, Cimbaro Canella JP, Macin SM, et al. Combined determination of natriuretic peptides and necrosis markers in decompensated heart failure: Should NT-pro BNP and troponin T be measured on admission, predischarge, or both? *J Card Fail* 2004;10:S102-S103.
41. Perna ER, Macin SM, Parras JI, et al. Cardiac troponin T levels are associated with poor short- and long-term prognosis in patients with acute cardiogenic pulmonary edema. *Am Heart J* 2002;143:814-820.
42. Peacock WF, Tallman TA. Critical Decisions in Emergency Medicine: B-Type Natriuretic Assay. 2006;20:13-22.
43. Silver MA, Maisel A, Yancy CW, et al. BNP Consensus Panel 2004; A Clinical Approach for the Diagnostic, Prognostic, Screening, Treatment Monitoring, and Therapeutic Roles of Natriuretic Peptides in Cardiovascular Diseases. *CHF* 2004;10:53, 1-36.
44. Mueller C, Scholer A, Laule-Killian K, et al. Use of B-type natriuretic peptide in the evaluation and management of acute dyspnea. *N Engl J Med* 2004;350:647-654.
45. Peacock WF. The evolving role of BNP in the diagnosis and treatment of CHF: A summary of the BNP Consensus Panel Report. Emergency Diagnosis and Treatment of ADHF. CME Monograph from ACEP 2005 Spring Congress: 16-26.
46. Kikenta K, Yasue H, Voshimura M, et al. Increased plasma levels of B-type naturetic peptide in patients with unstable angina. *Am Heart J* 1996;132: 101-107.

47. Roongsritong C, Warraich I, Bradley C. Common causes of troponin elevations in the absence of scute myocardial infarction. *Chest* 2004;125: 1877-1884.
48. Velazquez EJ, Pfeffer MA. Acute heart failure complicating acute coronary syndromes. A deadly intersection. *Circulation* 2004;109:440-442.
49. Mehra MR, Uber PA, Park M, et al. Obesity and suppressed B-type natriuretic peptide levels in heart failure. *J Am Coll Cardiol* 2004;43:1590-1595.
50. Silver MA, Maisel A, Yancy CW, et al. BNP Consensus Panel 2004: A clinical approach for the diagnostic, prognostic, screening, treatment, monitoring, and therapeutic roles of natriuretic peptides in cardiovascular diseases. *Congest Heart Fail* 2004;10:1-30.
51. Mueller C, Scholer A, Laule-Killian K, et al. Use of B-type natriuretic peptide in the evaluation and management of acute dyspnea. *N Engl J Med* 2004;350:347-654.
52. Januzza JL Jr, Camargo CA, Anwaruddin S, et al. The N terminal pro BNP investigation of dyspnea in the emergency department (PRIDE) study. *Am J Cardiol* 2005;95:948-954.
53. Dierck DB, Hisk JD, Peacock WF, et al. Identification of emergency department patients with decompensated heart failure at low risk for adverse events and prolonged hospitalization. *J Cardiac Fail* 2004;(Suppl) 10:5118.
54. Burkhardt J, Peacock WF, Emerman CL. Elevation in BUN predicts a lower discharge rate from the observation unit. *Ann Emerg Med* 2004;(Suppl)44(4):599.
55. Fonarow GC, Adams KF Jr, Abraham WT, et al. Risk statification for in-hospital mortality in acutely decompensated heart failure: Classification and regression tree analysis. *JAMA* 2005;293:572-580.

Physician CME Questions

11. A comparison of patients enrolled in ADHF randomized trials vs. ADHERE registry patients reveals that, in the registry, the average patient is:
    A. much younger.
    B. more often burdened by coexistent disease.
    C. more often male.
    D. none of the above.

12. Considering the etiologies for cardiogenic pulmonary edema, all of the following are considered causes of diastolic dysfunction *except*:
    A. left ventricle hypertrophy.
    B. acute hypertensive crisis.
    C. concurrent systolic dysfunction.
    D. mitral stenosis.

13. Which of the following statements regarding B-type natriuretic peptide (BNP) is *false*?
    A. BNP is released in response to increased ventricular stretch.
    B. Both BNP and NT-pro BNP are helpful in confirming a diagnosis of HF.
    C. Physiologically, BNP and NT-pro BNP are very similar to each other.
    D. Only BNP is available as a point of care testing platform.

14. Which of the following statements regarding the New York Heart Association classification is true?
    A. The NYHA class is directly proportional to survival.
    B. There is no direct relationship between NYHA and ejection fraction.
    C. The NYHA does suffer from interobserver variability.
    D. All of the above are true.

15. Risk stratification for ADHF can be divided into low- and high-risk predictors for adverse outcomes. A high-risk population of heart failure patients can be identified based on which of the following mortality predictors ?
    A. BUN < 30
    B. BUN > 43
    C. Systolic BP > 160
    D. Negative troponin

16. Phonocardiography is a measurement of heart sounds and can be used to help detect an S_3 or an S_4 that can be missed in the noisy environment of the ED. All of the following statements regarding S_3 are true *except*:
    A. S_3 may be a normal finding in ages < 40.
    B. The presence of an S_3 is highly correlated to LV diastolic dysfunction.
    C. An S_3 and JVD together carry a poor prognosis.
    D. In an acute MI, an S_3 usually indicates concomitant heart failure.

17. Ancillary testing can improve diagnostic accuracy in HF patients. Which of the following statements is correct regarding ancillary tests?
    A. The chest x-ray is highly sensitive in detecting pleural effusion.
    B. The resting ECG is highly sensitive to underlying coronary artery disease.
    C. Cardiac marker elevation, including troponin T, is associated with an increased risk of adverse outcomes.
    D. BNP levels of > 50 pg/mL are highly sensitive for heart failure.

18. NT-pro BNP differs from BNP because:
    A. NT-proBNP has a much shorter half-life and returns to normal more quickly.
    B. NT-pro BNP is available as a bedside test.
    C. NT-pro BNP levels vary significantly with age.
    D. A level >100 pg/mL of either BNP or NT-pro BNP is diagnositic of heart failure.

19. The best physical sign for estimating volume status is:
    A. jugular venous distention.
    B. peripheral edema.
    C. rales.
    D. hepatomegaly.

20. Modest elevations of BNP may occur in all of the following *except*:
    A. infection.
    B. renal disease.
    C. acute coronary syndrome.
    D. pulmonary edema.

CME Answer Key

11. B; 12. D; 13. C; 14. D; 15. B; 16. B; 17. C; 18. C; 19. A; 20. A

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Acute Decompensated Heart Failure Part I

Etiology of Cardiogenic Pulmonary Edema

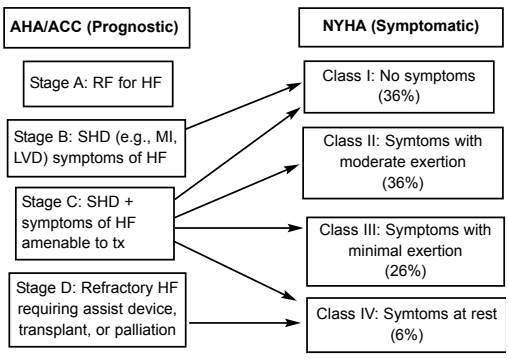
IMPAIRED LEFT ATRIAL OUTFLOW	SYSTOLIC DYSFUNCTION	DIASTOLIC DYSFUNCTION	LV VOLUME OVERLOAD
Elevated heart rate -V Tach	Coronary heart disease	LV hypertrophy	Ventricular septal rupture
-atrial fibrillation -exertion	Hypertension	Hypertrophic cardiomyopathy	Aortic insufficiency
-fever -stress	Valvular heart disease	Restrictive cardiomyopathy	Renal disease
Mitral stenosis	Idiopathic dilated cardiomyopathy	Ischemia	LV outflow obstruction
Atrial myxoma		Acute hypertensive crisis	Critical aortic stenosis
Prosthetic valve thrombus		Concurrent systolic dysfunction	Hypertrophic cardiomyopathy
Congenital defects		Reduced diastolic coronary perfusion	Severe systemic hypertension
Mitral insufficiency		Arrhythmia (Afib)	
Mitral regurg. Secondary to ischemic heart disease			

Randomized Controlled Trials vs. ED Patients

RANDOMIZED CONTROLLED TRIALS	
Age	50-60 years old
Sex	70-80% Men
Co-morbidities	Diabetes 20-25%
Renal insufficiency	Infrequent (mean Cr 1.1-1.3)
Ventricular function	75-80% Systolic Dysfunction (LVEF < 0.40)
PAC use	30-40%
In-hospital mortality	1.5-2.5%

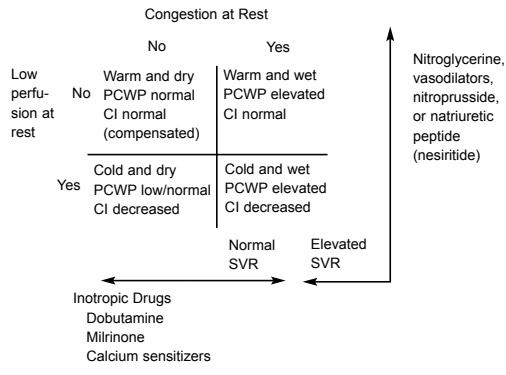
PATIENTS FROM ADHERE DATABASE	
Median age	75 years
Female gender	52%
Race White	72%
Black	20%
History of HF	75%
Ejection fraction < 40%	59%
Hospitalized within 6 months	23%
Chronic renal Insufficiency (creatinine > 1.5 mg/dL)	30%
Systolic BP > 90 mmHg at presentation	98%
Initial BNP	667 pg/mL

Heart Failure Classification Systems



Key: RF = risk factor; SHD = structural heart disease
Source: *Pharmacotherapy* 2003;23:997-1020

Hemodynamic Profile Assessment for Patient Selection and Treatment



Signs and Symptoms of Congestion
- Orthopnea/PND
- JVD
- Ascites
- Edema
- Rales

Possible Evidence of Low Perfusi
- Narrow pulse pressure
- Sleepy/obtunded
- Low serum sodium
- Cool extremities
- Hypertension with ACE inhibitor
- Renal dysfunction (one cause)

Physiologic Effects of Neurohormones

RAAS (RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM)	
Activation of AT_1 receptors by angiotensin	Vasoconstriction Sodium retention Increased aldosterone release Increased cellular growth Increased sympathetic nervous activity

NATRIURETIC PEPTIDE SYSTEM	
ANP, BNP	Vasodilation Sodium excretion Decreased aldosterone levels Inhibition of RAAS Inhibition of sympathetic nervous activity Antiproliferation of vascular smooth muscle cells

Key:
ANP = atrial natriuretic peptide; AT_1 = angiotensin I;
BNP = endogenous B-type natriuretic peptide

Reference: Burrell JC Jr. *J Hypertens* 1999;17(suppl 1):837-843.

