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**DEVELOPMENT OF THE ACUTE DECOMPENSATED HEART FAILURE RISK
MODEL FOR EMERGENCY ROOM RESIDENT TRAINING**

by

ZORA INJIC

DISSERTATION

Submitted to the Graduate School

of Wayne State University,

Detroit, Michigan

in partial fulfillment of the requirements

for the degree of

DOCTOR OF PHILOSOPHY

2016

MAJOR: EDUCATION EVALUATION AND
RESEARCH

Approved By:

Advisor

Date

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DEDICATION

This dissertation is dedicated to my family. My husband Miro of 32 years who agreed to support me in my pursuit of education never knowing my journey would take me this far.

To my children, Kristina and Anthony who walked me through each step of my doctoral coursework and dissertation. Each of my professors saw my children waiting each night

after they completed classes. I cannot begin to tell you how proud I am of their educational achievements and the wonderful accomplished adults they have become.

My parents who came from villages and instilled in me the importance of education – I wish they were here to see yet another graduation.

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Dr. Sawilowsky, I don't know where to begin. His stories in class always painted a picture so that his teaching would live on. I still remember receiving my first assignment from him with his note stating I had written the best critique of an article in the class by far. How could I not believe I could conduct a literature review for my dissertation after receiving such support? Without his encouragement, I would have never taken my written exams. Without his inspirational and gentle reminders, I would have left my paper in an electronic file yet to be done. When the definition of a mentor was written, although before his time, it was written with Dr. Sawilowsky as the ideal example.

To all current and future doctoral students, use whatever resources you have to complete your dissertation. Not only will you contribute to the literature but you will accomplish a great feat.

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If three simple questions and one well chosen laboratory test lead to an unambiguous diagnosis, why harry the patient with more?

Anonymous

CHAPTER 1 INTRODUCTION

Cardiovascular disease is the primary cause of death in the United States (U.S.). As the leading cause of cardiovascular morbidity, heart failure is a clinical syndrome with an estimated U.S. prevalence of 5.8 million adults and an incidence of 690,000 new cases annually. Acute decompensated heart failure (ADHF) accounts for nearly 2 million hospitalizations per year, remaining relatively unchanged from 1999 to 2009 in spite of new treatment modalities and improved understanding of the heterogeneity of these patients.

Post hospital discharge mortality and readmission rates are unacceptably high, reaching 10-20% and 20-30%, within 3 and 6 months (Abraham et al., 2008; Gheorghiade & Pang, 2009; Gheorghiade, Vaduganathan, Fonarow, & Bonow, 2013; Lee et al., 2003). Hospital readmission is defined as two or more consecutive admissions within a certain period of time. The initial hospitalization with the primary discharge of heart failure is termed the index hospitalization. The Centers for Medicare and Medicaid Services (CMS) requires public reporting of all-cause hospital readmissions within 30 days in patients with a primary diagnosis of heart failure at the index hospitalization. According to the Agency for Healthcare Research and Quality (n.d.), "Readmission of patients who were recently discharged after hospitalization with HF represents an important, expensive and often preventable adverse outcome" (para. 3). Readmission within 2, 3 and 6 months are also used for measuring clinical outcomes and used in the literature reviewed (Chin, 1997; Gheorghiade & Pang, 2009; A.F. Hernandez et al., 2010; Krumholz et al., 2000; Michtalik et al., 2011; Philbin & DiSalvo, 1999; Rich et al., 1995; Ross et al., 2009). According to the 2008 National Center for Health Statistics (NCHS), heart failure accounts for 35% of

cardiovascular disease deaths with nearly 50% of those diagnosed dying within the first five years (Bonow et al., 2012; Roger et al., 2012). It is not surprising that hospitalizations from to heart failure exceed those due to the total number of all forms of cancer (American Heart Association, 2012).

The economic burden of morbidity and mortality associated with the treatment of heart failure must be addressed in light of the spotlight placed on healthcare costs (Dunlay et al., 2011; Gheorghiade & Pang, 2009). In response, an increased number of drug trials investigating various approaches to treat patients with heart failure were sponsored by industry and governmental agencies over the last decade (Ahmed, Aronow, & Fleg, 2006; Burger et al., 2002; Felker et al., 2010; Follath et al., 2002; McDonagh et al., 2011; Morris, Hatcher, & Reddy, 2006; O'Connor et al., 2011; Publow & Branam; Simpson, Noble, & Goa, 2002). However, many of these trials exclude patients with low systolic blood pressure either directly, or indirectly. Low systolic blood pressure is often an exclusion criterion in drug studies. Indirectly, standard medications used to decrease blood pressure may be contraindicated in patients with low systolic blood pressure who are alternately treated with exclusionary medications. In addition, standard medications used to treat heart failure listed as part of the inclusion criteria are contraindicated in patients with low blood pressure. Patients admitted for acute decompensated heart failure with low systolic blood pressure have higher mortality rates during hospitalization and post-discharge than those with normal or high systolic blood pressure (Ambrosy et al., 2013; Felker et al., 2010; Franklin & Levy, 2011; Gheorghiade et al., 2012). Associations between hypertension and cardiovascular conditions such as heart failure and stroke abound in the literature (Psaty, Lumley, Furberg, & et al., 2003; Turnbull, 2003). However,

there is paucity of data characterizing the patient with low systolic blood pressure and heart failure and thus, this was the basis of this retrospective study conducted at a large urban transplant center.

Exploratory Factor Analysis (EFA) Approach

Quasi-experimental designed research typically involves advanced statistical procedures due to the lack of randomization found in the true experimental design. Patients selected for multicentered randomized clinical trials exhibit a different, often better, treatment response than those patients who are not enrolled (Badano, 2003; Rothwell 2005). Instead of the traditional gold standard randomized clinical trial, nonrandomized prospective and retrospective observational studies are becoming a tool used to investigate practical outcomes of a treatment in standard clinical practice. Entry criteria allow for a broader, less homogenous patient sample than do randomized clinical trials. This may improve generalizability of the results. In addition, current evidence-based therapies may be continued to allow for the best care of a patient and adherence to the Centers for Medicare and Medicaid Services (CMS) reimbursement guidelines. EFA can be performed on retrospectively collected data. In EFA, “A factor is a construct, a hypothetical entity, a latent variable that is assumed to underlie tests, scales items, and indeed, measure of almost any kind” (Hair, Tatham, Anderson, & Black, 2006, p. 826). The resulting factors can then be tested using more expensive experimental manipulation.

Although developed in 1904 by Charles Spearman, the father of modern factor analysis is considered Louis L. Thurstone who authored the 1947 classic historical work, *Multiple Factor Analysis* (Hair et al., 2006; Tabachnick & Fidell, 2013; Thurstone, 1931).

“Traditionally, factor analysis has been used to explore the possible underlying structure in a set of interrelated variables without imposing any preconceived structure on the outcome” (Child, 1990, p. 6). EFA is a mathematical technique used for theory development or data reduction as depicted in Figures 1 and 4 below (Hair et al., 2006). In theory development, EFA can detect the structure in the relationships between variables thereby classifying the variables. In data reduction, redundant and irrelevant variables are removed (Hair, Anderson, Tatham, & Black, 1992; Tabachnick & Fidell, 2013). In the heart failure literature reviewed, a variety of statistical approaches including multivariate analysis are used to develop risk scores or survival rates (Kannel et al., 1999; Sayers et al., 2007; Zugck et al., 2001). However, the use of factor analysis is limited to initial psychometric instrument development or assessment of construct validity when applied to a specific target group. The Minnesota Living with Heart Failure Questionnaire and Kansas City Cardiomyopathy Questionnaire used in research and the inpatient and outpatient clinical practice are examples of such instruments (Green, Porter, Bresnahan, & Spertus, 2000; Naveiro-Rilo et al., 2010).

$$R = P\Phi P^T + U$$

where

R = observed data

Correlation matrix: P matrix = factor loading matrix

P^T matrix = transpose of P (transformed matrix)

Φ matrix = correlation between factors

U = uniqueness within each variable

Figure 1. Fundamental Equation of Factor Analysis (Hair et al., 2006)

$$R' = P\Phi P^T + U$$

where

R' = reproduced correlation matrix

Solution for P, Φ , and U where R' and R differ by a small amount

Figure 2. Factor Analysis Equation (Hair et al., 2006)

Statement of the Problem

The problem addressed in this study was to determine if there are factors that characterize patients hospitalized with acute decompensated heart failure with and without low systolic blood pressure. Did the use of a variable reduction technique such as EFA determine the number of latent constructs in these groups of patients? The choice of variables collected were based on those used in the literature reviewed, Framingham criteria (see Framingham Heart Study in chapter 2), assessments used for standard clinical care at this study site, and others for exploratory evaluation. There were a select few variables found in the literature that could not be captured in this retrospective chart

abstraction study. Examples are quality of life questionnaires or certain serum laboratory results that were either not ordered or are not available.

American College of Cardiology (ACC) and American Heart Association (AHA) guidelines, hospital specific practices and the clinical judgment of the physician are used to treat patients with acute decompensated heart who present in the emergency room. Many decisions based on commonly collected assessments in the emergency room must be made quickly and treatment initiated early. However, many of these therapeutic measures are contraindicated in patients whose blood pressure is low at presentation or chronically low (Abraham et al., 2008; Buiciuc et al., 2011; Gheorghiade et al., 2006; Miller & Skouri, 2009). These patients are considered sicker and are treated with drugs such as intravenously administered inotropes to improve cardiac output. Yet these same drugs are associated with increased morbidity and mortality. In addition, invasive monitoring is more common in these patients. Database analysis of several heart failure registries support improved outcomes when treatment is initiated early upon presentation in the emergency room (Abraham et al., 2008; Mebazaa et al., 2008; Subbe, Kruger, Rutherford, & Gemmel, 2001). Thus a quick risk stratification system to score patients presenting with acute decompensated heart failure has the potential to lead to effective individualized treatment modalities.

Purpose of the Study

The purpose of this study was to characterize patients hospitalized with acute decompensated heart failure with and without low systolic blood pressure using EFA. Patients requiring hospitalization due to acute decompensated heart failure present with distinct clinical characteristics including severe symptoms that necessitate rapid

treatment that cannot be delivered in an outpatient setting. Response to treatments varies, and those requiring invasive diagnostic procedures, coronary interventions or surgical treatments meet the guidelines for admission. Each patient presents with clinical signs and symptoms, a heart failure history, and comorbidities. Direct and surrogate measurements are then measured. Safety and cost-effective management is paramount, but there is a deluge of clinical data. The question investigated was could the method of data reduction using EFA elicit a parsimonious group of factors to summarize the relationship between these variables?

A better understanding of the characteristics and outcomes of patients presented with acute decompensated heart failure with and without low systolic blood pressure could potentially lead to individualized treatment modalities tailored to effectively and economically improve care.

