DIAGNOSIS AND TREATMENT OF ACUTE
DECOMPENSATED HEART FAILURE IN THE EMERGENCY DEPARTMENT

by

Julie Lynn Ellis

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SIGNED: Julie L. Ellis

APPROVAL BY MASTER'S PROJECT DIRECTOR

This Master's Project has been approved on the date shown below:

Shu-Fen Wung, PhD, MS, RN, ACNP- BC, FAHA, FAAN
Associate Professor

Date:
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DEDICATION

This project is dedicated to the many women with heart failure, particularly those with peripartum cardiomyopathy. Caring for these very sick, very complex patients has made me passionate about acute decompensated heart failure. To the many families who have spent countless hours in the intensive care unit watching their loved ones battle through mechanical assist devices I applaud your strength and determination. I hope that this paper can aid in the understanding of acute heart failure and enable quick diagnosis and treatment.
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ABSTRACT

The number of hospitalizations due to heart failure has increased by 159 percent over the last few years. The non specific symptom presentation of heart failure can make diagnosis difficult and can cause a delay in treatment. The purpose of this literature review is to evaluate the current diagnosis of acute decompensated heart failure (ADHF) and to review different treatment algorithms for initial presentation in the emergency department (ED). DATA SOURCES: This literature review was performed by composing a PubMed search for articles from January 2004 to March 2010 containing the key words “heart failure,” “acute decompensated heart failure,” “heart failure guidelines,” “heart failure and ED,” “symptoms,” “acute heart failure,” “right and left heart failure,” “cardiomyopathy,” “ultra filtration,” “milrinone” and “treatment of heart failure.” Additional bibliographies were reviewed from the studies cited. FINDINGS: ADHF diagnosis requires excellent history taking as well as a combination of clinical findings, radiographic findings, and laboratory testing. Treatment options vary based on perfusion, volume overload, and severity of illness. CONCLUSION: A thorough history, brain natriuretic peptide levels, and echocardiogram are important in the diagnosis of ADHF in the ED. Treatment should start as soon as a diagnosis is made, and therapy should follow the hypotensive, normotensive, or hypertensive treatment pathways.
CHAPTER 1

Introduction

Approximately five million patients in this country have heart failure (HF), and over 550,000 patients are newly diagnosed each year (Hunt et al., 2009). Acute decompensated heart failure (ADHF) is the most common cause for hospitalization among patients over 65 years of age (Dec, 2007). It may result from new onset of ventricular dysfunction or, more typically, exacerbation of chronic HF symptoms. This growing number of cases typically presents in the elderly and in those who have previously suffered a myocardial infarction. Over the past decade, the rate of hospitalizations for HF has increased by 159 percent. One in eight deaths has HF listed on the death certificate (NCHS qtd in Rosamond, 2008). The growing number of patients with HF contributed to 27.9 billion dollars in healthcare costs in 2006 (Hunt et al., 2009). Patients with HF have 50% mortality within 5 years after the initial diagnosis (DiDomenco, et al., 2004).

ADHF is defined by three clinical profiles: 1) the patient with volume overload, manifested by pulmonary and/or systemic congestion, frequently precipitated by an acute increase in chronic hypertension; 2) the patient with profound depression of cardiac output manifested by hypotension, renal insufficiency, and/or a shock syndrome, and 3) the patient with signs and symptoms of both fluid overload and shock (Hunt et al., 2009). ADHF is sometimes used interchangeably with terms such as acute heart failure or acute heart failure syndromes.

ADHF is the primary diagnosis in approximately one million hospital admissions in the United States (U.S.) and the secondary diagnosis for nearly 2 million hospitalizations. The majority of the cost in HF is attributable to the management of episodes of ADHF resulting in
hospitalization. This includes prolonged length of stay, high readmission rates, and inpatient and outpatient morbidity and mortality (Abraham et al., 2005).

Clinical Significance/Clinical Implications

Clinicians in the ED have the daunting task of quickly identifying and treating ADHF patients. A delay in treatment of ADHF can occur because the presenting symptoms may or may not be clearly defined and can vary in severity (Chung & Hermann, 2006). In an article by Maisel et al. (2008), time of initial B-type Natriuretic Peptide (BNP) level being drawn and the first diuretic administered was associated with hospital mortality and length of stay (Maisel, et al., 2008). Early recognition and treatment of ADHF is vital to reduce morbidity, mortality, and hospital readmission rates.