Heart Failure Differential Diagnosis of Dyspneic States

- Chronic obstructive pulmonary disease exacerbation
- Asthma exacerbation
- Pulmonary embolus
- Acute myocardial infarction
- Physical deconditioning
- Obesity
- Pleural effusions
- Pneumonia/pulmonary infection
- Pneumothorax/tension pneumothorax
- Fluid retentive states
- Renal failure/nephritic syndrome
- Liver failure/cirrhosis
- Portal vein thrombosis
- Hypoproteinemia
- Deep venous thrombosis
- Dependent edema
- Low cardiac output states
- Pericardial tamponade
- Sepsis

Sensitivity of History and Physical Findings for an ADHF Diagnosis

VARIABLE	SENSITIVITY	SPECIFICITY	ACCURACY
Hx of HF	62	94	80
Dyspnea	56	53	54
Orthopnea	47	88	72
Rale	56	80	70
S3	20	99	66
JVD	39	94	72
Edema	67	68	68

Findings of Physical Examination

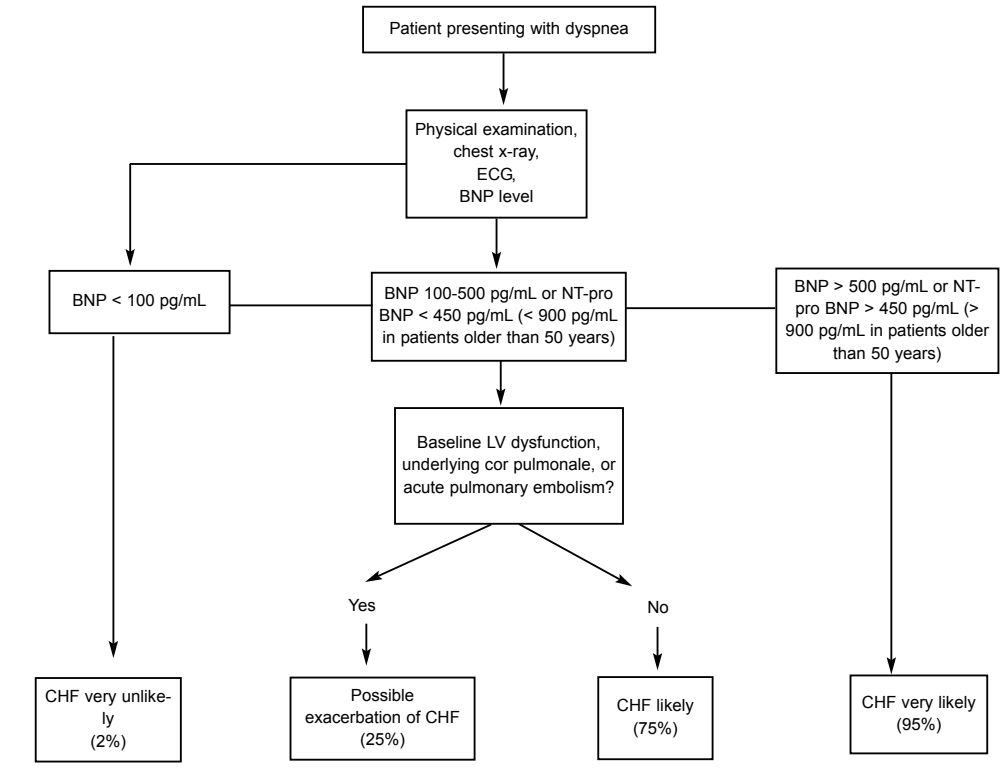
	SYSTOLIC DYSFUNCTION	DIASTOLIC DYSFUNCTION
Third heart sound	Frequent	Rare
Fourth heart sound	Rare	Frequent
Rales	Occasional	Occasional
Peripheral edema	Frequent	Rare
Jugular venous distention	Frequent	Rare
Cardiomegaly	Usual	Rare

Modified from Braunwald E. Heart disease. Philadelphia; WB Saunders, 1997.

Chest Radiograph

	↑ PRELOAD (%)	↓ EF (%)	INTEROBSERVER AGREEMENT
	Sensitivity		
Cardiomegaly	44	51	Moderate
Redistribution	65	37	Fair to moderate
Intensified edema	45	18	Moderate to almost perfect

Algorithm for Using BNP Levels in the ED to Diagnose CHF



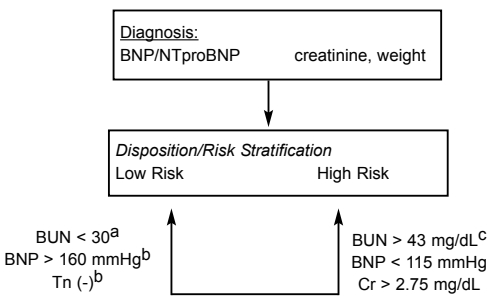
Pathophysiology

Acute LV systolic dysfunction
↓
Decreased myocardial contractility, ↓C.O.
↓
Catecholamine production
↓
Increased SVR (afterload) and ↑ blood pressure
↓
Increased myocardial wall tension and myocardial oxygen demand
↓
Myocardial ischemia
↓
Decreased myocardial contractility and C.O. (continue the cycle)
↓
Diastolic dysfunction, increased pulmonary artery and capillary hydrostatic pressures
↓
Transudation of fluid into pulmonary alveoli and interstitium
↓
Hypoxia
↓
Myocardial ischemia (continue the cycle)
↓
Increased anxiety
↓
Increased catecholamine production (continue the cycle)

As the left side of the heart fails, the right side continues to fill (preload). This ↑ greater pulmonary artery and capillary hydrostatic forces. (continue the cycle)

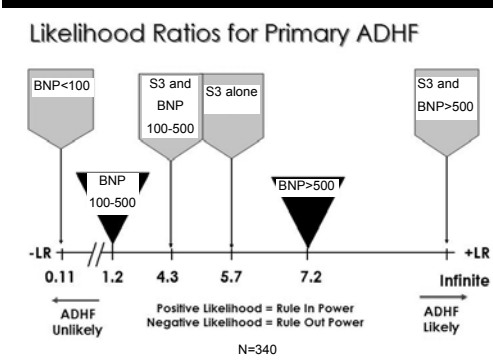
Adapted from: Mattu A. Heart Failure: Advances in Diagnosis and Treatment, ACEP Scientific Assembly 2005: 2.

Risk Stratification of Emergency Department Patients with Acute Dyspnea



a) Burkardt, *AJEM*, 2005; b) Diercks, *HFSA*, 2004;c) Fonorow, *JAMA*, 2005

Sensitivity, Specificity, and +LR for an S3 in Predicting HF in Challenging Subgroups



Special Circumstances in Interpreting BNP Levels

FACTORS OTHER THAN CHF THAT CAN ACCOUNT FOR HIGH BNP LEVELS

- Advanced age
- Renal failure
- Myocardial infarction
- Acute coronary syndrome
- Lung disease with right-sided failure
- Acute large pulmonary embolism
- High-output states (e.g., cirrhosis)

FACTORS THAT CAN ACCOUNT FOR LOWER-THAN-EXPECTED BNP LEVELS WHEN CHF IS PRESENT

- Flash pulmonary edema
- Obesity
- CHF secondary to causes upstream from the left ventricle
  - Acute mitral regurgitation
  - Mitral stenosis
  - Atrial myxoma
- Stable NYHA class I patients with low ejection fractions

Key:

BNP = B-type natriuretic peptide; CHF = congestive heart failure; NYHA = New York Heart Association.

Supplement to *Emergency Medicine Reports*, July 10, 2006: "Managing Acute Decompensated Heart Failure in the Emergency Department: Par I." *Authors:* **Thomas A. Tallman, DO, FACEP**, Associate Staff Physician, Department of Emergency Medicine, The Cleveland Clinic Foundation, Cleveland, OH; and **W. Frank Peacock, MD**, Director of Clinical Operations, Department of Emergency Medicine, The Cleveland Clinic Foundation, Cleveland, OH. *Emergency Medicine Reports' "Rapid Access Guidelines."* Copyright © 2006 Thomson American Health Consultants, Atlanta, GA. **Editor-in-Chief:** Gideon Bosker, MD. **Editors:** Sandra M. Schneider, MD, FACEP; and J. Stephan Stapczynski, MD. **Vice President and Group Publisher:** Brenda Mooney. **Editorial Group Head:** Glen Harris. **Specialty Editor:** Shelly Morrow Mark. For customer service, call: **1-800-688-2421.** This is an educational publication designed to present scientific information and opinion to health care professionals. It does not provide advice regarding medical diagnosis or treatment for any individual case. Not intended for use by the layman.

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Trauma Reports

Vol. 7, No. 4

Supplement to *Emergency Medicine Reports*, *Pediatric Emergency Medicine Reports*, *ED Management*, and *Practical Summaries in Acute Care*

July/ August 2006

Between 800,000 and 1,000,000 annual emergency department (ED) visits are for patients with head injury; 80% of these involve minor head injuries; 35% to 61% of patients seen in the ED undergo some form of radiographic evaluation.[1-4]

The use of computed tomography (CT) imaging specifically has seen exponential growth in the ED during the last 30 years. The first unit was installed at the Mayo Clinic in 1973; within only a few years, CT had virtually eliminated the use of pneumoencephalography and arteriography in head trauma.