Research Questions

1. What were the common factors in patients with acute decompensated heart failure?
2. What were the common factors in patients with acute decompensated heart failure and normal or high systolic blood pressure?
3. What were the common factors in patients with acute decompensated heart failure and low systolic blood pressure?
4. Was there a difference in the common factors in patients with and without low systolic blood pressure and acute decompensate heart failure?

Operational Definitions *(add as needed, definitions of signs and symptoms or other technical terms will be summarized in the appendices)*

Acute decompensated heart failure: Acute heart failure syndrome is defined as gradual or rapid change in heart failure (HF) signs and symptoms resulting in a need for urgent therapy. Acute heart failure syndromes encompasses at least 3 clinical distinct entities: (1) Worsening chronic HF associated with reduced or preserved LVEF (70% of all admissions); (2) de novo HF (e.g., after a large MI; sudden increase in blood pressure superimposed on a noncompliant LV) (25% of all admissions); and (3) advanced HF (i.e., refractory to therapy) with severe LV systolic dysfunction, associated with a continually worsening low-output state (5% of all admissions) (Gheorghiade et al., 2005, pp. 3594-3959).

Blood pressure: pressure exerted by the blood upon the walls of the blood vessels and especially arteries, usually measured on the radial artery by means of a sphygmomanometer, and expressed in millimeters of mercury either as a fraction having as numerator the maximum pressure that follows systole of the left ventricle of the heart and as denominator the minimum pressure that accompanies cardiac diastole or as a whole number representing the first value only a blood pressure of 120/80—abbreviation BP. Diastolic blood pressure is the lowest arterial blood pressure of a cardiac cycle occurring during diastole of the heart and systolic blood pressure is the highest arterial blood pressure of a cardiac cycle occurring immediately after systole of the left ventricle of the heart (Merriam-Webster, n.d.).

Heart failure: Heart failure is a syndrome caused by cardiac dysfunction, generally resulting from myocardial muscle dysfunction or loss and characterized by left ventricular dilation or hypertrophy [or both]. Whether the dysfunction is primarily systolic or diastolic or mixed, it leads to neurohormonal and circulatory abnormalities, usually resulting in

characteristic symptoms such as fluid retention, shortness of breath, and fatigue, especially on exertion. In the absence of appropriate therapeutic intervention, heart failure is usually progressive at the level of both cardiac function and clinical symptoms. The severity of clinical symptoms may vary substantially during the course of the disease process and may not correlate with changes in underlying cardiac function. Although heart failure is progressive and often fatal, patients can be stabilized and myocardial dysfunction and remodeling may improve, either spontaneously or as a consequence of therapy. In physiologic terms, heart failure is a syndrome characterized by elevated cardiac filling pressure or inadequate peripheral oxygen delivery, at rest or during stress, caused by cardiac dysfunction (Lindenfeld & Arnold, 2006, p. e10).

Heart failure with preserved ejection fraction: Also referred to as heart failure with a nondilated left ventricle. A clinical syndrome characterized by signs and symptoms of heart failure with preserved left ventricular ejection fraction. Most commonly associated with a nondilated left ventricular chamber. May be the results of valvular disease or other causes (Lindenfeld & Arnold, 2006, p. e10).

Heart failure with reduced ejection fraction: Also referred to as heart failure with a dilated left ventricle. A clinical syndrome characterized by signs and symptoms of heart failure and reduced left ventricular ejection fraction. Most commonly associated with left ventricular chamber dilation (Lindenfeld & Arnold, 2006, p. e10).

International Classification of Diseases, Ninth Revision, Clinical Modification code (ICD-9) - code and classify morbidity data from the inpatient and outpatient records, physician offices, and most National Center for Health Statistics (NCHS) surveys (n.d., para 3).

Statistical Definitions

The following statistical definitions were taken from Hair et al. (1992, p. 224).

Common factor analysis: A factor model in which the factors are based upon a reduced correlation matrix. That is, communalities...are inserted in the diagonal of the correlation matrix, and the extracted factors are based only on the common variance, with specific and error variance excluded.

Correlation matrix: A table showing the intercorrelations among all variables.

Eigenvalue: The column sum of squares for a factor; also referred to as the latent root. It represents the amount of variance accounted for by a factor

Factor: A linear combination of the original variables. Factors also represent the underlying dimensions (constructs) that summarize or account of the original set of observed variables

Factor loadings: The correlation between the original variables and the factors, and the key to understanding the nature of a particular factor. Squared factor loadings indicate what percentage of the variance in an original variable is explained by a factor.

Factor matrix: A table displacing the factor loadings of all variables on each factor.

Factor score: Factor analysis reduces the original set of variables to a new smaller set of variables, or factors. When this new smaller set of variables (factors) is used in subsequent analysis (e.g., discriminant analysis), some measure or score must be included to represent the newly derived variables. This measure (score) is a composite of all of the original variables that were important in making the new factor. The composite measure is referred to as a factor score.

Orthogonal: Refers to the mathematical independent of factor axes to each other (i.e., at right angles, or 90 degrees).

Orthogonal factor solutions: A factor solution in which the factors are extracted so that the factor axes are maintained at 90 degrees. Thus each factor is independent of, or orthogonal from, all other factors. The correlation between factors is arbitrarily determined to be zero.

Trace: The sum of the squares of the numbers on the diagonal of the correlation matrix used in the factor analysis. It represents the total amount of variance on which the factor solution is based...With common factor analysis, the trace is equal to the sum of the communalities on the diagonal of the reduced correlation matrix (also equal to the amount of common variance of the variables being analyzed).

Limitations

The nature of limitations in this study included data selection and collection, the timing of the study and the statistical approach chosen.

The data for this retrospective chart review of a convenience sample of patients was limited to the available variables collected as part of standard clinical care at one large urban transplant center. Patient characteristics such as socioeconomic status, ethnicity, and healthcare coverage, or even the level of care provided by the hospital (primary versus secondary versus tertiary) may differ in other centers. Thus, the generalizability, or external validity of the results in this sample of patients was limited. Alternatively, unintentional bias was reduced and clinical equipoise did not exist since the treatment had already occurred.

This study was performed on patient encounters prior to the Patient Protection and Affordable Care Act of 2010 (PPACA) which heralds a more reliable, accessible healthcare system offering higher quality and higher value care (Rak & Coffin, 2013). Information-sharing via electronic medical records and access to primary care may directly affect standard clinical practice. Thus the patient seen in the emergency room at the time of this study may look very different in the future. The generalizability of the study results to these potential changes in care is beyond the scope of this paper.

Lastly, but most importantly are the limitations of the EFA method for data reduction and the technical difficulty that may be experienced by novices such as the writer of this dissertation. Data reduction using EFA is a highly pragmatic function. Computer software programs such as SPSS® and SAS® allow for quick and easy computations and a large number of variables can be directly imported from databases such as Excel®. However, EFA is a complex procedure with fewer absolute guidelines or rules for selecting options compared to other statistical approaches. The steps taken were detailed, justified by the literature reviewed and alternate choices were discussed. The seven stages in factor analysis design as outlined by Hair et al. (2006) were employed in this analysis.

CHAPTER 2 REVIEW OF THE LITERATURE

The burden of heart failure on everyday living places a physical toll on patients, and increasingly so, when worsening symptoms result in hospitalizations placing them at a particularly high risk for adverse outcomes after discharge (Heidenreich et al., 2013). Between fifteen to twenty percent of patients present with low systolic blood pressure, with or without signs or symptoms of hypoperfusion (Buiciuc et al., 2011; Saito et al., 2010). Low blood pressure often precludes the use of drugs used to treat low cardiac output but could the causes of hypotension differ in this group (Gheorghide et al., 2012)? “Management of AHFS is challenging given the heterogeneity of the patient population, absence of a universally accepted definition, incomplete understanding of its pathophysiology, and lack of robust evidence-based guidelines” (Gheorghide & Pang, 2009, p. 557). The aim of this study was to use EFA for data reduction to elicit a parsimonious set of factors summarizing the relationships between variables by measuring intercorrelations of the clinical variables collected as part of standard care, and abstracted from electronic medical records.

A comprehensive review of the literature presented includes a discussion of heart failure, the Framingham Heart Study and the Heart Failure Classification System. The purpose of integrating clinical trial results and best clinical practices to produce evidenced-based guidelines opens the discussion of past drug development for heart failure.

Heart Failure

Heart failure is commonly defined as a pathophysiological state causing abnormal cardiac function resulting in a weakened heart that fails to adequately pump blood at a rate required by peripheral tissues and organs. Although only the right or left side may be affected, often both sides of the heart are involved. Chatterjee and Massie (2007) discussed the definition of heart failure stating, “confusions and controversies regarding the definitions, pathophysiology, prognosis and management of DHF and SHF continue” (p. 569). Heart failure is generally long term or chronic but may also have a sudden onset. Determining the underlying cause of heart failure in such a heterogeneous group has prognostic and therapeutic importance. Genetic, hormonal, and dietary factors play an important role in heart failure (Mosterd & Hoes, 2007; Yancy et al., 2013).

As indicated by the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines of 2009, “Heart failure is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood” (Hunt et al., 2009, p. e397). Thus by definition, heart failure has many causes and certain risk factors as listed in Table 1.

Table 1

Causes and Risks of Heart Failure

Most Common	Left-sided heart failure include
<ul style="list-style-type: none"> · ST-segment elevation myocardial infarction · Untreated or inadequately treated hypertension 	<ul style="list-style-type: none"> · Ischemic heart disease · Hypertension
Rare	
<ul style="list-style-type: none"> · Uncommon high-output states (osteitis deformans , beriberi) · Valvular disease (tricuspid incompetence, pulmonary stenosis) · Infective endocarditis · Isolated right ventricular cardiomyopathy · Hyperthyroidism · Vitamin deficiency · Myocarditis · Toxic substances · Illicit drugs (e.g., cocaine and amphetamines) · Endomyocardial fibrosis · Hemochromatosis · Amyloidosis 	<ul style="list-style-type: none"> · Arrhythmias (especially atrial fibrillation) · Valvular disease (aortic stenosis , aortic regurgitation , mitral regurgitation) · Cardiomyopathy · High-output states (anemia, hyperthyroidism) · Congenital heart disease · Volume overload (e.g., renal failure /dialysis) · Alcoholism
	Right-sided heart failure
	<ul style="list-style-type: none"> · Left-sided heart failure (most common) · Chronic pulmonary disease · Pulmonary embolism · Primary pulmonary hypertension · Valvular disease (mitral stenosis)
Risk Factors	
<ul style="list-style-type: none"> ° Obesity ° Obstructive Sleep Apnea ° Cigarette Smoking ° Infection, especially pulmonary infection 	<ul style="list-style-type: none"> ° Diabetes ° Physical Inactivity ° Renal Insufficiency

Adapted from Mann (2011)

Management of acute decompensated heart failure differs than that of chronic compensated heart failure. Common factors that precipitate acute decompensated heart failure as listed in Table 2 include factors attributed to the onset of heart failure, such as acute myocardial ischemia, hypertension and excessive alcohol or cocaine use (Murphy & Llyod, 2007; Yancy et al., 2013). The goal to optimize volume status and relieve the

signs and symptoms of acute decompensated heart failure in patients with chronic heart failure can be achieved by quantitative evaluation of hemodynamics indices and/or clinical assessments. In acute decompensated heart failure, the therapeutic approach depends on whether there is evidence of volume overload, low cardiac output or indicators of both. Invasive hemodynamic monitoring or signs and symptoms are used to classify patients in one of four Forrester Hemodynamic subsets (Forrester, Diamond, Chatterjee, & Swan, 1976). A pulmonary artery catheter is advanced through a large vein to the right side of the heart and into a small branch of the pulmonary artery during a right heart catheterization in complex patients who require intensive hemodynamic monitoring. However, most patients are classified based on the signs and symptoms exhibited at presentation.