Project Purpose

The goal of this paper is to identify diagnostic and treatment strategies of ADHF in the ED. These strategies can be used by healthcare providers

Review Method

This review includes information from the Acute Decompensated HEart Failure National REgistry (ADHERE), Heart Failure Society of America (HFSA), and American College of Cardiology Foundation (ACCF) and American Heart Association (AHA) current treatment guidelines. Other terms have been used to define ADHF. These are “acute heart failure syndrome,” “acute heart failure,” “cor pulmonale,” “congestive heart failure,” “cardiomyopathy,” “right or left sided heart failure,” and “systolic or diastolic” heart failure. The terminology variations can cause a gap in research and literature due to search engines
recognition. Searching the term “HF” yielded chronic HF, acute HF, acute decompensated, and right and left HF.
CHAPTER II LITERATURE REVIEW

Clinical Symptom Presentation of ADHF in the ED

The initial evaluation of a patient with symptoms that suggestive of ADHF should first start with a history and physical examination (Onwuanyi, A., & Taylor, M., 2007). History taking can help the provider determine if the person has had a preexisting diagnosis of HF which has the best predictive value for differentiating between pulmonary disease and ADHF. Clinical signs and symptoms of HF are defined as but not limited to: complaints of cough or dypsnea, chest discomfort, abdominal pain, tachypnea, wheezing, pulmonary edema, fatigue, presence of a S3 or S4 gallop, electrocardiographic and heart rhythm changes (e.g. atrial fibrillation), peripheral edema, decreased tissue perfusion, low cardiac output, low ejection fraction, elevated right heart filling pressures, hepatic congestion, and hyper- or hypotension (Kapoor & Perazella, 2007).

Assuring the correct diagnosis of HF is challenge because clinical presentations tend to be neither sensitive nor specific. The chief complaint of dyspnea challenges an accurate diagnosis due to multiple clinical conditions associated with dyspnea. Orthopnea has the highest sensitivity (~90%) for elevated pulmonary capillary wedge pressures but its specificity is low (Kapoor & Perazella, 2007). Jugular venous distension has a specificity of 90% but only a sensitivity of 30%. A S3 gallop, representing rapid ventricular filling, is highly specific but insensitive for HF and can be particularly hard to detect by auscultation in a noisy ED (Chung & Herman, 2006).
Precipitating Factors

Initial evaluation of patients should include fluid volume status, adequacy of circulatory support or perfusion, and presence of precipitating factors and/or co-morbidities (Hunt et al., 2009). Precipitating factors identified by the Heart Failure Society of America (HFSA) 2006 guidelines include: myocardial infarction (MI), coronary artery disease (CAD), acute coronary syndrome (ACS), atrial fibrillation or flutter and other tachy or brady arrhythmias, long QT syndrome, ventricular dyssynchrony, severe hypertension, renal failure, anemia, hypo- or hyperthyroidism, and pulmonary emboli (HFSA, 2006). Other proposed causes of ADHF due to left ventricular dysfunction include cardiac remodeling, myocarditis, postpartum cardiomyopathy, valvular dysfunction, pericardial tamponade or constriction, renal or liver dysfunction causing fluid volume overload, septicemia especially pulmonary in nature, or substance abuse such as with drugs (cocaine) or alcohol (HFSA, 2006).

Diagnostics

Laboratory tests used to diagnose ADHF can include elevated BNP levels, hypoxia and/or hypercapnia on arterial blood gases, low hemoglobin and hematocrit, elevated white blood cell count, elevated cardiac enzymes such as troponin, abnormal liver function tests, abnormal blood urea nitrogen (BUN) and serum creatine, and alterations in natremia or kalemia.

Natriuretic Peptide Levels

BNP or N-terminal pro b-type natriuretic peptide (NT- proBNP) levels have been used in the ED to determine the cause of dyspnea and rales as clinical symptoms and have been shown to be useful in the diagnosis and treatment of ADHF (Collins et al., 2008). BNP is a prohormone that is secreted by the ventricular myocytes in response to increased myocardial stretch (Mayo,
Colletti & Kuo, 2005). The secretion of pre-pro BNP is followed by enzymatic cleavage into biologically active BNP (32 amino acids in length) and the biologically inactive NT-proBNP (76 amino acids in length) (Mayo et al., 2005). BNP is eliminated by two mechanisms: 1) it binds to the natriuretic peptide clearance receptor, followed by cellular uptake where degradation occurs, and 2) it is enzymatically cleaved by neutral endopeptidase, found in endothelial cells, smooth muscle cells, cardiac myocytes, renal epithelium, and fibroblasts (Mayo et al., 2005). These clearance mechanisms account for BNPs relatively short half-life of approximately 20 minutes. NT-proBNP is cleared primarily by the kidney, resulting in a half-life of approximately 60-90 minutes. There was no statistical difference in the diagnostic accuracy of BNP versus NT-proBNP in patients with ADHF symptoms (Clerico, et al., 2007). In patients with renal insufficiency, the half-life of NT-proBNP is increased (Mayo et al., 2005). Most laboratories have the ability to run a BNP level from a blood sample and the result takes approximately 20 minutes.