In addition, the advent of CT scanning resulted in a reduction in the morbidity and mortality of head-injured patients when compared to historical controls studied by other methods.[5] This study also demonstrated a reduction in the number of skull radiographs, cerebral angiograms, and unnecessary surgical exploration. Early surgical intervention in head-injured patients with extra-axial hematomas was made possible with the advent of CT and was shown to improve morbidity and mortality in this population.[6,7]

Radiologic Evaluation of Head Trauma: Identifying the Spectrum of Injuries

Authors: **Howard A Werman, MD, FACEP**, Professor of Clinical Emergency Medicine, The Ohio State University College of Medicine and Public Health; Medical Director, MedFlight, Columbus, Ohio; **Martha Brogan, MD,** Neuroradiologist, Private Practice, Columbus, Ohio; **Robert Falcone, MD, FACS**, President, Grant Medical Center, Columbus, Ohio; Clinical Professor of Surgery, Ohio State University

Peer Reviewer: **Steven G. Rothrock, MD**, Professor of Emergency Medicine, Orlando Regional Medical Center, Orlando, Florida

As with all radiographic studies, an organized approach to interpretation of head CT imaging, as well as other cranial radiographic studies, is imperative. Missed radiographic findings account for 12% to 15% of all malpractice settlements.[8,9] Some studies have demonstrated that missed CT scan interpretations are significant among those without specific training in CT interpretation.[10,11] Fortunately, physicians with proper training have a high degree of accuracy in interpretation of radiographs when compared with that of radiologists.[12-18] The authors review indications for radiographic imaging, interpreting images, and specific injuries.

— The Editor

Basics Principles of Radiology

Traditional radiographs record images on film or computer, which convey information about the size, shape, and distribution of tissues within a patient. Radiographs are a two-dimensional representation of a series of x-ray beams projected through a

Statement of Financial Disclosure

In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, Dr. Dietrich (editor in chief) reports that she receives grant/research support from the National Institutes of Health and is the medical director for the Ohio Chapter of the American College of Emergency Physicians. Drs. Werman, Falcone, Brogan (authors), Ms. Finnerty (nurse reviewer), and Ms. Neff (nurse reviewer) report no relationships with companies related to this field of study. Dr. Rothrock reports that he owns stock in Gilead Sciences, Amgen, Oxigene, Medtronics, St. Jude, Pfizer, and IVAX.

three-dimensional object. The image represents a combination of black, gray, and white shades that are determined by the absorption and scattering of x-rays by each tissue through which the beam is projected. Tissues that possess little radiodensity (e.g., air or fat) allow the beam to pass easily through the body with little scatter or absorption, exposing the radiographic film and producing a black image. Very radiodense tissues (e.g., metal, calcium, or bone) obstruct the beam and produce a white image. Each radiographic plate is coated with chemicals that emit a fluorescent flash when struck by the beam, thus augmenting the actual effect of the x-ray beam. More recently, the photographic plate has been modified so that the image can be digitized and converted to a computer image. Most other tissues, especially those containing water, produce a gray image. The x-ray beam will pass through many tissues en route to the film, and the final shading will reflect the summation of all tissues through which the beam has traversed.

Plain radiographs are described in terms of the path that the beam takes from the x-ray tube to the film. For example, a plain radiograph of the skull shot from front to back is an anterior to posterior (AP) projection. If directed from one side through to the other, the radiograph is a lateral view. The oblique view is obtained by directing the beam obliquely.

In CT, images represent the same spectrum of radiographic densities as plain radiography but repeated dynamic imaging allows reconstruction of the body part in the axial plain. Newer scanners allow coronal, sagittal, and even three-dimensional reconstructions. Despite the advances in CT technology, the average cost is approximately $125 per study.[19]

Trauma Reports™ (ISSN 1531-1082) is published bimonthly by Thomson American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
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POSTMASTER: Send address changes to ***Trauma Reports***, P.O. Box 740059, Atlanta, GA 30374.

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The basic components of the CT scanner are the x-ray tube, detectors, and computers that allow for image reconstruction. Repeated slices are constructed as the beam rotates 360° around the patient while the table advances. Each CT image generated represents a matrix of picture elements or pixels. Each pixel is assigned an attenuation coefficient based upon its ability to absorb or scatter x-rays. This attenuation coefficient is expressed in Hounsfield units (HU), which vary from -1000 U (air) to +1000 (cortical bone). CT images can be viewed in a variety of 'windows' that accentuate tissues of a specific attenuation. In most trauma head CT protocols, 5-mm thick cuts are performed through the posterior fossa; 10-mm thick cuts are taken in the supratentorial region and will be displayed in brain, bone, and blood windows.

Newer high resolution scanners have thinner cuts, achieving submillimeter thickness. More recently, faster helical or spiral CT units that move the patient through the scanner in a continuous fashion and scan in a helical pattern around the patient have been introduced, reducing the scanning time to 20 to 30 seconds. This is particularly useful for imaging anatomy that is constantly in motion such as the chest.

Magnetic resonance imaging (MRI) uses computer technology to convert radiofrequency signals into shades of black, gray, and white. Instead of x-ray beams, an external magnetic field and radiofrequency waves are used to produce the image. The basic principle of MRI is the detection of spinning protons. When the patient is placed in the bore of the magnet, the magnetic fields of the protons align with the stronger magnetic field of the scanner. Radio waves are then intermittently and briefly applied to the tissue being examined to enhance the spin frequency by causing the protons to resonate (Larmor frequency) at a higher energy level. As the tissues return to their resting state, a radiofrequency wave is emitted and then detected by the receiving coil. The computer then converts the intensity of the detected signal to shades of black, gray, or white. The result is a three-dimensional plot of proton densities.

As noted, a signal is detected as the protons return to their resting state. This relaxation phase occurs by two major pathways: longitudinal or T1 decay, and transverse or T2 decay. Images generated from signals in the early portion of the relaxation phase (called T1-weighted sequence) produce a unique image in which fat appears as white and gray image contrast is excellent. During late relaxation (T2-weighted sequence), water appears white and fat appears gray. The T1-weighted sequence and T2-weighted sequence provide two perspectives on the same image. T1 images provide more useful architectural information; T2 images depict pathology more accurately because water (high in hydrogen protons) accumulates in many pathologic conditions (e.g., ischemia, edema, and tumor). In addition to producing images of high quality, MRI does not rely on radiation and thus, does not carry the biologic risk of x-rays.

Iodinated contrast agents used in x-ray-generated images are not useful for MR studies. Instead, paramagnetic agents such as gadolinium are used to enhance the image. Gadolinium acts by reducing T1 relaxation times and thus, improving the contrast

Table 1. High-Risk Criteria for Minor Head Injury [21]

- Glascow coma scale score < 15 after 2 hours
- Suspected open or depressed skull fracture
- Any sign of basilar skull fracture
- Vomiting ≥ 2 episodes
- Age ≥ 65 years

between tissues. It is most useful for delineating tumors and infections.

Indications for Radiographic Imaging in Head Trauma

CT scanning has virtually replaced plain skull radiographs in imaging the patient with head injury. Some physicians still will obtain plain radiographs in patients with suspected depressed skull fractures or with metallic foreign bodies. Plain radiography had previous utility in determining the presence of linear skull fractures. While 66% of patients with severe head trauma have an associated skull fracture, only 25% to 35% of patients with skull fractures have underlying cranial pathology.[20] The primary indications for obtaining skull plain films are to evaluate nonaccidental trauma (i.e., to look for fractures in different stages of healing), to assess ventricular shunt integrity, and as an adjunct to CT in penetrating skull trauma. It has been shown that the number of radiographs ordered for medicolegal considerations varies from 10% to 46%.[21,22]

Although MRI has the advantage of producing images with superior anatomic detail, CT provides several important advantages in the setting of trauma. The more rapid study completion time and greater availability of CT imaging makes it a far more useful study. Additionally, CT is as reliable as MRI in demonstrating intracranial lesions requiring operative intervention. Finally, CT has none of the stringent safety requirements of MRI imposed by the strong magnetic fields surrounding the scanner. MRI has a prominent role, however, in evaluating patients with subacute and chronic injuries. In these patients, MRI offers better discrimination between lesions and normal brain tissue, whereas subacute hemorrhage may appear isodense with normal brain tissue on CT.

Patients with altered level of consciousness documented by a Glascow coma scale (GCS) score less than 13 will require CT imaging in the acute setting. Controversy exists around the role of CT imaging in minor head trauma, defined as patients with blunt head injury, amnesia or disorientation or documented loss of consciousness, and a GCS score of 13 to 15. Several scoring systems have been suggested,[23-25] with the larger, validated Canadian CT rule[23] outperforming other published rules in adults with head trauma. Adults with risk factors delineated by the Canadian CT rule require emergent CT scanning. (See *Table 1.*) Using these criteria for obtaining a head CT in minor trauma, the authors noted a sensitivity of 100% and a specificity of 68.7% in detecting injuries requiring neurosurgical intervention. Additionally, they found a sensitivity of 98.4% and a specificity of 49.6% for clinically important brain injuries. Finally, the Canadian CT rule reduced the number of CT studies by almost 50%. Some additional patient populations must be considered for early use of CT. These include intoxicated patients,[26,27] patients with bleeding disorders,[28] patients with shunts from prior neurosurgical procedures,[29] and patients who return after initial evaluation following a head injury.[30] Other authors have independently found age greater than 65 years to be a risk factor for significant head injury.[31,32] However, even with a clinical model that includes these high-risk patients, it has been suggested that 100% sensitivity for intracranial injuries may not be achieved.[33,34] Patients with no loss of consciousness, normal neurologic findings, no vomiting, no amnesia, and minimal scalp injury can be discharged with careful instructions including 24 hours of close observation.

Clinicians should have a lower threshold for imaging infants and children with head injury as clinical features can be particularly subtle.[35] Independent risk factors for closed head injury include headache, vomiting, an altered mental status, physical signs of a basilar skull fracture, and focal neurological deficits. Importantly, a scalp hematoma alone signifies an increased risk of intracranial injury in infants younger than 1 to 2 years and careful consideration of CT imaging.[35,36] One additional consideration is the fact that significant pathology can arise after a presumably normal CT scan. This occurrence is particularly true of cerebral contusions, which commonly develop hours to days after injury. Even delayed epidural[37] and subdural[38] hematomas have been reported.