Table 2
Common Factors that Precipitate Acute Decompensated Heart Failure

•	Nonadherence with medication regimen, sodium and/or fluid restriction
•	Acute myocardial ischemia
•	Uncorrected high blood pressure
•	Atrial Fibrillation and other arrhythmias
•	Recent addition of negative inotropic drugs (e.g., verapamil, nifedipine, diltiazem, beta blockers)
•	Pulmonary embolus
•	Initiation of drugs that increase salt retention (e.g., steroids, thiazolidinediones, NSAIDs)
•	Excessive alcohol or illicit drug use
•	Endocrine abnormalities (e.g., diabetes mellitus, hyper- or hypothyroidism)
•	Concurrent infections (e.g., pneumonia, viral illnesses)
•	Additional acute cardiovascular disorders (.e.g, valve disease endocarditis, myopericarditis, aortic dissection)

Adapted from the 2013 American College of Cardiology Foundation and American Heart Association Practice Guidelines (Yancy et al., 2013, p. e285)

The 2013 American College of Cardiology Foundation and American Heart Association practice guides for the management of patients with acute decompensated heart failure and fluid overload include the following;

- intravenous diuretic treatment
- patients receiving loop diuretic therapy should receive an initial parenteral dose greater than or equal to their chronic oral daily dose; then dose should be serially adjusted
- heart failure with reduced ejection patients on guideline-directed medical therapy should continue this therapy except in cases of hemodynamic instability or where contraindicated

- initiation of beta-blocker therapy at a low dose is recommended after optimization of volume status and discontinuation of intravenous agents
- thrombosis/thromboembolism prophylaxis is recommended
- Serum electrolytes, urea nitrogen, and creatinine should be measured during titration of heart failure medications, including diuretics
- when diuresis is inadequate, it is reasonable to
- give higher doses of intravenous loop diuretics
- add a second diuretic
- low-dose dopamine infusion may be considered with loop diuretics to improve diuresis
- ultrafiltration may be considered for patients with obvious volume overload
- ultrafiltration may be considered for patients with refractory congestion
- intravenous nitroglycerin, nitroprusside, or nesiritide may be considered an adjuvant to diuretic therapy for stable patients
- patients with volume overload and severe hyponatremia, vasopressin antagonists may be considered. (Yancy et al., 2013)

Independent predictors of mortality in patients admitted to hospital with decompensated heart failure include age (per year), male gender, diabetes, renal dysfunction, ankle edema, weight, low blood pressure (defined as systolic pressure <110 mm Hg or diastolic <70 mmHg) and no use of beta blockers were reported by Mosterd and Hoes (2007). "In outpatients with chronic HF, a hospitalization is one of the strongest prognostic predictors for increased mortality" was concluded by Gheorghiade et al. (2013, p. 391). Abraham et al. (2008) analyzed registry data from 259 hospitals determining the risk of in-hospital mortality increased in patients who are older, exhibit a low systolic blood pressure, low sodium level, elevated heart rate, or high serum creatinine level at admission. Fonarow, Adams, Abraham, Yancy, and Boscardin (2005) identified variables such as vital signs, and laboratory data as low, intermediate, and high predictors of mortality in hospitalized patients. A low admission systolic blood pressure of <115 mm Hg was determined the second best predictor of mortality.

Classification of Patients with Acute Decompensated Heart Failure

Cardiac output is the volume of blood that the heart pumps or ejects each minute in liters per minute. Cardiac output is dependent upon preload and afterload. Patients with heart failure have a flatter Frank-Starling curve due to impaired ventricular performance requiring higher filling pressure to produce stretch to maintain output. Preload is measured by the pulmonary capillary wedge pressure. A value greater than 18 mmHg indicates volume overload, congestion or wet, whereas a value less than 18 mmHg indicates the patient is dry (Joseph, Cedars, Ewald, Geltman, & Mann, 2009). Signs and symptoms when a patient is wet include dyspnea, cough, paroxysmal nocturnal dyspnea, pulmonary congestion on chest x-ray, peripheral edema, ascites, hepatomegaly, splenomegaly and jugular vein distention. Afterload measured by systolic vascular resistance indicates adequate perfusion to the peripheral tissues. Higher than normal systolic vascular resistance indicates systemic vasoconstriction resulting in a cold state (Joseph et al., 2009). Noninvasive blood pressure readings may be used as surrogates for afterload.

Cardiac index is a measure of cardiac output that has been normalized for body size based body surface area. A cardiac index greater than 2.2 L/min/m², lower than the normal range of 2.8-4.2 L/min/m², indicates adequate perfusion in patients with heart failure (Joseph et al., 2009; Nohria et al., 2003). The term warm is used for cardiac indices greater than 2.2 L/min/m² and the term cold for lower values indicates poor perfusion of blood to the body's tissues (Nohria et al., 2003). Signs and symptoms of poor tissue perfusion are cold clammy extremities, fatigue, altered mental status and low blood

pressure and indicative of poor end organ perfusion are abnormal liver enzymes or serum creatinine.

Thus cardiac output can be assessed hemodynamically or clinically as warm and dry, warm and wet, cold and dry, and cold and wet, and the appropriate therapy can be applied (refer to Figure 3). These measures include optimizing chronic oral therapy, use of diuretics, vasodilators or inotropes, and/or gentle fluid hydration. Unfortunately, patients with low systolic blood pressure, as often seen in the cold and dry subset, may not be treated with vasodilators, or caution with diuretic use may be needed in the two wet subsets. Even optimization of chronic oral therapy in the warm and dry subset may be difficult as beta blockers and angiotensin-converting enzyme inhibitors lower blood pressure.

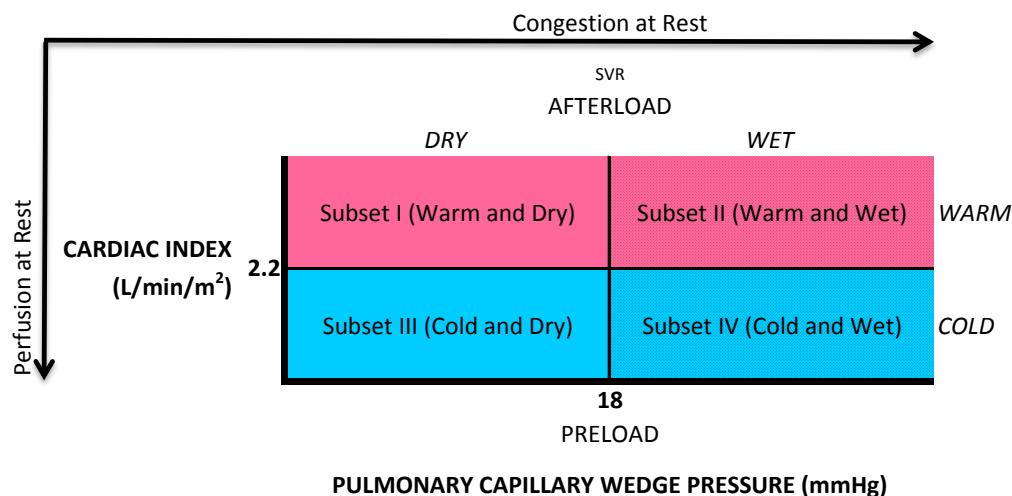


Figure 3. Classification of patients presenting with acutely decompensated heart failure. Adapted from Joseph et al. (2009) and Nohria et al. (2003)

Framingham Heart Study

President Franklin D. Roosevelt's death from hypertensive heart disease and stroke in 1945 prompted the Framingham Heart Study (McKee, Castelli, McNamara, &

Kannel, 1971). The Framingham Heart Study followed an original cohort and two subsequent generations to identify genetic and environmental factors influencing the development of cardiovascular and other diseases. As a result, the current understanding in the epidemiology, pathophysiology, diagnosis, treatment and seminal findings of the risk factors of cardiovascular disease has improved globally (Kannel et al., 1999; McKee et al., 1971). The Framingham Heart Failure Diagnostic Criteria applied to both acute and chronic heart failure are as follows in Table 3:

Table 3
Framingham Decompensated Heart Failure Criteria

Major Criteria		Minor criteria	
1	Paroxysmal nocturnal dyspnea or orthopnea	1	Bilateral ankle edema
2	Distended neck veins (in other than the supine position)	2	Night cough
3	Rales	3	Dyspnea on ordinary exertion
4	Increasing heart size by x-ray	4	Hepatomegaly
5	Acute pulmonary edema on chest x-ray	5	Pleural effusion by x-ray
6	Ventricular S(3) gallop	6	Decrease in vital capacity by one-third from maximum record
7	Increased venous pressure >16 cm H ₂ O	7	Tachycardia (10 beats per minute or more)
8	Hepatojugular reflux	8	Pulmonary vascular engorgement on chest x-ray
9	Pulmonary edema, visceral congestion, cardiomegaly shown on autopsy		
10	Weight loss on CHF treatment of 10 lbs/5days		

A definite diagnosis of congestive heart failure requires that a minimum of two major or one major and two minor criteria be present concurrently. The presence of other conditions capable of producing the symptoms and signs are considered in evaluating the findings.

Adapted from: Framingham Heart Study (2006, p. 6)

An expert consensus document defining acute heart failure resulted from the first and second International Workshop on Acute Heart Failure Syndrome held in May of 2004 and April of 2005. According to this seminal paper by Gheorghide and colleagues (2005),

AHFS is defined as gradual or rapid change in heart failure (HF) signs and symptoms resulting in a need for urgent therapy. These symptoms are

primarily the result of severe pulmonary congestion due to elevated left ventricular (LV) filling pressures (with or without low cardiac output). (p. 1)

Today, acute decompensated heart failure is defined by a disorder associated with sodium and water retention, left ventricular dysfunction of the heart, and neurohormonal activation.