The level of BNP can correlate with the severity of HF. A normal BNP level is less than 100 pg/mL, indicating no HF. BNP levels of 100-300 pg/mL suggest presence of HF. BNP levels above 300, 600, and 900 pg/mL indicate mild, moderate, and severe HF, respectively. A BNP level > 400 pg/mL has been shown to have a correlation to a New York Heart Association (NYHA) HF classification of II or greater (see table 1 for NYHA classification definition) (Kapoor & Perazella, 2007).

The use of BNP level should be combined with the patient’s clinical presentation of symptoms, past medical history, and other pertinent diagnostic tests such as chest x-ray, electrocardiogram, and echocardiogram. In a patient with a high likelihood of HF, such as
decreased ejection fraction and history of HF, the BNP level may not be needed as a diagnostic tool for an acute exacerbation but it is beneficial to use the number to rate HF severity (Mayo, et al., 2005). Following the trend in BNP level also allows the clinician to evaluate the treatment modality and effectiveness (Bhardwaj & Januzzi, 2009). The ADHF treatment algorithm focuses on a baseline BNP level drawn in the ED. Based on the baseline level admission to the intensive care unit may be warranted (Bhardwaj & Januzzi, 2009). A subsequent BNP level of < 350 pg/mL and resolution of clinical symptoms aids in the discharge criteria (Bhardwaj & Januzzi, 2009). The use of BNP level can aid the clinician in differentiating between pulmonary disease and congestive HF when the only presenting symptom was dyspnea (Morrison et al., 2002; Bhardwaj & Januzzi, 2009).

Maisel et al. (2003) conducted the largest clinical trial supporting the use of BNP levels in the ED which included 1,586 patients presenting with acute dyspnea. In this Breathing Not Properly trial, the physicians rated clinical probability of congestive HF on a 0-100% scale. The result showed that the BNP level accurately correlated with the clinical diagnosis of HF as well as severity of left ventricular dysfunction (Morrison et al., 2002 & Mayo et al., 2005 & Maisel, et al., 2003).

Electrocardiography

The Heart Failure Society of America (HFSA) and the European Society of Cardiology (ESC) recommend a 12-lead electrocardiogram (ECG) in the evaluation of patients with ADHF (Collins et al., 2008). The ECG is recommended for the following reasons: 1) Identification of rhythm abnormalities such as atrial fibrillation, 2) Identification of strain and hypertrophy patterns, 3) Identification of potential treatment options, 4) Detection of ischemia or other ACS,
and 5) Evaluation of other potential causes of ADHF such as sarcoidosis (Collins et al., 2008).

The ECG is also useful in identifying the risk for lethal rhythms such as prolonged QTc and bundle branch blocks, pericarditis by showing diffuse ST segment elevation, or pericardial tamponade by low voltage R waves in the precordial leads. It is useful to have a prior ECG to compare for acute versus chronic changes. ECG findings of left bundle branch block and left ventricular hypertrophy increase the likelihood of systolic dysfunction and, therefore, may have utility in identifying patients with ADHF (Chung & Herman, 2006). The ECG is not used solely but in conjunction with echocardiogram, laboratory tests, and chest radiograph to identify ADHF. Depending on the reason for ADHF (systolic or diastolic dysfunction), ECG changes include but are not limited to new onset bundle branch or fascicular blocks, left ventricular hypertrophy strain pattern, or low voltage R waves. If the cause of ADHF is related to ischemic heart disease ST elevation and/or depression may be seen on ECG tracings (Chung & Herman, 2006).

Echocardiogram

The ADHF treatment task force recommends obtaining an echocardiogram for accurate diagnosis and treatment of ADHF (Niemnen, et al., 2005). The echocardiogram can be obtained in the ED by the transthoracic approach (TTE); however, if a clearer view of the valves is needed, a transesophageal echocardiogram (TEE) is warranted. A TTE can view regional and global right and left wall motion abnormalities, valvular structure and function, right and/or left systolic or diastolic dysfunction, calculate an ejection fraction (EF) and cardiac output, evaluate for pericarditis, cardiac tamponade, thrombus formation or other space occupying lesion, and estimate pulmonary artery pressures (Collins, et al., 2008; Niemnen, et al., 2005).
**Chest Radiograph**

The chest radiograph or chest x-ray has been widely as a diagnostic tool in a patient presented to the ED with dyspnea. Chest x-ray can be used to identify pneumonia, pulmonary edema, or other thoracic abnormalities. A chest x-ray is highly specific (96%, 98%, and 99%) but not sensitive (41%, 27%, and 6%) in detecting signs of congestion in ADHF (cephalization, interstitial edema, and alveolar edema, respectively) (Collins et al., 2008). The low sensitivity makes chest x-ray a poor screening tool for ADHF (Collins et al., 2008). The chest x-ray findings are usually a late sign of congestion for ADHF and should be used primarily to rule out other causes of dyspnea such as pneumonia.