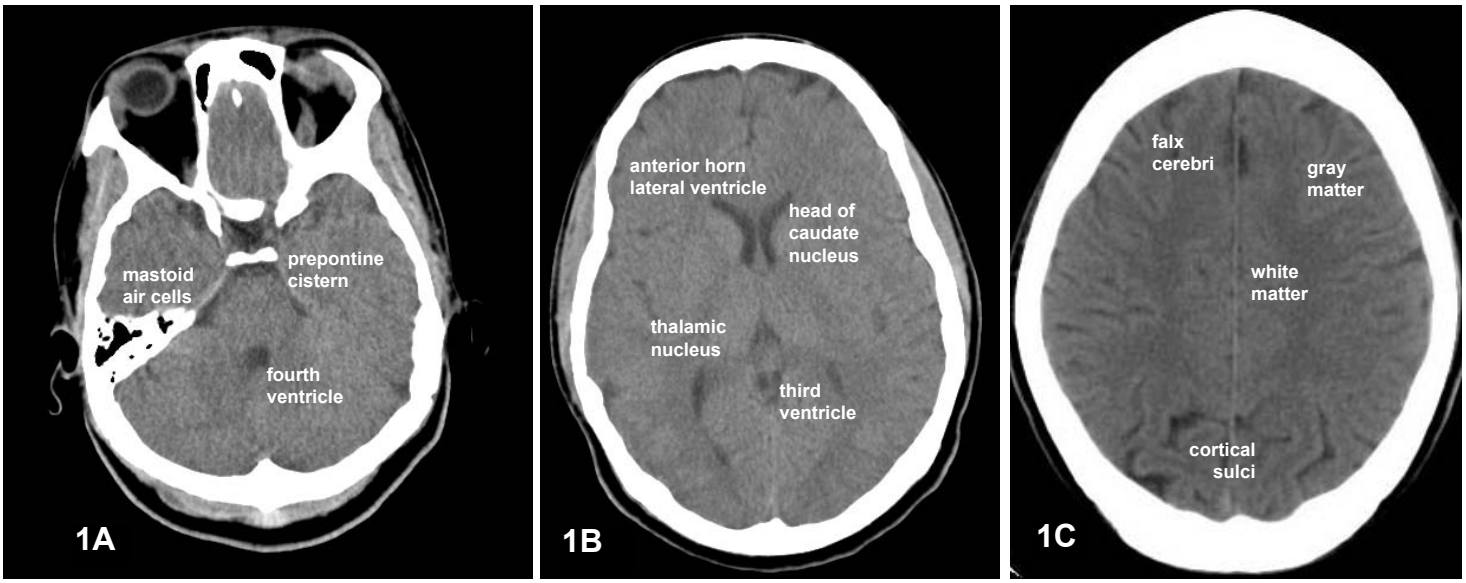
Interpretation of Radiographs in Head Trauma

As with other body systems, practitioners must have an organized approach in viewing radiographs of the cranial vault and its internal structures. Because of the importance of CT scanning in assessing the patient with head trauma, we will focus primarily on findings with this modality. The general principle is to divide head-injured patients into the following broad categories: normal intracranial findings, diffuse intracranial injury, and focal intra-axial or extra-axial findings.

Remember that the patient is placed supine on the CT table and the axial images are constructed as if one is viewing from the feet upward. As a result, the patient's left is on the right side of the image as with plain radiography. This is also true for MR images. It also should be remembered that black, gray, and white are defined by the tissue densities similar to plain radiography. However, specific tissues may be accentuated by viewing the various window settings as described previously.

The initial evaluation of the CT scan should consider the technical adequacy of the study. The axis of the x-ray beam is angled 20° downward along a line extending from the lateral canthus to the external acoustic meatus. If the patient's head is tilted as it enters the gantry, an oblique rather than a true axial cut is obtained. This can obscure an abnormality or create the appearance of an abnormality in a normal brain. The CT scout image is useful when assessing the technical quality of the study by demonstrating the orientation of the images, as well as the number of images produced and the anatomic area scanned. The

Figure 1. Cross-sectional Image of Brain at Various Levels



scout image is also valuable in assessing the skull for fractures that can be visible on this image. Images should also be evaluated for motion artifact or artifacts related to the presence of bullets or other dense foreign bodies.

Interpretation of CT imaging requires a thorough understanding of the cross-sectional anatomy of the brain. A complete review of neuroanatomy is beyond the scope of this article, however, excellent reviews on this subject are available.[39] With each slice, specific anatomic landmarks should be located as described below. Specific focus on symmetry, the cerebrospinal fluid (CSF) spaces, brain tissue, the skull, and soft tissues should be reviewed on brain window settings. Brain windows are used to highlight cerebral anatomy. Bone windows better delineate skull fractures and pneumocephaly. Blood windows more clearly demonstrate intracranial hemorrhage, which can be subtle, especially near the inner table of the skull. Localized soft-tissue swelling over the skull may be an indication of head trauma and may yield clues as to the location of coup and contre-coup injuries.

By convention, CT images progress cephalad from the foramen magnum to vertex of the skull. Important structures to identify on the lower images are the fourth ventricle, which lies on the midline, dorsal to the brainstem. Ventral to the brainstem are the prepontine and supracellar cisterns (*Figure 1A*). Because CSF is clear, these fluid-filled spaces appear dark gray. Air-filled spaces (e.g., the mastoid air cells and paranasal sinuses) are black on the image. Blood in either the CSF or sinuses will alter their appearance by increasing their densities. The posterior fossa structures demonstrated on lower CT images also include the cerebellar hemispheres and the dural sinuses.

Proceeding cephalad, the inferior portions of the cerebral hemispheres and the third and lateral ventricles can be seen (*Figure 1B*). The third ventricle is an important landmark for intracranial mass effect causing midline shift. The ambient cisterns that surround the upper pons are also visible and are important in assessing mass effect on the brainstem from injury to the temporal lobes. The third ventricle is flanked by the thalamic nuclei and each frontal horn of the lateral ventricles lies adjacent to the head of the caudate nucleus. These gray matter structures are of intermediate density as compared with the dark CSF in the adjacent ventricle.

Continuing cephalad, the cerebral cortex occupies virtually the entire image (*Figure 1C*). One should appreciate a distinct border between gray and white matter structures. In addition, the cortical sulci should be well delineated and should appear more prominent in the older brain. On the higher images, the falx cerebri becomes visible and becomes a second important midline structure. Ensure that equal volume occupies the right and left halves of the cranial vault and that no structure has crossed the midline.

Finally, there are three questions that you should consider as you view the images of a head CT scan:

- Is there blood in or around the brain?
- Is there a shift in the midline structures?
- Is there evidence of brain edema?

In the CT scan of a patient with acute injury, clotted blood is typically identified as a high density focus. Clotted blood tends to assume a characteristic shape depending upon the nature of the underlying abnormality, as will be discussed later. Next, one looks at all of the images for asymmetrical displacement of intracranial structures across the midline. Important landmarks that are useful midline indicators include the third and fourth ventricles and the falx cerebri. In addition, the ventricles may be obliterated by mass effect from edema or hematoma. Small volumes of blood in CSF can make the subarachnoid spaces appear isodense with brain, essentially obliterating the ventricles, sulci, and basal cisterns.

Table 2. Mnemonic for Reviewing Head CT Scan[14]

BLOOD:
- Epidural hematoma
- Subdural hematoma
- Intraparenchymal hemorrhage
- Intraventricular hemorrhage
- Subarachnoid hemorrhage

CAN (CISTERNS): EXAMINE FOR BLOOD AND EFFACEMENT
- Circummesencephalic
- Suprasellar
- Quadrigeminal
- Sylvian

BE (BRAIN):
- Symmetry
- Gray-white differentiation
- Shift
- Hyper- or hypodensity
- Pneumocephalus

VERY (VENTRICLES):
- Effacement
- Shift
- Blood

BAD (BONE): FRACTURES, SOFT-TISSUE INJURY, BLOOD IN SINUSES

Finally, one should appreciate a clear delineation between the gray and white matter structures in the brain. Significant edema reduces or eliminates these density differences to produce a more uniform, isodense appearance of brain tissue. Edema may manifest as either a localized or diffuse hypodense area of brain (usually 12 to 24 HU) with ill-defined gray/white boundaries resulting from increased brain water content. Edema also may result in the loss of sharp delineation of sulci. An area of suspected edema can be compared with the contralateral hemisphere to better appreciate the abnormality.

Perron[14] has suggested that the pneumonic "Blood Can Be Very Bad" be used to ensure that the examiner has reviewed all of the important features of the CT scan (*Table 2*).

MRI has not gained widespread use in the acute management of the injured patient due to a number of factors including cost, availability, speed of scanning, potential for motion artifact, limitations on the use of life support equipment in the strong magnetic field and insensitivity to bone involvement. On the other hand, MRI does not utilize ionizing radiation, can better discriminate between similar tissues, is better at defining cerebral edema, and is not complicated by bone artifacts. It is also able to provide delineation of structures in three orthogonal planes (axial, sagittal and coronal); CT scanning is generally limited to the axial plane when imaging the acutely injured patient. Small parenchymal injuries and collections of blood at the vertex and skull base are better elucidated by MRI. At least one study has demonstrated the superiority of MRI in identifying intracranial lesions;[40] however, these radiologic findings did not affect clinical management. The basic anatomy and approach are similar to reviewing CT scans. In the setting of acute trauma, MRI may be useful in assessing the extent of parenchymal injury, the exact location of extra-axial blood collections (i.e., subdural, epidural, and subarachnoid hemorrhage), the integrity of vascular structures using MR angiography, and potentially to determine the metabolic impact of injury using MR spectroscopy.

Figure 2. Linear Skull Fracture

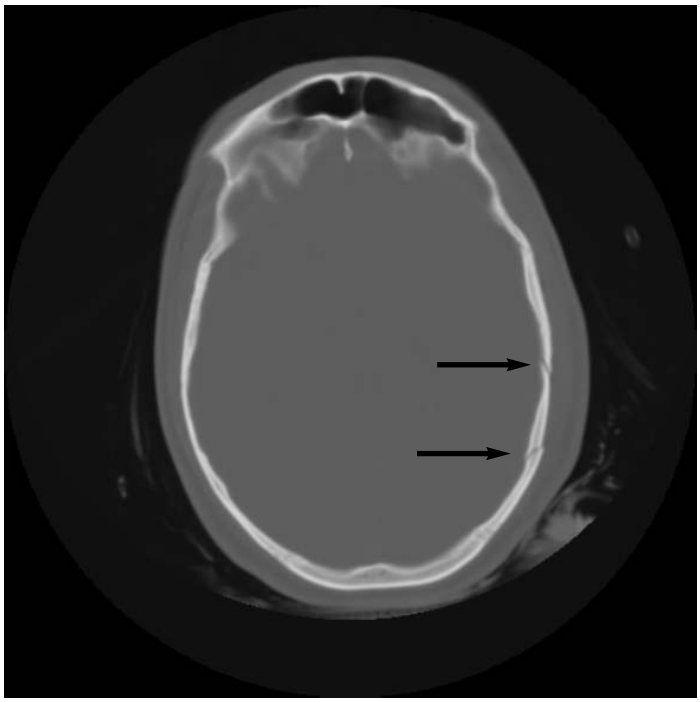


Figure 2. Black arrows indicate site of fracture.