Heart Failure Classification System

Heart failure is a progressive disorder that can be represented as a clinical continuum. Stratification of the severity of heart failure is key to therapeutic management and long-term prognosis. In 1928, the first version of the New York Heart Association (NYHA) classification system was used to classify heart failure based on functional limitations and symptomatic status depending on the degree of effort due to fatigue or dyspnea (Appendix A). Capable of readily shifting with emergency treatment such as diuresis, or with the addition of a guideline recommended therapy over time, the functional class of a given patient is not always static. The American College of Cardiology Foundation (ACCF) and the American Heart Association (AHA) stages are used to determine risk for developing the disease, disease severity and prognosis (Hunt et al., 2001). Staging includes both asymptomatic and symptomatic phases in the development of heart failure (Hunt et al., 2001). Patients without structural heart disease or symptoms of heart failure with other comorbidities such as diabetes, hypertension, atherosclerotic vascular disease, metabolic disease, obesity or cardiotoxin exposures such as (i.e., due to chronic alcoholism, cocaine use, chemotherapy, radiotherapy, or certain medications) are at high risk for heart failure and classified as Stage A. Stage B includes those patients with previous myocardial ischemia, asymptomatic valvular heart disease and left ventricular remodeling of the heart. Patients with symptoms of heart failure with either

reduced left ventricular ejection fraction (typically 40% or less), borderline preserved (41-49%) or preserved (50% or greater), are Stage C even if they later become asymptomatic. Those with marked heart failure symptoms at rest and multiple hospital readmissions are Stage D. However, patients may skip a stage, or revert to an earlier stage when treatments result in reverse remodeling. The functional rating scale for left ventricular ejection fraction measured by echocardiography is depicted in Table 4.

Table 4
Left Ventricular Ejection Fraction as Measured by Echocardiogram

Ejection Fraction	Functional Rating	Heart Failure Spectrum
45%–70%	Normal	Heart failure with preserved ejection fraction
35%–45%	Mild impairment	Heart failure with reduced ejection fraction
25%–35%	Moderate impairment	Heart failure with reduced ejection fraction
<25%	Severe impairment	Heart failure with reduced ejection fraction
<15%	End-stage/transplant candidates	Heart failure with reduced ejection fraction
5%	Is compatible with life but not long life	Heart failure with reduced ejection fraction

Adapted from (Ashley & Niebauer, 2004)

Many clinical trials are designed using NYHA class as an entry criterion, or as the basis for analyzing interactions and outcomes (Ahmed et al., 2006; Bouvy, Heerdink, Leufkens, & Hoes, 2003; Kubo et al., 2004; Sakurai et al., 2003; Soejima et al., 2000). Concordance between NYHA classification, ACCF/AHA staging and/or other objective measures such as left ventricular ejection fraction measured in diagnostic imaging studies, functional exercise testing using the six minute walk test (6MWT), serum analysis measuring brain natriuretic peptide levels (BNP) and symptoms such as dyspnea are extensively studied (Beatty, Schiller, & Whooley, 2012; Carvalho, Garrod, Bocchi, Pitta, & Guimaraes, 2010; Rostagno et al., 2003; Wieczorek et al., 2002). Raphael et al. (2007) studied the use of NYHA classification using three methods. A Medline review of clinical trials often did not explain the criteria used to determine NYHA class. Peak oxygen consumption as measured during cardiopulmonary testing did not correlate to a patient's self-reported walking distance. Their interoperator study using class II and class III

patients also failed to show reliability. Thus, they concluded, “No consistent method of assessing NYHA class is in use and the interoperator study on class II and class III patients gave a result little better than chance” (p. 2). Another functional test, the six minute walk test (6MWT), was found to be predicted by NYHA class (Athilingam, D’aoust, Zambroski, McMillan, & Sahebzemani, 2013). However, cardiac structural abnormalities defined by the ACC/AHA staging system did not exhibit any correlation to the six minute walk test. As well, there was no concurrent validity between NYHA class and ACC/AHA Stages of heart failure.

Is there a legitimate reason to use either NYHA class or ACC/AHA stages in clinical trial design? Gary G. Koch, biostatistics professor and director of the Biometrics Consulting Laboratory at the University of North Carolina at Chapel Hill is a noted expert in categorical data analysis especially in medical research. According to Koch (as cited in Dmitrienko, Molenberghs, Chuang-Stein, & Offen, 2005), “randomization- and model-based methods have been historically motivated by two different sampling schemes. As a result, randomization-based inferences are generally restricted to a particular study, whereas model-based inferences can be generalized to a larger population of patients” (p. 2). If NYHA class or ACC/AHA staging are valid and reliable functional or structural measures of heart failure, respectively, then each model can be used to test diagnostic and prognostic markers.

Evidence-Based Guidelines

Coined by Dr. Gordon Guyatt in 1990 at McMaster University, evidence- based medicine combines clinical expertise with scientifically sound research (Guyatt et al., 1992). Research in the form of clinical trials and other findings aim to provide evidence

whether or not a new treatment is safe and more effective compared to current treatments. The foundation of evidence-based medicine is the evidence itself, which must be gathered and collated from a systematic review of relevant individual published research studies. Traditional narrative reviews and expert commentaries previously used to summarize research evidence are replaced by systemic reviews of the best available research. The Evidence Hierarchy places systemic reviews of randomized controlled trials at the highest level notated by Level 1a with expert opinion at the lowest level notated by Level 5 (Yancy et al., 2013).

A systematic review is a summary of the critical appraisal of clinical literature used to answer a focused clinical question without bias using a predetermined, organized methodology. The methodology includes pre-specified eligibility criteria and a systematic search strategy to identify relevant comparable (homogenous) high quality studies. Unlike traditional reviews, the evidence from each study is systematically summarized. In a literature review, the evidence is summarized using a qualitative approach to provide a summary of the targeted topic, not a specific question.

The term meta-analysis is an unbiased summary of all of the highest quality information from several studies using set rules or standards in a systematic review that is statistically analyzed to provide a combined estimate. Over the past 20 years, the Cochrane Collaboration electronically publishes review from the medical literature in the Cochrane Database of Systemic Reviews. "This is an international organization whose goal is to help scientists, physicians, and decision makers make well-informed decisions about health care by coordinating systematic reviews of the effects of health care

intervention” (Jadad & Haynes, 1998, p. 2). Systematic reviews and meta-analyses are key elements of evidence-based healthcare.

Management of heart failure ought to be guided by evidence-based guideline directed diagnosis, evaluation and treatment. Evidence from clinical trials often guides selection of therapy in the clinical setting. Judicious care should be taken when treatment outcomes from clinical trials are extrapolated to patients in the clinical setting whose characteristics differ than those enrolled in clinical trials.

Although race is a poor marker of variation, the combination of isosorbide dinitrate and hydralazine exhibits efficacy in the treatment of African Americans with heart failure reducing mortality by 43% (Golwala et al., 2013; Hammermeister et al., 2009; Sharma, Colvin-Adams, & Yancy, 2014). However, the association between the use of non-potassium-sparing diuretics for fluid management and increased mortality and rehospitalization is less understood (Brandimarte et al., 2010; Golwala et al., 2013). One reason that accounts for this inability to draw conclusions is that patients entered in clinical trials must meet strict entry criteria to optimize the likelihood of measuring efficacy and safety of the treatment with positive outcomes that are acceptable to approval agencies such as the Food and Drug Administration (FDA), Health Canada or European Medicines Agency. Entry criteria include several inclusionary and exclusionary criterions that limit the heterogeneity of patients and decrease the potential risks of the treatment. To improve the characteristics of the study population, the identification of novel biomarkers and surrogate endpoints is needed (Fonarow et al., 2007; Gheorghiade & Rushchitzka, 2011). Patients selected from the target population may not reflect the characteristics of those in the general heart failure population. Thus, the key goal is to select patients reflective of

the heart failure condition, yet homogenous enough to reveal the effect of the treatment rather than differences between individual patients in the study.

In part due to ethical considerations, most studies require patients to be medically stable for at least a few months prior to study entry and to likely be stable for duration of the study. Less stable patients are usually offered standard therapy of FDA approved drugs whether or not their effectiveness has been shown in clinical trials (Morris et al., 2006). Medical stability is defined by the type of study and mechanism of action of the drug. A patient who is medically stable on a standard heart failure therapy may be the ideal candidate to measure the superiority of a new drug compared to its FDA approved Counterpart. In this case, entry criteria may define medical stability as no change in cardiovascular drug(s) class or significant dose adjustment up to six months of study entry, no hospitalization within the past twelve months, and no new treatment plan that may affect heart failure. At the other end of the spectrum, medical stability may be defined as stability other than the acute decompensation of heart failure that resulted in the hospitalization. However, regardless of the definition of medical stability, patients with a chronically low systolic blood pressure are often excluded in study participation.

Another issue seen is when patients meet entry criteria but decline to enroll, or are deemed inappropriate by the clinical investigator or sponsor for various reasons raising questions about the applicability of the study results to the greater population (Kaptchuk, 2001; Rothwell, 2005). In a commentary published in a peer reviewed journal, Professor Ted J. Kaptchuk addressed the potential bias of the gold standard placebo controlled randomized clinical trial by stating, "It seems that the most 'rigorous' evidence may produce deviations from the truth" (2001, p. 546). Patients who are asked to stop excluded

drugs or therapies may decline to participate. Patients deemed compliant with a drug requiring once a day dosing may not be compliant with more than once a day dosing. And most applicable to the current study, although not an exclusion criterion, the clinical investigator may deem a patient low systolic blood pressure ineligible, in part due to the potential risk of a significant drop in blood pressure. The negative effect is twofold: an increase in serious adverse events and removing the patient from the study treatment and/or the study. In an intent-to-treat study, this could have a deleterious effect on study outcomes, especially if the patient was randomized to the active group (active study drug or treatment). Underrepresentation of patients with borderline or low systolic blood pressure in heart failure clinical trials makes it difficult to understand the full impact of medical interventions and the generalizability or external validity of the use of such interventions.

Past drug development for heart failure. Over the past 20 years, chronic heart failure was the primary focus of clinical and epidemiological research with only a few studies targeting patients hospitalized with acute decompensated heart failure (Adams et al., 2005; Amin, 2008; Badano et al., 2004; Smith et al., 2004; Tavazzi et al., 2006). Most of the heart failure research has focused on developing new drugs for FDA approval, or testing approved drugs in the initial hours of presentation in the emergency room (Felker et al., 2010). An example of the latter is the much studied drug nesiritide, approved in 2001 by the FDA for use as a first line treatment in the emergency room for dyspnea in acute decompensated heart failure. In a 2005 meta-analysis of trials, nesiritide was associated with short-term risk of mortality when used to treat acute decompensated heart failure. Prompted by such a strong signal of risk, five years later the Acute Study of

Clinical Effectiveness of Nesiritide in Decompensated Heart Failure (ASCEND-HF), a multicenter randomized trial with over 7,000 patients demonstrated an acceptable safety profile but no apparent efficacy (O'Connor et al., 2011; Sackner-Bernstein, Kowalski, Fox, & Aaronson, 2005). Registries were created based on these clinical trials and volumes of clinical data were collected. Although the evidence for nesiritide's clinical efficacy may not be robust, expert opinions based on retrospective chart reviews or anecdotal information guide the decision to treat or not to treat with nesiritide.