**Diagnostics Summary**

The identification and subsequent diagnosis of ADHF is multifaceted and is based on clinical presenting symptoms and diagnostic tests. ADHF presentation in the ED can manifest as new onset pulmonary edema or an exacerbation of an underlying condition. The most common complaint is dyspnea without exertion. Diagnostic tests include chest x-ray, echocardiogram, BNP level, and ECG. Early and accurate diagnosis of ADHF in the ED is pertinent to initiating early treatment.
Treatments

Treatment Goals

Treatment goals of ADHF vary based on symptom presentation. In patients with pulmonary edema, dyspnea, congestion, and signs of fluid overload, the goal is to decrease fluid volume and ease the work of breathing (Niemen, Bohm, Cowie, Drexler, Filippatos, et al., 2005). Initial treatment goals are to decrease symptoms, such as dyspnea and/or fatigue, maximize optimal oxygenation by keeping saturations > 95%, obtain hemodynamic stability such as mean arterial pressure (MAP) > 60mmHg and systolic blood pressure (SBP) > 90mmHg, and obtain baseline BNP level. Intermediate treatment goals are to maintain pulmonary capillary wedge pressure (PCWP) at around 18mmHg and maintain adequate cardiac output and/or stroke volume (Niemen, et al., 2005). Long term goals for discharge include decreased body weight with diuresis or ultrafiltration, resolved dyspnea, hemodynamic stability, serum electrolyte normalization, reduction of BNP level, and ultimately decreased morbidity and mortality and length of time in hospital readmission (Niemen, et al., 2005).

Diuretics

The 2009 American College of Cardiology Foundation and American Heart Association (ACCF/AHA) task force developed a focused update to the 2005 practice guidelines to include the diagnosis and treatment of ADHF (Jessup, et al., 2009). Fluid overload is defined as > 10% over baseline bodyweight, or evidence of edema, auscultated lung crackles and pulmonary edema (Cereda, Sheinfeld, & Ronco, 2010). The use of diuretics, the most common being furosemide, is considered the standard of care in patients with volume overload ADHF (Cleland, Coletta & Witte, 2006). In a study by Alan Maisel, et al. (2008), early intervention of diuretic
therapy in the ED provided better outcomes in patients with ADHF (Maisel, et al., 2008).

Furosemide given intravenously (IV) has the onset of 5 minutes with duration of approximately 2 hours. The use of a loop diuretic in ADHF has the benefit of quickly reducing the fluid volume status, preload, and pulmonary edema that is associated with fluid overload. Some of the risks of using a loop diuretic include hypotension, reduction of volume at too high of a rate, and worsening renal function (Jessup, et al., 2009). The goal for diuretic therapy should be a steady reduction in volume status with minimal side effects.

Diuretics differ in metabolism and mechanism of action. Therefore, assessing renal function is beneficial in determining which loop diuretic to use. Furosemide is excreted and metabolized by the kidneys (Cleland, et al., 2006). Bumetanide and torsemide are both excreted by the liver and their half-lives are prolonged by patients with liver disease but not unaffected by renal insufficiency (Cleland, et al., 2006).

The efficacy of loop diuretics is directly related to creatinine clearance; in low clearance states, the dose of diuretic needs to be increased to produce natriuresis and adequate diuresis (Cleland et al., 2006). The suggested dosage for furosemide IV push is 20-40 mg or double the home dosage (Maisel, 2003). Furosemide should be prescribed at 20-40mg 2-4 times daily in patients with new onset ADHF or without maintenance diuretic therapy and creatinine clearance > 60mL/min (Ezekowitz et al., 2009). If creatinine clearance is < 60mL/min, furosemide prescription should be 20-80mg 2-3 times daily (Ezekowitz et al., 2009). In continuous infusion, furosemide dose is titrated based on a desired urine output. For patients with normal renal function, the goal urine output is ≥ 500 ml in the first 2 hours. An acceptable urine output for
patients with serum creatinine $\geq 2.5 \text{ mg/dL}$ is $> 250 \text{ ml}$ in the first 2 hours (DiDomenico, et al., 2004).

If the desired response is not reached with a loop diuretic, then a thiazide diuretic should be administered. The common thiazide diuretic in IV form is chlorothiazide. The side effects are similar to that of furosemide. Volume status as well as electrolyte abnormalities should be monitored (Jessup, 2009).