Specific Injuries

Skull fractures can be identified using plain radiographs or CT scans. When the latter is used, bone windows best define the fracture line. Skull fractures are described as *linear*, *basilar*, or *depressed*. Diastatic fractures occur along existing suture lines. In the plain radiograph, a linear fracture appears as a clean sharply demarcated radiolucent line that is typically wider in its central portion and tapered at both ends. Linear fractures appear more sharply delineated than vascular grooves or suture lines. Fractures can further be differentiated from suture lines by the irregular corticated border seen with sutures. On a CT scan, the fracture line may be readily apparent. However, the fracture may be difficult to appreciate if it is transverse in its orientation, lying within the plane of the image. Examination of the scout film may reveal the fracture line (*Figure 2*). One should look for evidence of focal soft-tissue swelling in the scalp to identify areas of potential skull fractures. MRI is not useful in the assessment of skull fractures because this imaging modality requires that protons be mobile, and the protons of cortical bone are immobile.

Figure 3. CT Scans Demonstrating Depressed Skull Fracture, Basilar Skull Fracture, and Cerebral Contusion

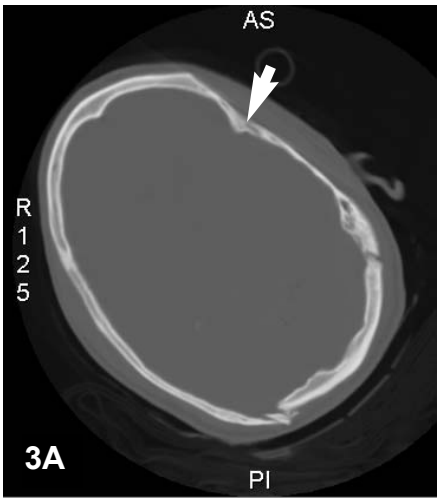


Figure 3A. Depressed Skull Fracture. Arrow denotes site of depression.

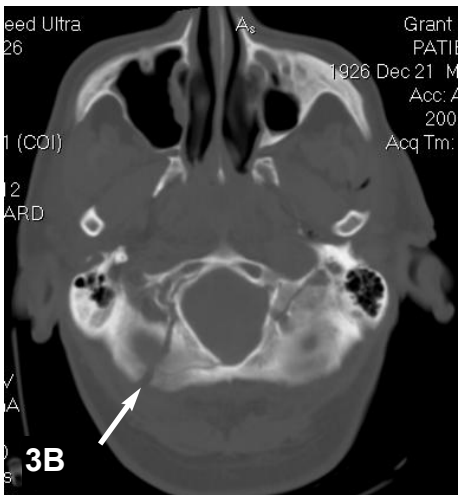


Figure 3B. Basilar Skull Fracture. Arrow demonstrates fracture through the skull base.

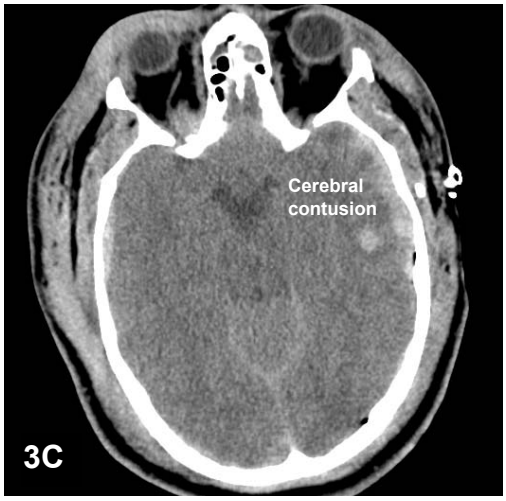


Figure 3C. Cerebral Contusion.

Thus, cortical bone appears uniformly black with MRI.

A depressed skull fracture is seen as overlapping cortical shadows on plain radiographs (*Figure 3A*). These injuries can be further elucidated by obtaining tangential views in the area of the fracture. Skull fractures are typically the result of direct trauma to the skull, most commonly in the frontal or parietal regions of the skull. A depressed skull fracture is believed to be significant when the deepest portion of the fracture extends below the inner table of the skull. These fractures are typically associated with injuries to brain parenchyma and dural tears. CT scanning offers the advantage of accurately defining the depth of the fracture and demonstrating any underlying brain injury.

Finally, basilar skull fractures are typically defined by clinical findings of raccoon eyes, Battle's sign, hemotympanum, CSF rhinorrhea, or otorrhea. A Towne view taken with plain skull radiographs occasionally may demonstrate the fracture. CT images of the posterior fossa with 1 to 2 mm cuts are the best method of demonstrating the fracture site (*Figure 3B*). Extension of the fracture line through the outer table or into an air-filled cavity (e.g., paranasal sinuses, nasal cavity, mastoid air cells) defines a compound basilar skull fracture. Pneumocephalus may accompany a compound basilar skull fracture. Basilar skull fractures through the petrous or sphenoid bone also may result in cranial nerve injuries and are best demonstrated on high-resolution CT.

Intracranial blood is classified as being *intra-axial* (within the brain substance) or *extra-axial*. The most common intra-axial traumatic lesions are cerebral contusions, hematomas, and shearing injuries. Contusions are ill-defined collections of blood along the brain surface or within the brain substance, whereas hematomas are well-circumscribed. Contusions are typically seen in the gray matter but may extend into the subcortical white matter (*Figure 3C*). There is typically a high density focus representing hemorrhage surrounded by low density edema. These are typically defined as *coup* or *contrecoup* injuries. Coup injuries occur beneath the surface of the skull at the site of impact, whereas contrecoup injuries occur on the opposite side of the brain along the line where the force was directed. Contusions have been found in high association with depressed skull fractures. MRI also may be used to define cerebral contusion. T2-weighted images demonstrate a low intensity signal (hematoma) surrounded by a high-intensity signal (edema).

Cerebral hematomas occur in a distribution similar to cerebral contusions and are most common in the frontal and temporal regions of the brain (*Figure 4*). Hematomas may be the result of the dissection of the clot through the brain white matter or the disruption of a penetrating vessel in this region. Large clots can actually dissect through the white matter into the ventricular system that may result in obstructive or communicating hydrocephalus. Cerebral hematomas are found in association with other brain injuries in 50% of cases, and intraventricular extension occurs in 33% of cases; 20% of these lesions are bilateral.[41-42] On CT, hematomas are identified by distinct collections of hyperdense blood surrounded by a hypodense area of edema in the acute injury and serum from clot degradation in the subacute injury. Typically, these are found in the frontal or anterior temporal lobe. On MRI, cerebral hematomas are similar in appearance on T2-weighted images in that the clot is hypointense and the edema or serum is hyperintense. Delayed appearance of cerebral hematomas 1 to 7 days after injury has also been reported.[43,44]

Shear injuries typically occur at the junction of the gray and white matter of the brain as the result of rotational forces, resulting in disruption of axons and their small accompanying blood vessels. These injuries can produce diffuse cerebral edema but also may appear as multiple distinct hemorrhagic foci varying from punctate to a few centimeters in diameter. Cisternal and ventricular compression may be seen but is not common in acute injury. Typically, lesions are found in four areas: the corpus cal-

Figure 4. Cerebral Hematoma

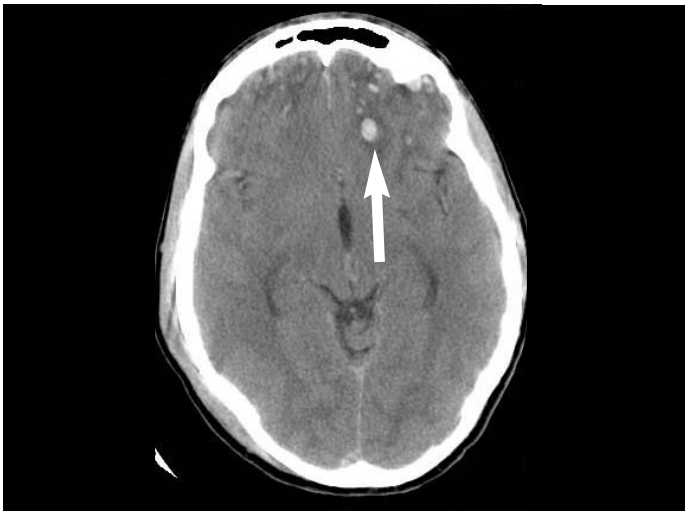


Figure 4. Cerebral Hematoma. Arrow indicates area of cerebral hematoma.

Figure 5. Subdural Hematoma

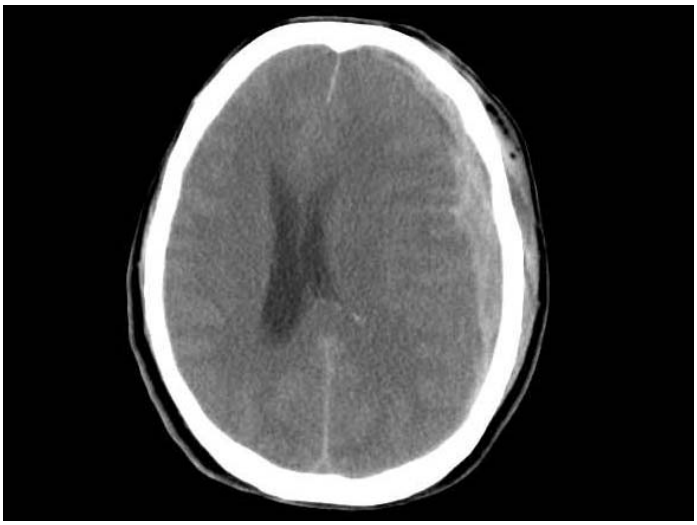


Figure 5. Subdural Hematoma. Note midline shift of lateral ventricle.

losum, the corticomedullary junction, the upper brainstem, or the basal ganglia. Hemorrhage into the corpus callosum may extend into the ventricular system where it is seen more easily.[45] It has been demonstrated that only one-fourth of shearing injuries initially present with visible hemorrhage. Thus, a negative CT study does not eliminate the possibility of shearing injury. Delayed presentations of diffuse axonal injury also have been reported.[46]

MRI has been shown to better demonstrate findings of diffuse axonal injury.[47,48] MRI should be performed when there is a disparity between the patient's clinical picture and the CT image. A high intensity signal is seen on T2-weighted images in the four regions where typical lesions occur. However, when demonstration of microhemorrhage is required, the preferred scan technique uses gradient-refocused imaging. This technique is highly sensitive to the presence of iron within hemoglobin.