The controversy of nesiritide's efficacy continues to this day (Felker et al., 2010; A. F. Hernandez et al., 2009; McDonagh et al., 2011; O'Connor et al., 2011; Sackner-Bernstein et al., 2005; Wang et al., 2004; Yan et al., 2014). Even so, nesiritide and other natriuretic peptide drugs continue to be trialed for acute decompensated heart failure. However, due to its hypotensive properties, patients with low systolic blood pressure are excluded from study participation. The result is that one of the most intensively studied drug, nesiritide, where volumes of clinical data were used to paint the clinical picture of a patient with acute decompensated heart failure, excludes patients with low systolic blood pressure. This is reminiscent of the days when the majority of drugs were tested on white males spurring such action as the National Institutes of Health (NIH) Revitalization Act of 1993 (PL103-43) requiring inclusion of women and minorities as participants in NIH-funded clinical trials (Swanson & Ward, 1995; U.S. food and Drug Administration, n.d., para 8).

Characteristics and outcomes from published studies evaluating the efficacy and safety of nesiritide were used to define the heart failure patient. Conclusions were made and accepted based on database analysis of several heart failure registries developed

from the nesiritide studies. A conclusion supported by these registries is that when treatment is initiated early, outcomes improve, as is the case in other acute illnesses (Adams et al., 2003; Rivers et al., 2001; Rowe, Spooner, Ducharme, Bretzlaff, & Bota, 2001; Vidt, 2001). Clinical characteristics and outcomes can be limited by the selection criteria used to study the safety and efficacy of specific investigational drugs or devices. Constraints on standard treatment options while on study are often inherent in clinical trials. For these reasons measurements of the causes of mortality and morbidity and readmission rates from randomized clinical trials cohorts may not provide external validity (Murray-Thomas & Cowie, 2003).

Buiciuc et al. (2011) conducted a long-term study investigating the association between low systolic blood pressure and long-term mortality in patients newly diagnosed with heart failure and preserved ejection fraction. The authors state, "An epidemiologic approach was taken without the use of modelbuilding" (p. 909). Clinical data and laboratory testing results were used as measures of association to draw inferences concerning causal relationships between these events and the five year all-cause mortality rates. A low systolic blood pressure defined in this study as < 120 mmHg was linearly associated with a five year all-cause mortality and cardiovascular death. Future clinical research is needed to determine if standard of care blood pressure readings are useful in improving current risk stratification models.

Investigators of the Organized Program to Initiate Lifesaving Treatment In Hospitalized Patients With Heart Failure (OPTIMIZE-HF) clinical trial suggests using a risk-prediction algorithm to identify patients who are at high risk of in-hospital mortality. Systolic blood pressure and six other multivariables were used in the scoring system to

predict mortality. Patients with high risk scores may be appropriate candidates for more aggressive monitoring and intervention than patients with average or low risk of in hospital death (Abraham et al., 2008). Alternatively, patients with low risk scores may be treated conservatively without exposure to additional risks from aggressive interventions. Risk-prediction algorithms are medical calculators that are easy, quick, reliable, and rely on evidence-based medical guidelines to compute risk for decision-making to support healthcare delivery. Free and premium versions of mobile apps for risk calculation are increasing used in clinical practice. An example of a cardiology tool is the Framingham Risk Calculator that measures the 10 year coronary heart disease risk using seven simple questions (i.e., age, gender, total cholesterol, HDL cholesterol, smoking status, systolic blood pressure reading, and use of medication to treat hypertension).

This restriction of range in selected patients, while providing homogeneity for analyzing the results of specified investigational drugs, may not mirror the average heart failure admitted for acute decompensated heart failure. Several trials exclude or limit entry of patients admitted with common comorbidities such atrial fibrillation, diabetes, or severe hypertension. A low systolic blood pressure often excludes patients either directly, or indirectly. Safety concerns with drugs that lower blood pressure result in exclusion of patients with a systolic blood pressure of less than 110 mmHg. Intravenously administered drugs such as inotropes or vasodilators used to treat patients with low systolic blood pressure indirectly exclude these patients. Thus the clinical characteristics of patients enrolled in clinical trials may differ significantly from patients encountered in the real-world clinical practice.

Use of Retrospective Data

Evaluation of the disease course, prognosis and response to therapy over time is critically important to collect. Documentation dictated by national guidelines and required for reimbursement has standardized the information collected at each patient visit.

The investigator starts with the disease or its outcome(s) and works backwards to find possible causes. Retrospective studies by definition are nonexperimental and play no role in assigning patients to treatment. Thus a cause and effect cannot be measured. Most IRBs will not require patient consent which avoids the Hawthorne Effect. The Hawthorne Effect refers to the phenomenon whereby a temporary change in behavior or performance occurs when people know they are being observed.

There are many advantages of using existing data collected from electronic medical records. Prospectively collected new data is expensive, labor-, and time-intensive. The use of existing data allows a quicker, more efficient and less expensive source for data analysis. Large samples ($N > 1,000$) can be realistically conducted in a short period of time. They can be analyzed to detect uncommon outcomes to provide odds ratios which are an estimate of relative risk. The data can be randomly sampled or used to create a cohort. In addition, data in an electronic query enabled format represents less chance of transcription errors and missing data than manual chart abstraction. Variables are stored in discrete fields in electronic medical records. Demographic information, laboratory results, administered inpatient drugs, outpatient prescriptions (used to measure compliance) and many other variables can be imported directly to other database platforms. Data recorded in clinical notes that are not in a designated numeric text field would need to be manual extracted. Although, data recognition software such

as Optical Character Recognition software (OCR software) translates text images into text read by computer may eventually eliminate the need for manual extraction.

The use of existing data from various sources including financial and medical insurance coverage allows for more identifiers which may potentially illustrate relationships between variables that would otherwise not be determined. Results vary whether or not insurance benefit coverage is associated with improved health care outcomes (Fowler et al., 2010; Kapoor et al., 2011; B. S. Mann et al., 2014; Mansi, Shi, Altenburg, Mukoosa, & Huang, 2011). Existing knowledge can be reinforced by the data or prompt the need for change. Interestingly, retrospective collection of data performed without any consideration of the study may reduce the chance of unintentional bias. As stated earlier, there is an issue of generalizability or externality since prospective randomized clinical trials have restrictive entry criteria and a strict protocol must be followed (Rothwell, 2005). There is careful monitoring, adherence and follow-up inherent in the study schedule of visits. The question remains whether or not these results can be extrapolated to the general heart failure population. The selection of patients entered in a retrospective study is much looser and their care is often what is normally seen in clinical practice.

Other pitfalls include clinical equipoise, refusal to be randomized, and early withdrawal. Clinical equipoise applies to randomized controlled trials that use placebos, an inferior comparator, or a comparator that exhibits inferiority at some point in the study. "Clinical equipoise, also known as the principle of equipoise, provides the ethical basis for medical research that involves assigning patients to different treatment arms of a clinical trial. The term was first used by Benjamin Freedman in 1987" (The Free

Dictionary, para 1). Freedman, a bioethicist at the Clinical Trials Research Group at McGill University's Biomedical Ethics unit wrote, "According to this concept of clinical equipoise, the requirement is satisfied if there is genuine uncertainty within the expert medical community - not necessarily on the part of the individual investigator - about the preferred treatment" (1987, p. 141). The ethical issue of clinical equipoise does not exist in retrospective studies since the treatment has occurred. The ethical concern over the fiduciary duty found in the Hippocratic Oath to do what is best for the patient rather than the physician may be in direct conflict of the randomization to treatment in the experimental design. A detailed discussion of clinical equipoise was beyond the scope of this paper. Also, this may not be particularly important in this paper as patients with low systolic blood pressure are often not entered in clinical trials either by design or by the decision of the clinical investigator for fear that the active arm may be deleterious to the patient's health.

As there are advantages to using existing data there are important disadvantages. Unlike prospective studies, treatments are not controlled or manipulated and since the activities have occurred in the past, measurement of outcomes is limited to what is documented. However, the data may not be suited to test the current research questions since it was collected for other purposes. Additionally it may be incomplete especially if there a variety of treating physicians or institutions. This may result in sampling bias.

Other limitations include patient and treatment selection biases and this source of error is greater in retrospective studies compared to prospective studies. As more physicians abide by evidence-based guidelines set by the American Heart Association (AHA), American College of Cardiology (ACC), American Stroke Association (ASA), and

other organizations for cardiovascular care, this is less of an issue. Even so, these patient populations are less homogenous than those selected for prospective studies. Unmeasured factors can have an influence on treatment. Multivariable analysis can be used to attempt to control for these confounding variables or factors that threaten validity.

In conclusion, every experiment is flawed. According to Campbell and Stanley (1966),

From the standpoint of the final interpretation of an experiment and the attempt to fit it into the developing science, every experiment is imperfect. What a check list of validity criteria can do is to make an experimenter more aware of the residual imperfections in his design so that the relevant points he can be aware of competing interpretations of his data. He should, of course, design the very best experiment which the situation makes possible.
(p. 34)

Although Campbell and Stanley were not addressing the EFA approach, criticism of this method stems from the series of steps and decisions least understood by most investigators. There is no hypothesis. Instead, relationships among variables are explored to identify patterns and the number of factors to be estimated will not be specified a priori but determined based on what the data show. Therefore, the next chapter details the choices and assumptions made at each step of the process combining the steps of EFA with the literature reviewed.

CHAPTER 3 METHODOLOGY

Purpose of the Study

The purpose of this study was to characterize patients hospitalized with acute decompensated heart failure with and without low systolic blood pressure using factor analysis. A better understanding of the characteristics and outcomes of patients presenting with acute decompensated heart failure with and without low systolic blood pressure can potentially lead to individualized treatment modalities tailored to effectively, and economically improve care.

Research Questions

1. What were the common factors in patients with acute decompensated heart failure?
2. What were the common factors in patients with acute decompensated heart failure and normal or high systolic blood pressure?
3. What were the common factors in patients with acute decompensated heart failure and low systolic blood pressure?
4. Was there a difference in the common factors in patients with and without low systolic blood pressure and acute decompensate heart failure?

Setting

The study site was a large urban, academic teaching hospital in the heart of the city just west of a major freeway in Detroit, Michigan. The original hospital built in 1915 by Henry Ford was initially staffed mostly by physicians and surgeons from Johns Hopkins Hospital. The Detroit campus services the Detroit metropolitan area. The emergency room became an independent department in 1982, and in 2006, it was expanded and renovated as a part of \$90 million expenditure. The 38,000 square foot emergency department provides care to approximately 93,000 patients annually and accounts for 44 percent of all hospital admissions (Henry Ford Health System, n.d.-b).

The study site was located in one of the six hospitals of a system that includes specialty programs and medical centers which comprises Michigan's largest mixed model managed care plan. As Michigan's fifth largest employer with more than 23,000 employees, the system accounts for more than an annual economic stimulus of \$1.7 billion (Henry Ford Health System, n.d.-a).