**Ultrafiltration**

Diuretics have the ability to remove excess volume from the body by increasing diuresis through the renal system. In the ADHF patient with hypervolemia and renal insufficiency, the use of diuretics may not work. If all diuretic strategies are unsuccessful, extracorporeal ultrafiltration can be used (Costanzo, et al., 2007 qtd. in Jessup, et al., 2009, & Hunt, et al., 2009). Ultrafiltration moves water and small to medium-weight solutes across a semipermeable membrane to reduce volume overload (Costanzo, et al., 2007). Relatively more sodium can be removed by ultrafiltration than by diuretics (Jessup, et al., 2009). Water and electrolytes such as sodium are simultaneously moved across the membrane, therefore, the electrolyte concentration of the ultrafiltrate is similar to that of blood plasma. This avoids sudden shifts in electrolyte concentrations and results in more sodium removal that would be achieved than with the use of diuretics (Hill, Yancy & Abraham, 2006). Because of this, monitoring electrolyte balance is critical with the use of ultrafiltration.

The use of ultrafiltration does not necessitate admission to an intensive care unit (ICU) or specialized nursing care of technical oversight (Hill, et al., 2006). This allows for decreased hospital admission costs. Minimal data have been published on the efficacy and decrease in
morbidity or mortality with the use of ultrafiltration. A recent study, Ultrafiltration versus Intravenous Diuretics for Patients Hospitalized for Acute Decompensated Congestive Heart Failure (UNLOAD), included 200 patients with ADHF, randomized to treatment with peripheral ultrafiltration or standard IV diuretic therapy (Constanzo, et al., 2007, qtd in Hill, et al., 2006). Ultrafiltration was shown to safely produce greater weight and fluid loss than IV diuretics and can be an effective alternative to IV diuretics (Costanzo, et al., 2007). In addition, fewer patients receiving ultrafiltration required rescue therapy with vasoactive drugs than IV diuretics. Ultrafiltration was not associated with hypokalemia or adverse changes in serum creatinine (Hill, et al., 2006).

The use of extracorporeal ultrafiltration does have risks and potential side effects. Extracorporeal ultrafiltration can result in hemorrhage from using systemic anticoagulation and catheter-related complications such as infection. Excess ultrafiltration can result in hemorrhage from anticoagulation, hypotension, worsening renal function, and membrane bio-incompatibility (Brandimarte, F., Mureddu, Boccanelli, Cacciatore, Brandimarte, C., et al, 2010). Membrane bio-incompatibility is caused by the ultrafiltration membrane reacting with the patients blood causing an inflammatory response. It is important to note that ultrafiltration cannot be used as a substitution to dialysis in patients with renal insufficiency, uremia, or metabolic abnormalities (Hill et al., 2006).

Inotrope Therapy

In patients with low cardiac output states, inotrope support should be considered. Dobutamine and milrinone are recommended inotropes for treatment of ADHF with low cardiac output states by the HFSA and ACC/AHA guidelines. Dobutamine and milrinone have inotrope,
chronotrope, and systemic and pulmonary vasodilator effects (Maisel et al., 2009). IV dobutamine and milrinone may be used to relieve the symptoms and improve end-organ function in patients with advanced HF characterized by left ventricle (LV) dilation, reduced LVEF, marginal SBP (less than 90mmHg), symptomatic hypotension despite adequate filling pressure, low cardiac output states, and elevated PCWP (HFSA, 2006).

Dobutamine is a synthetic catecholamine with mainly beta 1 receptor agonist and some beta 2 receptor activity (Maisel, 2003). This means dobutamine causes increased heart contractility and cardiac output. The actions of dobutamine should be considered in patients who are already on beta blocking agents (Hunt et al., 2009) because the effect of dobutamine is hindered until beta-blocker is removed from circulation.

Milrinone, a phosphodiesterase III inhibitor, produces elevated levels of cyclic adenosine monophosphate (cAMP) in the myocardium and smooth muscle, which leads to increased cardiac contractility and vasodilation (Maisel, 2003). Milrinone has hemodynamic changes similar to dobutamine; however, due to the different cellular signaling pathways, it can be used simultaneously with catecholaminergic agonists or antagonists.

The Outcomes of Prospective Trial of Intravenous Milrinone for Exacerbations of Chronic Heart Failure (OPTIME–CHF) included 951 patients admitted to the hospital with hemodynamically stable exacerbations of systolic HF (Maisel, 2003 & Strain, 2004). Within 48 hours after hospital admission, patients were randomly assigned to receive either a 48-72 hour infusion of milrinone or placebo. The outcomes showed that milrinone had an increased rate of mortality and no statistical difference in the total hospital days as compared to placebo (Maisel,
2003). Milrinone was associated with a higher rate of early treatment failure, more sustained hypotension, new atrial arrhythmias, and a trend toward higher in-hospital mortality (Dec, 2007).