Extra-axial injuries include subdural hematoma, epidural hematoma, and traumatic subarachnoid hemorrhage including intraventricular hemorrhage. In each case, the characteristic findings are defined by the relationship of the bleeding source to the brain and the meninges.

A subdural hematoma collects between the inner layer of the dura and the subarachnoid space when there is a tear in the bridging veins. The subdural space extends over both sides of the tentorium, cerebellar surfaces, convexities of the brain, and the interhemispheric fissure. Because a subdural hemorrhage lies deep to the dura, it can cross suture lines. Occasionally, a rupture of a parenchymal artery may produce these lesions. Subdural hematomas typically are associated with underlying brain injury and, therefore, carry a poor prognosis. Typical associated injuries include cerebral hematoma or contusion. Associated skull fractures are rare.

A typical subdural hematoma appears as a crescent-shaped collection of blood between the inner table of the skull and brain parenchyma (*Figure 5*). The classic shape may be modified in patients with previous injuries or meningeal infection. Small subdural injuries may be difficult to identify (S*ee* Pitfalls *section, page 8.*) and also may coexist with an epidural hematoma. These injuries tend to extend from front to back. They also may extend around the frontal or occipital pole into the interhemispheric fissure or beneath the brain along the tentorium. These injuries are often associated with displacement of intracranial contents by mass effect.[49] Collapse of the ventricular system and midline shift commonly are associated with acute subdural hematoma. In the acute phase of bleeding associated with a subdural hematoma, MRI is very insensitive in defining this injury and has gained a prominent role in the detection and defining the extent of subacute and chronic subdural hematomas.

An epidural hematoma is typically lens-shaped with blood collecting between the inner table of the skull and the dura. Because the dura attaches to the skull at suture lines, an epidural hematoma does not cross suture lines. Classically, an epidural hematoma results from a skull fracture in 85% to 95% of cases.[50] The most common site of an epidural hematoma is in the temporoparietal region and is due to disruption of the main trunk of the middle meningeal artery (*Figure 6*). This also may give rise to epidural blood on the floor of the middle cranial fossa. Disruption of the anterior trunk of the middle meningeal artery gives rise to frontal epidural hematomas. Other injuries are venous in origin and result from the tear of a dural sinus.

Epidural hematomas are almost exclusively unilateral injuries. An epidural hematoma in the posterior fossa is typically caused by venous bleeding; occipital fractures may be seen. These lesions account for 4% to 13% of epidural hematomas.[51,52] These injuries must be identified early; with prompt neurosurgical intervention a good prognosis can be expected. The size of the lesion depends upon the rate of bleeding, the time from injury to presentation, the severity of injury, and the formation of clot. Large epidural hematomas compress adjacent brain tissue, cause

Figure 6. Epidural Hematoma

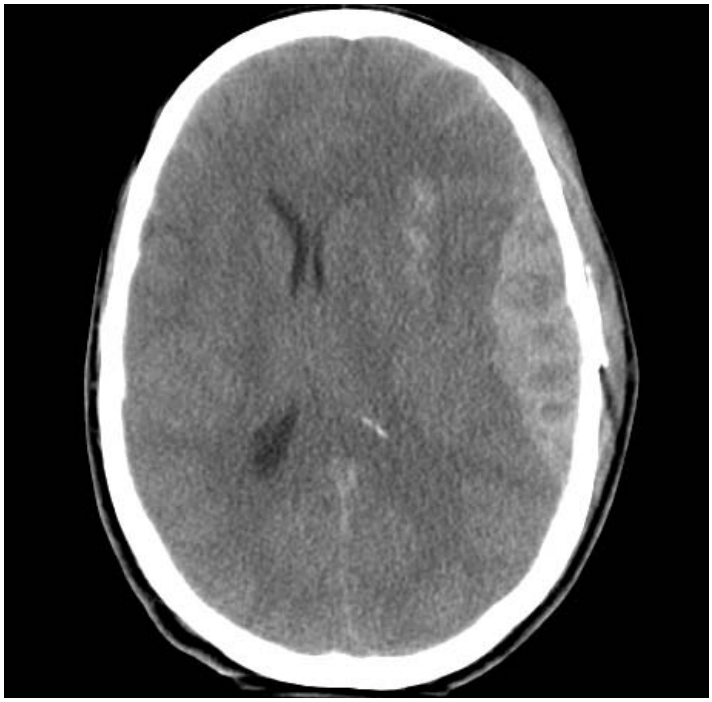
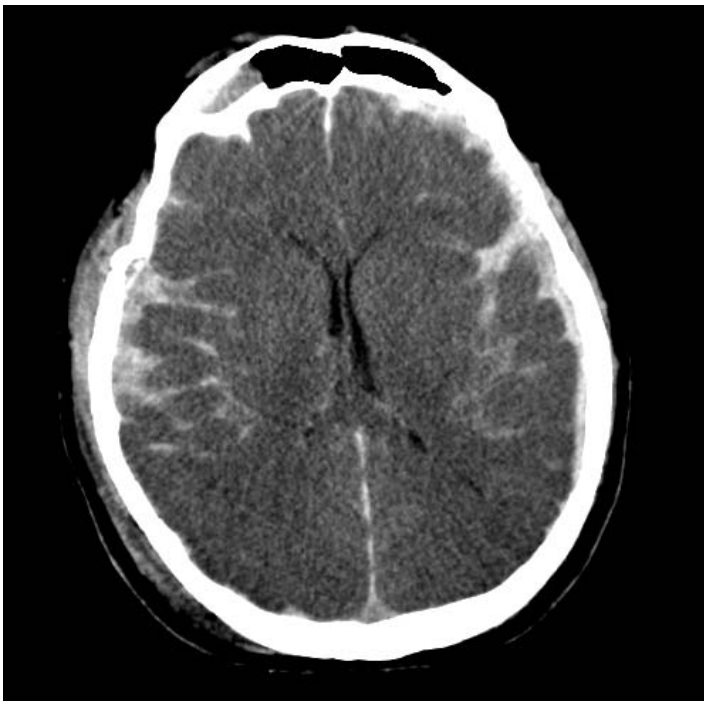


Figure 7. Traumatic Subarachnoid Hemorrhage



collapse of the ventricular system, and result in brain herniation. The lesion may not be uniform in appearance with an area of less-dense fluid representing both active bleeding and breakdown of clot.[50]

Finally, epidural hematomas commonly are associated with cerebral contusion or subdural hematoma.[53] Because these lesions must be promptly evacuated surgically, there is typically no role for MRI in acute management of epidural hematomas. Finally, it should be mentioned that epidural hematomas may have a delayed presentation arising hours to days after injury. These are typically found in areas beneath known skull fractures and frequently occur after evacuation of other intracranial lesions.[54]

In traumatic subarachnoid hemorrhage, disruption of the leptomeningeal vessels leads to bleeding, which tracks into the subarachnoid space. These collections course along the cerebral sulci and also may be seen along the interhemispheric fissure (*Figure 7*). Blood also can track into the ventricular system and can be visualized along the surface of the brain or layering dependently in the occipital horns of the lateral ventricle. Traumatic subarachnoid hemorrhages tend to be more focal than the spontaneous subarachnoid hemorrhage associated with a ruptured aneurysm.

Finally, disruption of the subependymal veins can result in traumatic intraventricular hemorrhage (*Figure 8*). This appears as a hyperdense localized collection within the ventricular system following trauma. Blood also may be seen in association with traumatic subarachnoid hemorrhage that extends into the ventricular system as described above.

Pitfalls in Radiology Interpretation

Bone averaging (*Figure 9*) occurs where a high-density structure (e.g., bone) abuts a low density structure (e.g., brain tissue). Where this occurs, the high and low density pixels are 'averaged' and give the appearance of an intermediate density that can mimic blood. Similarly, areas of calcification within the brain (e.g., the pineal gland, the choroid plexus, within the basal ganglia, or even a partial cut through the bony floor of the skull) may be mistaken for blood. Additionally, the falx cerebrum and tentorium tend to calcify with age and may be mistaken for subdural blood.

Small collections of intra-axial (cerebral contusion) or extra-axial blood (subdural or epidural hematoma or subarachnoid hemorrhage) located near the inner table of the skull may be difficult to appreciate, especially if only brain windows are used. The high density of blood may be indistinguishable from the adjacent cortical bone, a fact particularly true for extra-axial hematomas and cerebral contusions located in the parietal vertex and inferior temporal lobe. Extra-axial hematomas or subarachnoid blood that collects along the transverse plane may be difficult to distinguish from bone when present along the floor of the posterior fossa (*Figure 10*). Collections in the vertex or along the tentorium are better detected when coronal images are obtained.

CT does not provide adequate contrast between structures with similar densities, especially when one considers the high water content of brain structures. Artifact from metal structures (e.g., bullet fragments and aneurysm clips) can degrade the CT image by a phenomenon known as 'beam hardening,' where high density structures (e.g., the skull) may produce a streak artifact that can obscure the images.

Figure 8. Traumatic Intraventricular Hemorrhage

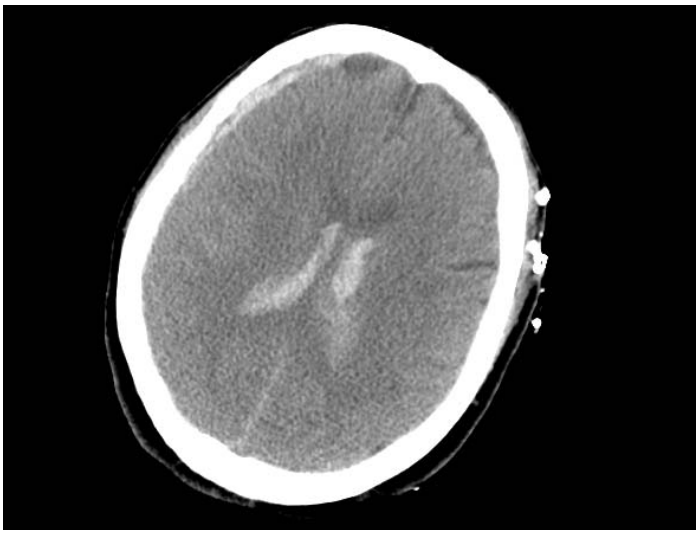


Figure 8. Traumatic Intraventricular Hemorrhage. Note that this patient also has a right subdural hematoma in the frontoparietal region.