Special features of the department include two major resuscitation rooms, a 16 bed critical care area and centralized nursing stations allowing for maximum visibility of patients with improved efficiency and traffic flow for staff (Henry Ford Health System, n.d.-c). Medical personal include 30 senior staff physicians, 52 residents, over 300 registered nurses, and other support personnel such as pharmacists, physician assistants, and medical technicians. The emergency department includes an onsite pharmacy, active research programs and since 1976, an emergency medicine resident education program. Clinical pharmacy services in the emergency department are associated with increased safety, decreased cost, and improved outcomes (Cohen, Jellinek, Hatch, & Motov, 2009;

Fairbanks, Hays, Webster, & Spillane, 2004). Industry, governmental and investigator initiated research investigating heart failure biomarkers and congestive heart failure research is conducted or initiated in the emergency department. Training programs for medical students, registered nurses, physician assistants and emergency medical services personnel from the University of Michigan, Wayne State University, and over 20 other U. S. medical schools are housed in the emergency department.

Internal Review Board Approval

The institutional review board of record was located at the study site and the Wayne State University Human Investigation Committee was the internal review board for the University. In accordance of the Code of Federal Regulations on human subjects research (45 CFR §46 and 21 CFR §56. 110), the initial review qualified for expedited review, “(1) some or all of the research appearing on the list and found by the reviewer(s) to involve no more than minimal risk” (U.S. Department of Health and Human Services, n.d., Section §46.110). The condition this study met was number five, “Research involving materials (data, documents, records, or specimens) that have been collected, or will be collected solely for nonresearch purposes (such as medical treatment or diagnosis)” (U.S. Department of Health and Human Services, n.d., para 11). The expedited approval letters are found in appendices C and D.

Collaborative Institutional Training Initiative by the researcher for good clinical practice, information privacy and security, human subjects research and responsible conduct of research certification as required by the site’s and the University’s internal review are found in appendices G and H (CITI Program, n.d.).

Cases

Eligible cases were patients hospitalized with a primary diagnosis of acute decompensated heart failure at the study site from January 1, 2014 to October 31, 2014 with an International Classification of Diseases, Ninth Revision, Clinical Modification code 428 (refer to Appendix I). The diagnosis of acute decompensated heart failure was established by the presence of typical symptoms, signs, and objective evidence of pulmonary congestion, elevated b-type natriuretic peptide or impaired cardiac function (Fonarow, Peacock, Phillips, Givertz, & Lopatin, 2007; Gheorghiade & Ruschitzka, 2011; Harinstein et al., 2011; Joseph et al., 2009; Michtalik et al., 2011; Murray-Thomas & Cowie, 2003; Varughese, 2007). All entry criteria (exclusion and inclusion) were met. Case selection was not based on race, ethnic or gender criteria.

In compliance to Health Insurance Portability and Accountability Act of 1996 (HIPAA), the legal conditions for a waiver of authorization were met (U.S. Department of Health and Human Services, n.d.). Protected health information collected but not disclosed were elements of dates and medical record numbers. There was no more than minimal risk of privacy from the use of protected health information since only the investigator had access to the link between the participant's medical record number and the coded identifier used in the database (the term participant rather than case is used in this section). The link was maintained in an electronic format in a password protected computer accessible only to the investigator. A second security measure was employed to secure and prevent opening of the file by using password protection. The data analysis was performed only on the de-linked data. The data were overridden and permanently

deleted six months after the final close-out of the study with the site's and the University's internal review board.

This research could not practically be conducted without the waiver of authorization since dates and medical record numbers are required for data evaluation. No further gain either to the research study or to the participant would result and conversely, there was a potential increased risk of emotional stress if the participant was contacted or approached to consent (Schuck, 1994).

Description of the data set. Variables collected were identified based on review of the literature, Framingham criteria, clinical relevance, and were routinely availability upon the first 24 hours of hospital presentation and throughout hospitalization until discharge. Variables selected for abstraction were further guided by the writer's extensive research experience and clinical expertise. Descriptive variables include gender, race, heart failure etiology and implantable device(s) used to treat heart failure or its associated symptoms such as an implantable cardioverter-defibrillator or a cardiac resynchronization therapy defibrillator. Variables listed in Table 3 were used for data analysis using the guidelines for EFA include ordinal, interval and ratio data. Ordinal data include the New York Heart Association functional class (before hospitalization) and interval data include left ventricular ejection fraction. Ratio data include heart rate, serum laboratory results and total IV diuretic dosage during hospitalization.

Table 5
List of Variables

Exploratory Factor Analysis - Variables		
Descriptive Variables	Ordinal	Continuous
Gender	1 Peripheral edema (none, +1, +2, +3, +4)	1 Age
Race	2 LVEF% (0-10, 10+-20, 20+35, 35+-40, 40+-49, 50-50 >55)	22 Protein, serum
HF etiology	3 HF + CV comorbidities (HF alone, HF+1, HF +2)	23 ALT, serum
ICD/CRT-D	4 HF + nonCV comorbidities (HF alone, HF+1, HF +2)	24 AST, serum
	5 A-fib (none, paroxysmal, perm)	25 Bilirubin, serum
	6 Dyspnea at rest	26 Albumin, serum
Legend		27 Total Protein, serum
ACEi=angiotensin-converting-enzyme inhibitor		28 # of hospitalizations within last 12 months
A-fib=atrial fibrillation		29 # of years of Heart Failure (diagnosis)
ALT=alanine aminotransferase		30 # of Social Risks (tobacco, alcohol, cocaine, no insurance)
ARB=angiotensin II receptor blocker		31 # of Heart Failure meds (ACEi/ARB & BB) + 1 (for each additional)
AST=aspartate aminotransferase		32 # of days hospitalized
B/P=blood pressure		33 total IV diuretic dosage during hospitalization
BB=beta blocker		34 LVEDd diameter (most recent echo)
BMI=body mass index		35 # of HF drugs added at discharge
BNP=B-type natriuretic peptide		36 ACEi/ARB dosage (adjusted), chronic
BUN=blood urea nitrogen		37 Beta blocker dosage (adjusted), chronic
Ca ⁺ =calcium		38 Diuretic dosage (adjusted), chronic
CO ₂ =carbon dioxide		39 Anticoagulation dosage, chronic
CRT-D=cardioverter-defibrillator		
CV=cardiovascular		
eGFR=estimated glomerular filtration rate		
HF=Heart failure		
ICD= implantable cardioverter defibrillator		
LDH=Lactate dehydrogenase		
LVEDd=left ventricular end diastolic diameter		
LVEF=left ventricular ejection fraction		
Mg ⁺ =magnesium		
O ₂ =oxygen saturation		
TSH=thyroid stimulating hormone		

Entry Criteria

Inclusion

1. men or women aged 18-89 years
2. hospitalized for management of acute decompensated heart failure defined as,
 - a. dyspnea at rest or with minimal activity (i.e. , difficulty breathing at rest while sitting, or difficulty breathing while lying flat or with 1 pillow, or difficulty breathing with minimal activity such as talking or eating)
 - b. requiring intravenous treatment with loop diuretic (i.e. , furosemide)

Exclusion

1. 24 hour or less admission defined as discharge from hospital within 24 hours or less (including clinical decision unit at study site)
2. developed heart failure after admission or transferred from another acute care facility
3. advanced AHA stage D CHF requiring consideration for heart transplant, mechanical assisting devices, or chronic inotropic therapy
4. heart failure due to,
 - a. myocarditis and cardiomyopathies due to inflammation
 - b. congenital
 - c. cardiotoxicity related to cancer (chemo- or radiation induced), alcoholic and/or cocaine induced cardiomyopathy permitted
 - d. pregnancy (peripartum/postpartum)
5. planned transfer to hospice care or other end-of-life care

6. planned cardiac surgery or coronary intervention (i.e., angioplasty with or without stenting) at index hospitalization
7. participating in an investigational drug or device clinical trial or behavioral modification study during hospitalization (biomarker study participation permitted)
8. concurrent treatments or conditions including,
 - a. end stage renal disease and/or current dialysis treatment
 - b. current chemo- or radiation therapy
 - c. pregnancy

Pregnancy. Normal hemodynamic changes in pregnancy include an increase in blood volume, heart rate, and cardiac output, a decrease in blood pressure and systemic vascular resistance, and changes in stroke volume due to the growing fetus. A patient with heart failure may be more compromised hemodynamically when pregnant and the variables collected in this study may be very different compared to the target group. An example of such variable is the dosage of angiotensin converting enzyme inhibitors or angiotensin receptor blockers. While treatment with either an angiotensin converting enzyme or angiotensin receptor blocker is a standard guideline treatment in patients with heart failure, both of these medications are contraindicated in pregnancy (Siu et al., 2001; Thorne, 2004).

Sampling. EpicCare EMR by Epic Systems Incorporated is an excellent user interface allowing reports to be exported to other software applications without requiring programming language by the user. Although there is a lag time, real-time data is not required for the retrospective chart review design of this study.

After internal review approval, an online service request order was placed and routed to the senior performance measurement analyst in the department of operational analytics at the study site. An Epic report using the variables of interest was run for the study dates January 1, 2014 to October 31, 2014 for patients in the emergency room a primary ICD-9 diagnosis code of 428 (heart failure, see Appendix J and K) who were subsequently admitted. The raw data was used to build the database. The variables extracted were visually inspected to detect any data quality problems and cleaned. Only eligible cases were retained in sequential order of emergency room presentation. Missing data was evaluated and the data was further cleaned by eliminating cases without documentation of core variables. This was most prominent in patients whose past medical records were retained at an outside institution (e.g., echocardiogram results).

Non structured data, that is, data not found in discrete fields was manually abstracted. This data was not structured or actionable since it was embedded in a report that was uploaded rather than imported to the electronic medical record from the original program such as Siemens Syngo® radiology imaging software.

It was expected up to 500-600 patients were hospitalized with congestive heart failure over the nine month research time period. The first 300 cleaned records were retained for the group EFA addressing research question number 1.

- What were the common factors in patients with acute decompensated heart failure?

The first 150 cleaned records for each of the groups were retained for EFA for the low and normal or high systolic blood pressure group EFA addressing research questions number 2 and 3.

- What were the common factors in patients with acute decompensated heart failure and normal or high systolic blood pressure?
- What were the common factors in patients with acute decompensated heart failure and low systolic blood pressure?

The factors extracted in each of the groups were compared and described.

Sample Size. As is the case in empirical studies, determining sample size in EFA, a large sample size technique, is based on the minimum necessary to obtain reliable results from the analysis. Comrey and Lee (1992) state,

Correlation coefficients tend to be less reliable when estimated from small samples. There, it is important that sample size be large enough that correlations are reliably estimated. The required sample size also depends on magnitude of population correlations and number of factors: if there are strong correlation and a few, distinct factors, a smaller sample size is adequate. (p. 618)

Guidelines or a rule of thumb by expert opinions such as Gorsuch (1983) and Kline (1994) include absolute numbers of at least 100 cases. Comrey and Lee (1992) recommended a scale such that 50 is very poor, 100 is poor, 200 is fair, 300 is good, 500 is very good and 1,000 or more is excellent. Others proposed minimum ratios of number of variable to cases (n:p). Cattell (1978) recommended three to six cases (or subjects) per variable, Gorsuch (1983) recommended a ratio of at least five and Nunnally (1978) recommended at least ten times as many cases as variables. An easy to follow detailed discussion of sample size is found in an article by (MacCallum, Widaman, Zhang, & Hong, 1999).