Inotropic agents work by increasing cardiac output via increasing the force of contraction, and hence increasing adenosine triphosphate (ATP) and oxygen consumption leading to increased risk of fatal dysrhythmias (Strain, 2004). Milrinone has been associated with worsening of HF, new atrial arrhythmias and a trend toward increased in-hospital and 60 day mortality (Strain, 2004; HFSA, 2006 & Dec, 2007). Dobutamine and milrinone can be lifesaving for patients with rapidly progressive hemodynamic collapse by increasing cardiac contractility and providing improved cardiac output (Dec, 2007). Patients who present with obtundation, anuria, and/or lactic acidosis may only respond to inotropic therapy or mechanical circulatory support, which should be continued until the cause of shock is determined and definitive therapy is implemented (Dec, 2007).

Positive inotropic agents should be reserved for patients with refractory hypotension, cardiogenic shock, end-organ dysfunction, or failure to respond to conventional oral and/or IV diuretics and vasodilators where the benefit of an inotrope outweighs the risk (HFSA, 2006). The current practice guidelines of ACC/AHA accept the use of IV inotropic support for stage D HF patients (i.e. refractory symptoms) as palliative treatment or as a bridge to cardiac transplant but only after all therapies to achieve stability have failed (Hunt et al., 2006 qtd. in Dec, 2007). Dobutamine and milrinone may be effective in low cardiac output states, where reduced renal function may improve with inotropic support. Patients with cardiogenic shock may need inotropes to maintain the minimal cardiac output necessary for survival (Strain, 2004 & HFSA, 2006). In patients with hypotension and low cardiac output states, dobutamine is preferred over
milrinone because it does not cause hypotension. Since milrinone can cause hypotension, it can be used in relatively normotensive low cardiac output states or when a patient has had beta-blocker therapy. Dobutamine works on the beta adrenergic sites thus limiting its effects or not working at all in patients receiving beta blockers (DiDomenico, et al., 2004). In the hypotensive low cardiac output state, a combination of milrinone and dobutamine may be needed to obtain enough cardiac output to sustain life, or to bridge to alternative treatment such as assistive mechanical device (HFSA, 2006 & Strain, 2004).

**Vaspressors**

Vaspressors, such as dopamine, should be considered in the ADHF patient with hypotensive shock for adequate perfusion and normalization of blood pressure. Dopamine has inotrope, chronotrope and vasoconstrictor effects (Maisel, 2003). Typically, this agent should be avoided in HF with high systemic vascular resistance (SVR), but such resistance may be low in ADHF owing to activation of systemic inflammatory response or circulatory collapse (Maisel, 2003).

**Vasodilators**

Nesiritide, nitroglycerin, and sodium nitroprusside (SNP) are the IV vasodilators of choice for the treatment of ADHF with adequate blood pressure (Hunt et al., 2009). Vasodilators work to decrease the preload and/or afterload to quickly reduce pulmonary congestion. In the hypertensive ADHF patient, the role of vasodilators is critical in decreasing the blood pressure to allow for adequate perfusion.

Nitroglycerin also has an anti-ischemic effect with coronary vasodilation which is beneficial in ischemic cardiomyopathy. Nitroglycerin can be administered sublingually,
topically, orally, and IV after sublingual administration. IV administration of nitroglycerin is the route of choice in ADHF patients due to its rapid bioavailability (Maisel, 2003).

SNP is a potent vasodilator used for ADHF and hypertensive urgency/emergency (Ezekowitz et al., 2009). SNP works as a venodilator to reduce preload and an arterial dilator to reduce afterload. Nitroglycerin is often the initial drug of choice in the ED because of its slightly longer half life than SNP thus avoiding the profound hypotension that SNP can cause without invasive monitor such as an arterial line (Hunt et al., 2009). SNP has the benefit of rapidly reducing the blood pressure. Care should be taken in titration of SNP, it should be increased at intervals no more frequently than 10 minutes (0.3-3mcg/kg/min) and must be tapered down to avoid rebound effect (Ezekowitz et al., 2009). Another side effect of SNP after prolonged infusion is toxicity of its metabolites, thiocyanate, and cyanide and should be avoided in patients with severe renal or hepatic failure (Ezekowitz et al., 2009).

Nesiritide or recombinant human BNP acts by increasing cyclic guanosine monophosphate (cGMP) and is used to reduce LV filling pressure but has variable effects on cardiac output, urinary output, and sodium excretion (Dec, 2007 & Maisel, 2003). It acts as a vasodilator in both arterial and venous circulation and leads to decreased systemic vascular resistance (SVR), resulting in reduced systemic arterial pressure and mean pulmonary artery pressures without reflex tachycardia (Strain, 2004). It also increases cardiac output through its effects on ventricular afterload, without negative effects on creatinine clearance or potassium excretion like a diuretic would. In patients receiving nesiritide, mild to moderate hypotension may occur due to increased sodium excretion and decreased aldosterone and adrenaline levels (Strain, 2004 & Peacock et al., 2007). These side effects can be controlled with dose
adjustments. The initial dose of nesiritide starts with a bolus, usually 2 mcg/kg followed by an infusion of 0.01mcg/kg/min with a max dose of 0.03mcg/kg/min (Maisel, 2003). The severity of dyspnea from volume overload is reduced more rapidly by natriuresis than diuretics alone (Hunt et al., 2009).