Figure 10. Tentorial Hemorrhage

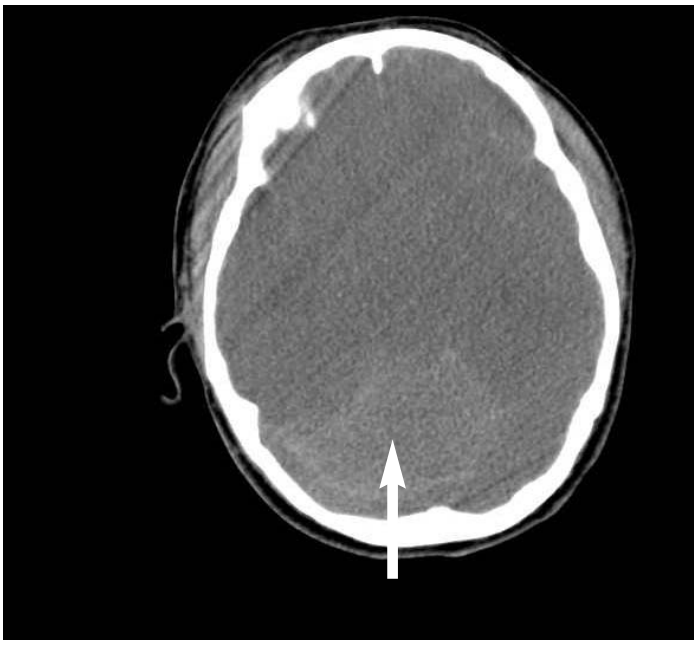


Figure 10. Tentorial Hemorrhage. Arrow depicts blood along the tentorium.

An important CT indicator of brain swelling is effacement of the subarachnoid spaces surrounding the brainstem (basal cisterns). However, blood within the basal cisterns can make them isodense with the brainstem, mimicking herniation.

Interhemispheric blood is often confused with thickening of the falx cerebri. The falx may be prominent along its posterior portion. However, subdural blood that collects along the falx can be difficult to distinguish from the more benign condition (*Figure 11*). When there is significant thickening along the anterior portion of the falx or when there is outlining of the adjacent sulci, interhemispheric blood must be suspected. Localized subdural hematomas may be found along the interhemispheric fissure. These are the result of tears in bridging veins and tend to occur in older patients with whiplash injury. Interhemispheric subdural hematomas tend to have a straight medial border and a convex lateral margin. The bleeding from a parasagittal subdural hematoma tends to remain contained in this region by arachnoid granulations. In addition, subarachnoid or subdural hemorrhage in the posterior falx may produce a high density ('pseudo-delta') sign that can be confused with the image of dural sinuses in a contrast-enhanced CT scan. Subarachnoid hemorrhage along the interhemispheric fissure is characterized by increased density outlining the parasagittal cortical sulci.

The sensitivity of CT may be limited when imaging subacute injuries. Subdural hematomas in the elderly patient are notorious for their subacute presentation. This relates to the breakdown of hemoglobin over time, with blood clot becoming isodense with brain and less visible on CT (usually around two weeks after the injury). In this circumstance, MRI becomes a more sensitive imaging modality for defining subacute or chronic collections of blood that will demonstrate a high-intensity signal on T1-weighted images.

Figure 9. Bone Averaging

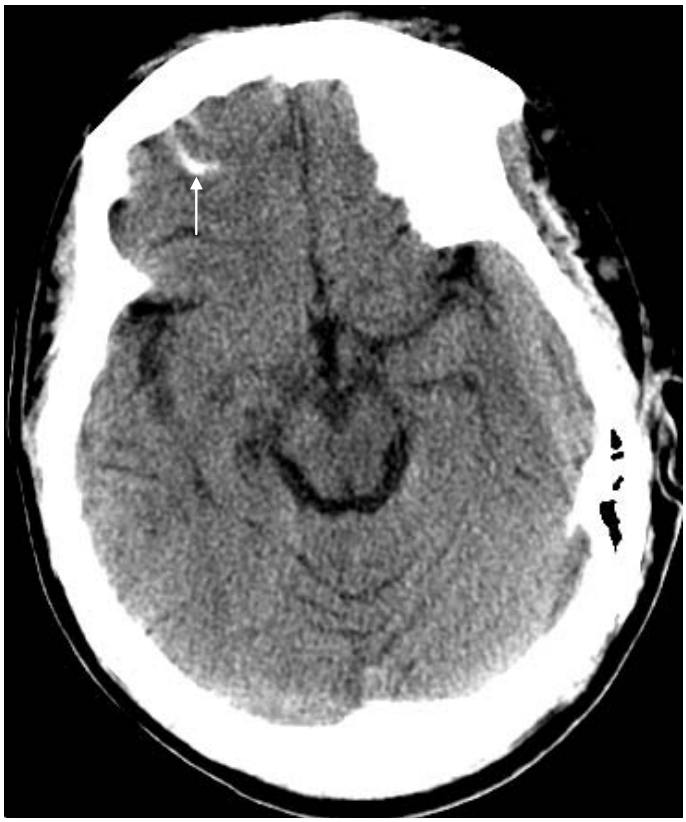


Figure 9. **Bone Averaging**. White arrow depicits bone averaging artifact from orbital roof originally interpreted as subarachnoid blood.

Figure 11. Subarachnoid Blood Along Falx

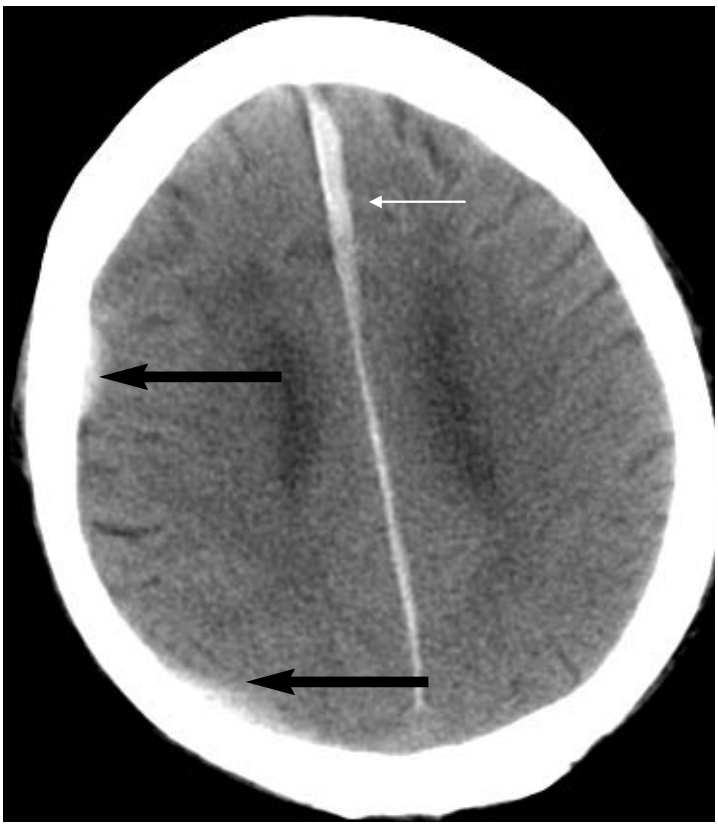


Figure 11. Subarachnoid Blood Along Falx. White arrow depicts blood along the falx. Black arrows demonstrate subdural hemorrhage along frontal and parietal convexities.

Conclusions

The clinician should be aware of the role of the various imaging modalities in the assessment of the patient with head trauma. CT imaging has revolutionized the evaluation of head injury, providing a rapid and reliable method for detecting acute traumatic hemorrhage and facilitating early surgical intervention when appropriate. Skull radiographs and MRI have limited roles in the management of early head injury. The clinician should be aware of the characteristic shape and location of intra-axial and extra-axial blood collections. In addition, when interpreting CT scans in the acute setting the clinician should be cognizant of difficulties in interpreting these radiographs caused by artifacts inherent in the imaging modality and unusual presentations of the resultant injury.