Data Management

There were three separate spreadsheets using Microsoft Excel software PC version. The first was a spreadsheet containing the Epic report data. Manually abstracted data were added to this spreadsheet. The second was a spreadsheet containing the link

between the abstracted cases that included the medical record number of the cases and the coded identifier or study case number. The third was the fully abstracted data where the medical record number was replaced by the study case number. This latter spreadsheet was imported to the Statistical Package for the Social Sciences® (SPSS) for Windows™, Student Version, Personal Computer Version, v. 17 for data analysis. To ensure confidentiality, access to the Epic report and the spreadsheet containing the link between cases was limited to the principal investigator.

An algorithm of the study design flow from the study proposal phase to the end of the study as in Figure 4 followed by a description and justification of the EFA approach completes the methodology followed in this study.

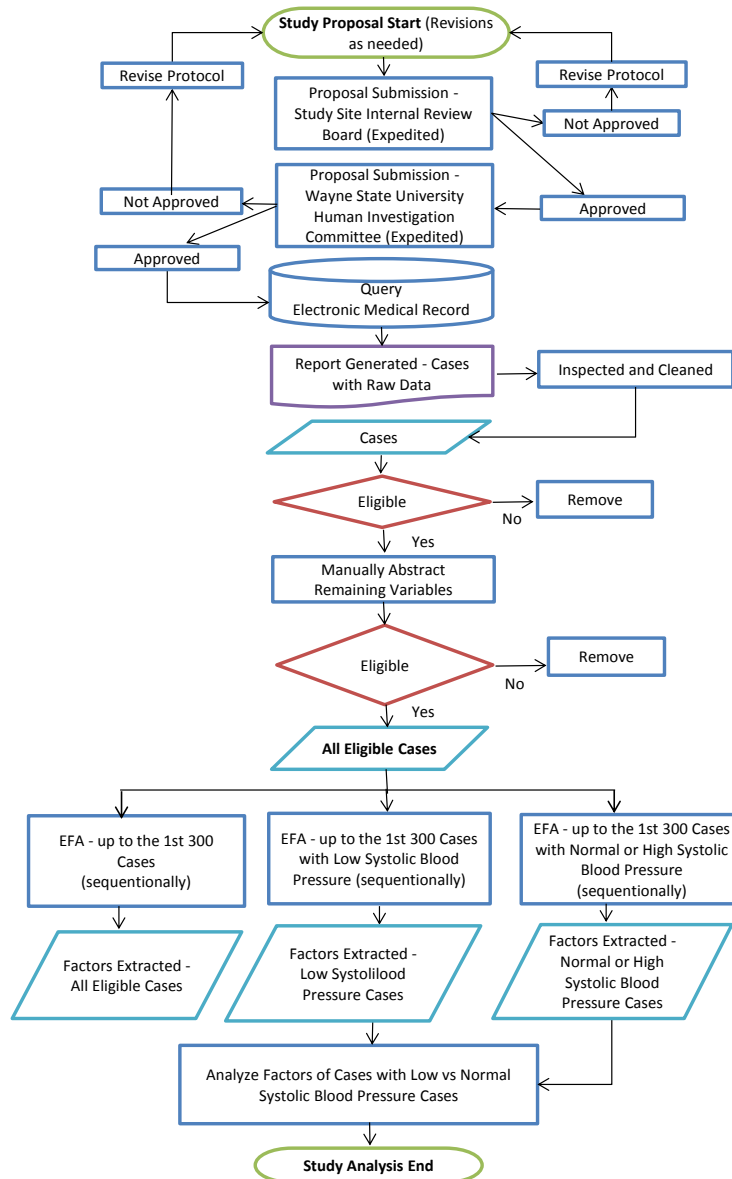


Figure 4 Study Process Algorithm

Data Analysis

EFA is heuristic and used to discover summary constructs when their nature is still unknown. Using a subset of variables that correlate with each other but which are mostly independent of other variables, EFA combines these variables into factors. “Factor analysis consists of a number of statistical techniques the aim of which is to simplify complex sets of data” (Kline, 1994, p. 3). The multivariate approach using EFA to create

composite factor scores is appropriate as listed by Hair et al. (1992) in their fourth purpose of this method, “Create an entirely new set of a smaller number of variables to partially or completely replace the original set of variables for inclusion in subsequent regression, correlation, or discriminate analysis” (p. 226). The model is not specified a priori, the number of latent variables is not specified a priori and all latent variables affect all observed variables. EFA performed in the early stages of research does not require a potential theoretical basis for selection of the variables of interest (Tabachnick & Fidell, 2013).

EFA run on the desired variables produced a data matrix. A correlation matrix was used for EFA rather than a covariance matrix traditionally used for confirmatory factor analysis. Each case was represented in a row and each variable was represented in a column and the resulting matrix has N rows and K columns. The correlation matrix was computed resulting in a square and symmetric mirror image along its diagonal of K rows and K columns. The factor matrix is a linear combination of the common factors and a unique factor that contains error variability and variability of the specific variable. A pictorial representation of the each matrix in the EFA process is exhibited in the following figure.

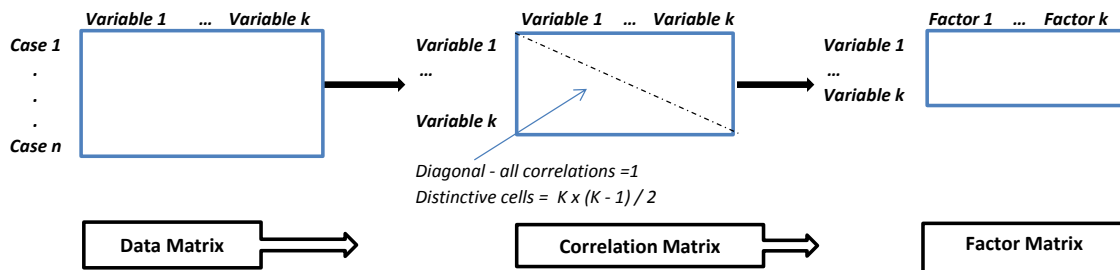


Figure 5 Matrix Process in EFA

For simplicity, suppose 2 factors were determined in the sample used to address research question number 3.

- What were the common factors in patients with acute decompensated heart failure and low systolic blood pressure?

These factors represent unobservable latent variables. Patients with decompensated heart failure with a low systolic blood pressure and several other measurable evaluations, signs and symptoms were seen in the emergency room. Let the other measurable evaluations, signs and symptoms be represented by the term heart failure variables. Such a patient randomly selected from a large population would also have a random set of heart failure variables. The average score in each of the heart failure variables for a patient with a particular level of factor one and a particular level of factor two is x multiplied by factor one plus y multiplied by factor two. This is a linear combination of the two factors (see below). The x and y values, or factor loadings are the same for other patients with heart failure who present to the emergency room with decompensated heart failure and a low systolic blood pressure (Ivancevic & Ivancevic, 2007, p. 50).

$$|(x,y)| \leq ||x|| \cdot ||y|| \quad (1)$$

The seven stages in factor analysis design will be employed in this analysis.

According to Hair et al. (2006) the stages are:

- State 1: Objectives
- Stage 2: Designing Factor Analysis
- Stage 3: Assumptions in Factor Analysis
 - 5 variables for each factor
 - Sample must have more observations than variable (minimum $N=50$)
 - Maximize the number of observations per variable (10 or more)

- Assess data and perform pairwise deletion of missing data or imputation with overall mean if missing data are not too numerous
 - A strong conceptual foundation need to support the assumption that a structure does exist before the factor analysis is performed
 - A statistically significant Bartlett's test of sphericity ($\text{sig} > 0.05$) indicates that sufficient correlations exist among the variable to proceed
 - Cronbach alpha, a measure of sampling adequacy (MSA) values must exceed 0.50 for both the overall test and each individual variable
- Stage 4: Deriving Factors and Assessing overall Fit
- Choosing a model
 - Criteria for number of factors to extract
 - Latent root criteria
 - Scree Test Criterion
- Stage 5: Selecting a Rotational Method
- Assess Communalities
- Stage 6: Validation of Factor Analysis
- Use of a confirmatory perspective
 - Assessing Factor Structure Stability
- Stage 7: Additional Uses of Factor Analysis results.
(pp. 108-116)

Cronbach defined Cronbach alpha as, "shown to be the mean of all split-half coefficients resulting from different splittings of a test. Alpha is therefore an estimate of the correlation between two random samples of items from a universe of items like those in the test. Alpha is found to be an appropriate index of equivalence and, except for very short tests, of the first-factor concentration in the test." (Cronbach, 1951, p. 297). By splitting the data randomly, the data is split in two in every possible way and the correlation coefficient for each split is computed. The average of the values obtained used as an index of reliability, Cronbach's alpha, is the most common measure of scale reliability. This value estimates the amount of measurement error in a test. The correlation is squared and subtracted from one to produce the index of measurement error. Factor 8 has a reliability of 0.687 ($\alpha=0.687$) and therefore there is a 0.528031

variance or random error in values (variables abstracted) ($0.687 * 0.687 = 0.471969$; $1.00 - 0.471969 = 0.528031$). The measurement error for Factor 9 is 0.605616 indicating that as the estimate of reliability decreases, the fraction of a value (variable) that is attributable to error will increase and vice-versa. The reliability of a variable is measured on the full cohort of patients rather than on one individual patient.

The computational formula for the standardized Cronbach's alpha:

$$\alpha = \frac{N \cdot \bar{c}}{\bar{v} + (N - 1) \cdot \bar{c}}$$

Where N is equal to the number of items, c-bar is the average inter-item covariance among the items and v-bar equals the average variance.

CHAPTER 4 RESULTS

Dimension reduction of factors via SPSS (ver 23) was conducted on all cases regardless of presenting systolic blood pressure (Group 1), cases with normal to high systolic blood pressure (Group 2) and cases with low systolic blood pressure (Group 3) separately, for a total of groups. The descriptive options for the correlation matrix requested was coefficients, significance levels, determinant and KMO and Bartlett's test of sphericity. The method employed was principal components and the analysis of the correlation method was selected. The scree plot and Eigenvalues greater than 1 with a maximum of 250 iteration for convergence were selected. The rotation method selected was Varimax with a maximum of 250 iterations. Varimax, an orthogonal rotation method was chosen since it produces independent factors minimizing the number of variables that have high loadings on each factor resulting in a solution with no multicollinearity. Thus the interpretation of the factors is simplified. Score options for variables selected were Bartlett and display of the factor score coefficient matrix. Options selected were to replace missing values with the mean, sorting the coefficient by size and suppressing coefficients less than $|.4|$.