In the Vasodilation in the Management of Acute CHF (VMAC) trial, 498 patients with NYHA class IV acute HF were randomly assigned to receive nesiritide, IV nitroglycerin, or placebo as treatment options. The results demonstrated that nesiritide was most effective in reducing pulmonary capillary wedge pressure at three hours after infusion, however, there was no significant difference at 48hrs (Maisel, 2003). Hypotension was more common in the nesiritide group, likely due to its long half-life, in comparison to nitroglycerin. Nesiritide was, however, associated with shorter hospital LOS compared to IV nitrates, and a lower cost of care (Strain, 2004).

The Acute Study of Clinical Effectiveness of Nesiritide in subjects with Decompensated Heart Failure (ASCEND-HF) trial is an ongoing double-blind placebo-controlled multicenter study to evaluate whether treatment with nesiritide improves patient outcomes or HF symptoms compared with placebo when each is administered in addition to other standard therapies in subjects with ADHF (Russell et al, 2008 qtd in Ezekowitz et al., 2009). Patients hospitalized for HF will be randomly assigned to receive either intravenous nesiritide or placebo for 24 hours to 7 days. The two primary end points are (1) assessment of acute dyspnea at 6 or 24 hours and (2) death or rehospitalization for HF within 30 days (Hernandez et al., 2009). The trial is expected to complete in 2010 once 7,000 patients have been enrolled (Hernandez et al., 2009).
Invasive Hemodynamic Monitoring

Hemodynamic monitoring should be considered for use in patients with uncertain volume status or filling pressures or who are refractory to initial therapy (Hunt et al., 2009). These patients include: 1. presumed cardiogenic shock requiring escalating vasopressor therapy and consideration of mechanical support; 2. severe clinical decompensation in which therapy is limited by uncertainty regarding relative contributions of elevated filling pressures, hypoperfusion, and vascular tone; 3. apparent dependence on IV inotropic infusions after initial clinical improvement; or 4. persistent severe symptoms despite adjustment of recommended therapies (Hunt et al., 2009).

Treatment Pathways

Early diagnosis and treatment of ADHF should start in the ED. Potential patients with ADHF should undergo two levels of triage and concurrent treatment as diagnostic testing occurs (Collins et al., 2008). The initial triage should focus on stabilization of the extremes and immediate intervention to prevent further deterioration. Patients with tachycapnea, hypoxia, and mental status changes may need non invasive positive pressure ventilation or endotracheal intubation initially while definitive care is being established (Collins et al., 2008). Figure 1 highlights the initial diagnosis and treatment plan for patients with ADHF presenting to the ED (Collins et al., 2008). Once initial triage has occurred, secondary triage of hemodynamic stability into hypotensive, hypertensive, and normotensive should occur to help guide the medication and treatment regimen in the ED (Collins et al., 2008).
**Hypotensive Pathway**

Patient with hypotensive ADHF have clinical findings of low cardiac output, end-organ hypoperfusion, and/or cardiogenic shock, such as decreased perfusion, altered mental status, reduced urine output, cool extremities, and systolic blood pressure less than 90mmHg (Collins et al., 2008). Treatment with hypovolemic hypotension should start with a fluid bolus (Figure 2). Vasopressors, such as norepinephrine or dopamine, as well as IV intropes, such as milrinone or dobutamine, should be initiated (Collins et al., 2008). Patients presented with initial hypotension have an increased chance of recurrent drop in BP and thus should be admitted to an intensive care unit (Collins et al., 2008). Once hemodynamic stability is obtained, invasive monitoring such as arterial line or pulmonary artery catheter should be considered.

**Hypertensive Pathway**

Approximately 50% of patients with ADHF present to the ED with a SBP of more than 140 mmHg. These patients tend to be older, are women, have history of diastolic dysfunction, have symptoms of hypertension, and are in pulmonary edema for 24-48 hours prior to entering the ED (Collins et al., 2008). Pulmonary edema from ongoing hypertension and poor LV function can increase afterload. Initial BP control should include sublingual or topical nitroglycerin followed by IV nitroglycerin (Collins et al., 2008) (Figure 3). An IV vasodilator, such as nitroglycerin, SNP, or nesiritide, can be used in patients who remain hypertensive > 160/100mmHg. Treatment of hypertensive ADHF should focus on aggressive BP control and minimize diuretic use. Due to maldistribution of fluids, a diuretic can result in hypotension and renal dysfunction (Collins et al., 2008). Once antihypertensive is administered and blood pressure becomes normalized then assessment for diuretic administration should be considered.
(Collins et al., 2008). After initial blood pressure control and mild diuresis, hypertensive ADHF patients tend to have dramatic symptom improvement (Collins et al., 2008).