References

1. McCraig LF, Ly N. National Hospital Ambulatory Medical Care Survey: 2000 Emergency Department Summary. *Advance Data* 326:1-26, 2000.
2. Mayhue FE, Rust DD, Aldag JC, et al. Accuracy of interpretation of emergency department radiographs: Effects of confidence levels. *Ann Emerg Med* 1989;18:826.
3. Reisdorff EJ. Radiologic studies in the ED at Ingham Regional Medical Center, Lansing, MI (unpublished data). January 1996.
4. Gratton MC, Salomone JA III, Watson WA. Clinically significant radiograph misinterpretation at an emergency medicine residency program. *Ann Emerg Med* 1990;19:497.
5. Zimmerman RA, Bilaniuk LT, Gennarelli T. Computed tomography of shearing injuries of the cerebral white matter. *Radiology* 1978; 127:393-396.
6. Miller JD, Tocher JL, Jones PA. Extradural hematoma – earlier detection, better results [editorial]. *Brain Inj* 1988;2:83-86.
7. Servadei F, Piazzi G, Seracchioli A, et al. Extradural hematoma: An analysis of the changing characteristics of patients admitted from 1980 to 1986: Diagnostic and therapeutic implication in 158 cases. *Brain Inj* 1988;2:87-100.
8. Berlin L. Malpractice and radiologist, update 1986: An 11.5 year experience. *AJR* 1986;147:1291.
9. Trautlein J, Lambert RL, Miller J. Malpractice in the emergency department – Review of 200 cases. *Ann Emerg Med* 1984;13:709.
10. Alfaro D, Levitt MA, English DK, Williams V, Eisenberg R. Accuracy of interpretation of cranial computed tomography scans in an emergency medicine residency program. *Ann Emerg Med* 1995; 25:169-174.
11. Arendts G, Manovel A, Chai A. Cranial CT interpretation by senior emergency department staff. *Australas Radiol* 2003;47:368-374.
12. McLain PL, Kirkwood CR. The quality of emergency room radiograph interpretation. *J Fam Pract* 1985;20:443.
13. Overton DT. Quality assurance assessment of radiograph reading accuracy by emergency medicine faculty (abs). *Ann Emerg Med* 1987;16:503.
14. Perron AD, Huff JS, Ullrich CG, Heafner MD, Kline JA. A multicenter study to improve emergency medicine residents' recognition of intracranial emergencies on computed tomography. *Ann Emerg Med* 1998 Nov;32(5):554-562.
15. Eng J, Mysko WK, Weller GE, Renard R, Gitlin JN, et al. Interpretation of Emergency Department radiographs: a comparison of emergency medicine physicians with radiologists, residents with faculty, and film with digital display. *AJR* 2000;75:1233-1238.
16. Galasko CSB, Monahan PRW. Value of re-examining x-ray films of outpatients attending accident services. *BMJ* 1977;1:643.
17. de Lacey G, Barker A, Harper J, et al. An assessment of the clinical effects of reporting accident and emergency radiographs. *Br J Radiol* 1980;53:394.
18. Rhea JT, Potsaid MS, DeLuca SA. Errors of interpretation elicited by a quality audit of an emergency radiology facility. *Radiology* 1979;132:277.
19. Stockburger WT. CT imaging, then and now. *Radiol Management* Nov/Dec 2004:20-27.
20. Macpherson BC, Macpherson P, Jennett B. CT evidence of intracranial contusion and haematoma in relation to the presence, site and type skull fracture. *Clin Radiol* 1990;42:321.
21. de Lacey GJ, Barker A, Wignall V, et al. AJR for requesting radiographs in an accident department. *BMJ* 1979;1:159.
22. Eliastam M, Rose E, Jones H, et al. Utilization of diagnostic radiologic examinations in the emergency department. *J Trauma* 1980; 20:61.
23. Stiell IG, Wells GA, Vandernheen K, et al. The Canadian CT head

rule for patients with minor head injury. *Lancet* 2001;357: 1391-1396.
24. National Institute for Clinical Excellence (NICE). Head injury triage, assessment, investigation and early management of head injury in infants, children and adults. *Clinical Guideline 4.* London: NICE 2003;96-7.
25. Haydel MJ, Preston CA, Mills TJ, Luber S, et al. Indications for computed tomography in patients with minor head injury. *NEJM* 2000;343:103-105.
26. Nagy KK, Joseph KT, Krosner SM, et al. The utility of head computed tomography after minimal head injury. *J Trauma* 1999; 46:268-270.
27. Cook LS, Levitt MA, Simon B, Williams VL. Identification of ethanol-intoxicated patients with minor head trauma requiring computed tomography scans. *Acad Emerg Med* 1994;1:227-234.
28. Reinus WR, Swerner FL Jr. Clinical prediction of emergency cranial computed tomography results. *Ann Emerg Med* 1994;23: 1271-1278.
29. Schutzman SA, Greenes DS. Pediatric minor head trauma. *Ann Emerg Med* 1992;37:65-74.
30. Voss M, Knottenbelt JD, Peden MM. Patients who reattend after head injury. *BMJ* 1995;311:1395-1398.
31. Mack LR, Chan SB, Silva JC, Hogan TM. The use of head computed tomography in elderly patients sustaining minor head trauma. *J Emerg Med* 2003;24:157-162.
32. Arienta C, Caroli M, Balbi S. Management of head-injured patient in the emergency department: a practical protocol. *Surg Neurol* 1997;48:213-219.
33. Ibanez J, Arikan F, Pedraza S, et al. Reliability of clinical guidelines in detection of patients at risk following mild head injury: Results of a prospective study. *J Neurosurg* 2004;100:825-834.
34. Borczuk P. Predictors of intracranial injury in patients with mild head trauma. Predictors of intracranial injury in patients with mild head trauma. *Ann Emerg Med* 1995;25:731-736.
35. Schutzman SA, Greenes DS. Pediatric minor head trauma. *Ann Emer Med* 2001;37:65-73.
36. Isaacman DJ, Poirier MP. Closed head injury in children. *Pediat Emerg Care* 2002;18:48-52.
37. Milo R, Razon N, Schiffer J. Delayed epidural hematoma: A review. *Acta Neurochir* 1987;84:13-23.
38. Snoey ER, Levitt MA. Delayed diagnosis of subdural hematoma following normal computed tomography scan. *Ann Emerg Med* 1994;23:1127-1131.
39. Huddle DC, Glazer M, Chaney DB. Emergency imaging of the brai. In: Schwartz DT, Reisdorff E (eds.). *Emergency Radiology*. New York:McGraw-Hill;2000:385-442.
40. Pitts LH. The role of neuroimaging in minor head trauma. *Ann Emerg Med* 1991;20:1387-1388.
41. Zimmerman RA, Bilaniuk LT, Gennarelli T, et al. Cranial computed tomography in diagnosis and management of acute head trauma. *AJR* 1978;131:27.
42. Dublin AB, French MD, Rennick JM. Computed tomography in head trauma. *Radiology* 1977;122:365-369.
43. Dolinskas C, Bilaniuk LT, Simmerman RA, et al. Computed tomography of intracerebral hematomas I. Transmission of CT observations of hematoma resolution. *AJR* 1977;129:681-688.
44. Kishore PRS. Delayed sequelae of head injury. *J Computed Tomogr* 1980;4:287-295.
45. Abraszko RA, Zurynski YA, Dorsch NW. The significance of traumatic intraventricular hemorrhage in severe head injury. *Br J Neurosurg* 1995;9:769-773.
46. Corbo J, Tripathi P. Delayed presentation of diffuse axonal injury: A case report. *Ann Emerg Med* 2004;44:57-60.
47. Gentry LR, Godersky JC, Thompson B, et al. Prospective comparative study of intermediate-field MR and CT in the evaluation of closed head trauma. *AJR* 1988;150:673-682.
48. Gentry LR, Thompson B, Godersky JC. Trauma to the corpus callosum: MR features. *AJNR* 1988;9:1129-1138.
49. Forbes GS, Sheedy PF II, Piepgras DG, et al. Computed tomography in the evaluation of subdural hematomas. *Radiology* 1978; 126:143-148.
50. Zimmerman RA, Bilaniuk LT. Computed tomographic staging of traumatic epidural hematoma. *Radiology* 1982;144:809.
51. Ammirati M, Tomita T. Posterior fossa epidural hematoma during childhood. *Neurosurgery* 1984;14:541-544.
52. Garza-Mercado R. Extradural hematoma of the posterior cranial fossa: Report of severe cases with survival. *J Neurosurg* 1983; 59:664-672.
53. Tapiero B, Richer E, Laurent F, et al. Post-traumatic extradural hematomas. *J Neuroradiol* 1984;11:213-226.
54. Fukamachi A, Misumi S, Kaneko M, et al. Traumatic extradural hematomas with delayed development. *Comput Radiol* 1984;8: 197-201.

CE/CME Objectives

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

a.) discuss conditions that should increase suspicion for traumatic injuries;
b.) describe the various modalities used to identify different traumatic conditions;
c.) cite methods of quickly stabilizing and managing patients; and
d.) identify possible complications that may occur with traumatic injuries.

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. Which of the following are indications for a patient to receive a head CT?
   A. Glascow coma scale score of 13
   B. Open skull fracture
   C. Signs of a basilar skull fracture
   D. All of the above

2. Which one of the following groups of patients should physicians have a lower threshold for obtaining a head CT following trauma to the head?
   A. Intoxicated patients
   B. Patients taking antidepressant medications
   C. Patients with a history of a previous concussion
   D. Patients from prisons

3. In children, clinicians should have a lower threshold for obtaining a head CT after head trauma. Which of the following is *not* an independent risk factor for closed head injury?
   A. Crying
   B. Altered mental status
   C. Large scalp hematoma in a child younger than 1 year
   D. Focal neurologic deficit

4. The CT scout image is useful when assessing the technical quality of the study by demonstrating the orientation of the images as well as the number of images produced and the anatomic area scanned.
   A. True
   B. False

5. MRI has been shown to be superior to CT for the detection of:
   A. acute epidural hematomas.
   B. acute subdural hematomas.
   C. diffuse axonal injury.
   D. None of the above

6. Which of the following statements is *not* true regarding an epidural hematoma?
   A. It is usually a unilateral injury.
   B. The lesion is typically lens shaped.
   C. It is associated with a skull fracture in 10-15% of cases.
   D. The size of the lesion depends upon the rate of bleeding, time from injury to presentation, and severity of the injury.

7. Typically there is no role for the use of MRI in the acute evaluation of an epidural hematoma.
   A. True
   B. False

8. Which of the following statements regarding traumatic subarachnoid hemorrhage is true?
   A. Disruption of the leptomeningeal vessels leads to bleeding.
   B. Blood may track into the ventricular system and can be visualized along the surface of the brain or layering dependently in the occipital horns of the lateral ventricle.
   C. Traumatic subarachnoid hemorrhage tends to be more focal than the spontaneous subarachnoid hemorrhage associated with a ruptured aneurysm.
   D. All of the above

9. An important indicator of brain swelling is blood accumulation within the lateral ventricles.
   A. True
   B. False

10. Which of the following statements regarding interhemispheric blood is *not* true?
   A. It is often confused with thickening of the falx cerebri.
   B. Significant thickening along the anterior portion of the falx or outlining of the adjacent sulci should lead the clinician to suspect interhemispheric blood.
   C. Typically these are a result of an arterial bleed.
   D. Interhemispheric subdural hematomas tend to have a straight medial border and a convex lateral margin.

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

In Future Issues:

The use of ultrasound in the hypotensive trauma patient

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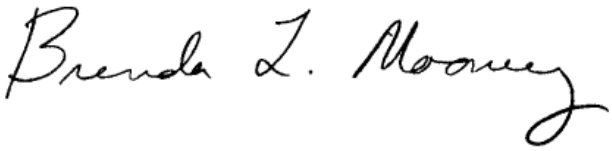
On behalf of Thomson American Health Consultants, we thank you for your trust and look forward to a continuing education partnership.

Sincerely,



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