**Normotensive**

Approximately 35% of patients with ADHF presents with mild, subacute worsening of their symptoms over several days to weeks. These patients tend to be younger and have systolic dysfunction and a history of coronary artery disease (Collins et al., 2008). Normotensive is defined as systolic blood pressure ranging from 90-140mmHg (Collins et al., 2008). Due to the gradual worsening of symptoms, patients initially benefit from aggressive diuresis to relieve symptoms of congestion and reducing total body fluid and peripheral edema (Collins et al., 2008). Normotensive ADHF patients may respond well to initial diuresis depending on renal function, therefore, they will need to be reevaluated after diuretic therapy to monitor for hypotension. If hypotension occurs, then the patient should be managed according to the hypotensive rather than the normotensive pathway (Figure 4) (Collins et al., 2008). Patients with a previous history of HF who are seen in the ED for an acute decompensation can be medically managed in an observation unit or on a sub-acute telemetry unit and then sent home 24 hrs later once medication regimen has been optimized (Collins et al., 2008). Those patients with no improvement or minimal improvement to initial therapy will need to be admitted to the intensive care unit for additional invasive and/or non-invasive intervention (Collins et al., 2008).

**Treatment Summary**

ADHF should be treated based on the clinical symptoms, ventricular function, and cardiac output states. The recommendations for treating ADHF are to follow HFSA, ACCF/AHA evidence based algorithms and begin initiating treatment early in the ED when diagnosis has
been suspected or confirmed. Nesiritide has been shown to have improved outcomes and symptoms within the first 24 hours of administration and has the highest benefit if started in the ED (Peacock et al., 2007 & Strain, 2004). Inotropic agents, such as dobutamine and milrinone, are frequently used in the intensive care setting for the management of ADHF only in patients with cardiogenic shock, low cardiac output states, and evidence of end-organ perfusion. The purpose of using an inotrope in the management of ADHF is to improve cardiac output as a life-saving measure, palliative symptom management, or a bridge to mechanical device or transplant.
CHAPTER III CONCLUSION

Significance for the Advance Practice Nurse

The number of nurse practitioners in the ED is growing. According to the Health Resources and Services Administration (HRSA), the number of nurse practitioners increased significantly in virtually every state from 1992 to 2000. The overall increase was 160 percent over the eight year period (Bureau of health professions, 2001). The responsibility of diagnosing and treating ADHF falls on emergency care providers. Healthcare providers such as physicians and nurse practitioners have the increasing responsibility to recognize and treat ADHF by following the current national guidelines. Early recognition of symptoms and early treatment of ADHF can contribute to better overall outcomes, decrease morbidity and mortality, decrease length of stay, and decrease readmission rates.

Summary

The Advance Practice Nurse (APN) has been trained in diagnosis and treatment of acute and chronic illnesses in a variety of hospital settings. The diagnostic tools used to identify ADHF such as ECG, chest x-ray, laboratory values, and thorough history and clinical assessment are all components of the APN’s scope of practice. With the growing number of ADHF patients and the increasing number of APN’s in the ED and hospital, it is vital for APNs to have current knowledge on evidence based guidelines when treating patients with acute and complex illnesses, such as ADHF. ADHF patients should be managed according to hypotensive, normotensive, and/or hypertensive algorithms. Future studies are needed to assess if these treatment algorithms are adhered for treating ADHF in the ED and/or ICU settings.
LIST OF ILLUSTRATIONS

FIGURE 1: Suggested initial treatment pathway for Acute Heart Failure Syndrome

FIGURE 2: Suggested treatment algorithm for hypotensive acute heart failure syndromes

FIGURE 3: Suggested treatment algorithm for hypertensive acute heart failure syndromes

FIGURE 4: Suggested treatment algorithm for normotensive acute heart failure syndromes

TABLE 1. New York Heart Association Heart Failure Functional Classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Patient Symptoms</th>
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<tbody>
<tr>
<td>Class I (Mild)</td>
<td>No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea (shortness of breath).</td>
</tr>
<tr>
<td>Class II (Mild)</td>
<td>Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.</td>
</tr>
<tr>
<td>Class III (Moderate)</td>
<td>Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.</td>
</tr>
<tr>
<td>Class IV (Severe)</td>
<td>Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.</td>
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Heart Failure Society of America (2002)
REFERENCES