The initial analysis resulted in correlation matrices that were not positive definitive. A second analysis was conducted by removing all variables with linear dependency defined as 1.0 on the inter-item correlation matrix when each of the resulting factors were run for a reliability analysis using the alpha model. Variables that loaded on more than one factor, with a factor loading less than $|.4|$, or did not load were removed. Missing data were replaced with the mean which may have contributed to linear dependency.

Initial Analysis

Group 1: All Cases Regardless of Systolic Blood Pressure at Presentation

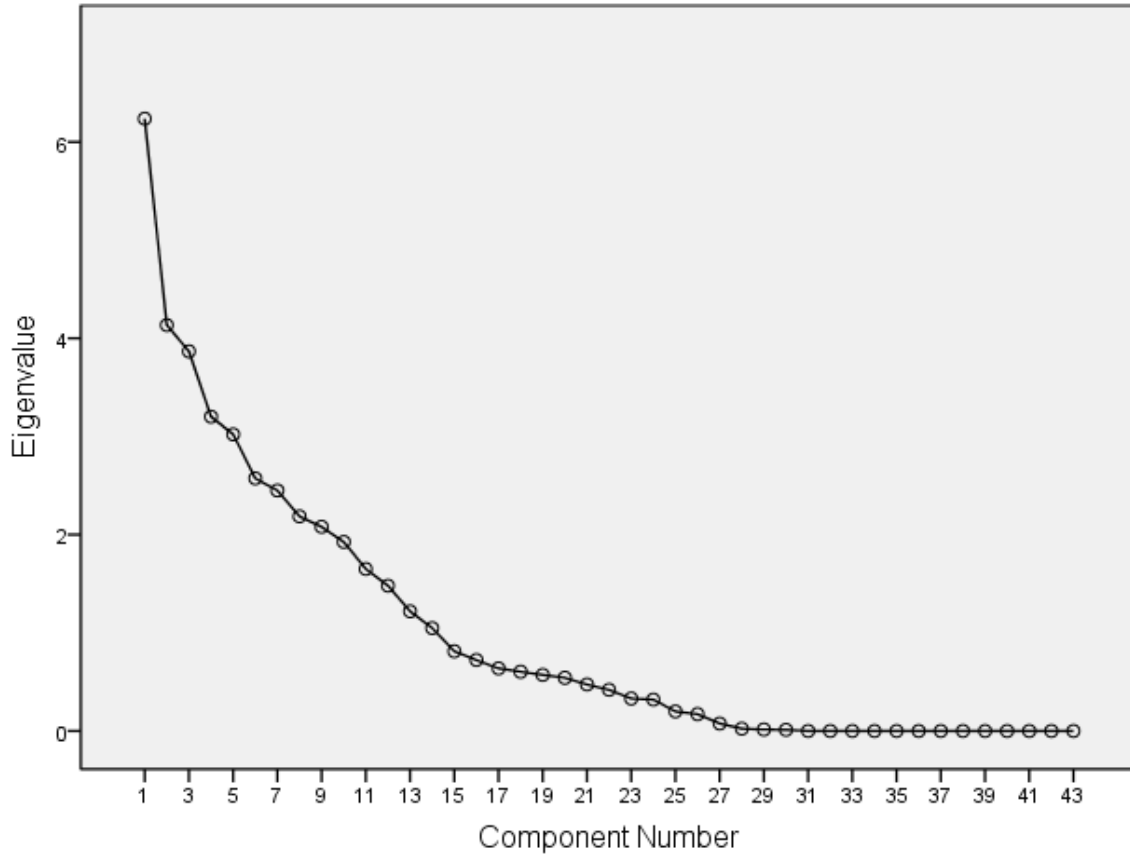


Figure 6 Scree Plot All Cases Regardless of Systolic Blood Pressure at Presentation

Table 6
Group 1 Total Variance Explained for All Cases Regardless of Systolic Blood Pressure at Presentation

Component	Extraction Sums of Squared			Rotation Sums of Squared		
	Total	Loadings		Total	Loadings	
		% of Variance	Cumulative %		% of Variance	Cumulative %
1	5.067	13.333	13.333	4.721	12.422	12.422
2	3.922	10.321	23.654	3.756	9.885	22.307
3	3.223	8.480	32.135	3.087	8.124	30.431
4	3.097	8.150	40.284	2.593	6.823	37.254
5	2.761	7.266	47.550	2.423	6.376	43.630
6	2.650	6.974	54.524	2.350	6.185	49.815
7	2.463	6.483	61.007	2.170	5.711	55.525
8	2.157	5.677	66.684	2.069	5.444	60.969
9	1.972	5.189	71.873	2.060	5.421	66.390
10	1.774	4.669	76.542	2.038	5.363	71.753
11	1.523	4.008	80.551	2.012	5.295	77.049
12	1.384	3.643	84.194	1.999	5.262	82.310
13	1.177	3.098	87.292	1.893	4.982	87.292

Extraction Method: Principal Component Analysis.

Table 7
Rotated Component Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation

	Component												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Low Hematocrit	.978												
High Hematocrit	.978												
Low Hemoglobin	.957												
High RBC_Count	.932												
Low RBC_Count	.932												
Low Eosinophils, Absolute		.966											
High Eosinophils, Absolute		.966											
High Eosinophils – Rel (Diff)		.964											
Low Eosinophils – Rel (Diff)		.964											
High PT			.989										
Low PT			.989										
Low INR			.987										
SBP				.746									
Abnormal_Labs				.741									
DBP				.669									
Co-morbidity				.656									
Readmission_12 Mos				.580									
Ethnicity				-.477									
Low Mean_Corpuscula r+ Hemoglobin					.983								
High Mean_Corpuscula r_ Hemoglobin					.983								
High Troponin I						.945							
Tropinin						.930							
LVEDd						.758							

	Component												
	1	2	3	4	5	6	7	8	9	10	11	12	13
Lowco2							.953						
Carbon dioxide							.953						
Low Basophils – Abs (Diff)								.970					
High Basophils – Abs (Diff)								.970					
High WBC_Count									.980				
Low WBC_Count									.980				
Low Sodium										.987			
High Sodium										.987			
High Lymphocytes - Abs(Diff)											.972		
Low Lymphocytes – Abs (Diff)											.972		
Creatinine												.973	
Low creatinine												.973	
Illicit Drug (cocaine, heroin, etc., marijuana not included)													.774
alcoholol_abuse													.756
illicit drug (marijuana)													.740

Note: Extraction Method: Principal Component Analysis.
Rotation Method: Varimax with Kaiser Normalization.

Group 1: All Cases Regardless of Systolic Blood Pressure at Presentation

Table 8

Group 1 Factor 1 for Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation

	Low Hemotocrit	High Hemotocrit	Low Hemoglobin	High RBC_Count	Low RBC_Count
Low Hemotocrit	1.000	1.000	.987	.864	.864
High Hemotocrit	1.000	1.000	.987	.864	.864
Low Hemoglobin	.987	.987	1.000	.817	.817
High RBC_Count	.864	.864	.817	1.000	1.000
Low RBC_Count	.864	.864	.817	1.000	1.000

Table 9

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 1 Anemia*

Reliability Statistics

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.836	.980	5

Table 10

*Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 2 Heart Injury Lab Predictor*

	High Eosinophils - Rel (Diff)	High Eosinophils, Absolute	Low Eosinophils - Rel (Diff)	Low Eosinophils, Absolute
High Eosinophils - Rel (Diff)	1.000	.870	1.000	.870
High Eosinophils, Absolute	.870	1.000	.870	1.000
Low Eosinophils - Rel (Diff)	1.000	.870	1.000	.870
Low Eosinophils, Absolute	.870	1.000	.870	1.000

Table 11

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 2 Heart Injury Lab Predictor*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.754	.977	4

Table 12

*Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 3 Coagulopathy*

	HighPT	Low PT	Low INR
High PT	1.000	1.000	.987
Low PT	1.000	1.000	.987
Low INR	.987	.987	1.000

Table 13

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 3 Coagulopathy*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.836	.997	3

Table 14

*Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 All Factor 4 Severity*

	SBP	Abnormal_Labs	DBP	Co-morbidity	Readmission_12 Mos	Ethnicity
SBP	1.000	.381	.637	.264	.208	-.245
Abnormal_Labs	.381	1.000	.281	.447	.461	-.309
DBP	.637	.281	1.000	.221	.151	-.276
Co-morbidity	.264	.447	.221	1.000	.422	-.219
Readmission_12 Mos	.208	.461	.151	.422	1.000	-.256
Ethnicity	-.245	-.309	-.276	-.219	-.256	1.000

Table 15

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 All Factor 4 Severity*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.466	.503	6

Table 16

*Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 5 Hemoglobin Kidney Injury*

	HighMEAN_CORPUSCULAR_Hemoglobin	LowMEAN_CORPUSCULAR_Hemoglobin
Highmean_Corpuscular_Hemoglobin	1.000	1.000
Lowmean_Corpuscular_Hemoglobin	1.000	1.000

Table 17

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 5 Hemoglobin Kidney Injury*

Cronbach's Alpha	Cronbach's Alpha Based on	
	Standardized Items	N of Items
1.000	1.000	2

Table 18

*Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 6 Heart Injury*

	High Troponin I	Tropinin	LVEDd
High Troponin I	1.000	.999	.558
Tropinin	.999	1.000	.559
LVEDd	.558	.559	1.000

Table 19

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 6 Heart Injury*

Cronbach's Alpha	Cronbach's Alpha Based on	
	Standardized Items	N of Items
.069	.878	3

Table 20

*Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 7 Electrolyte Kidney Injury*

	LowCO2	Carbon Dioxide
Low CO2	1.000	1.000
Carbon Dioxide	1.000	1.000

Table 21

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 7 Electrolyte Kidney Injury*

Cronbach's Alpha	Cronbach's Alpha Based on	
	Standardized Items	N of Items
1.000	1.000	2

Table 22
Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 8 Inflammation B

	High Basophils - Abs (Diff)	Low Basophils - Abs (Diff)
High Basophils - Abs(Diff)	1.000	1.000
Low Basophils - Ab (Diff)	1.000	1.000

Table 23
Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 8 Inflammation B

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 24
Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 9 Incidental Hypertension

	High WBC_Count	Low WBC_Count
High WBC_Count	1.000	1.000
Low WBC_Count	1.000	1.000

Table 25
Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 9 Incidental Hypertension

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 26
Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 10 Natremia

	High Sodium	Low Sodium
High Sodium	1.000	1.000
Low Sodium	1.000	1.000

Table 27
Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 10 Natremia

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 28
Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 11 Inflammation L

	High Lymphocytes – Abs(Diff)	Low Lymphocytes – Abs(Diff)
High Lymphocytes – Abs(Diff)	1.000	1.000
Low Lymphocytes – Abs(Diff)	1.000	1.000

Table 29
Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 11 Inflammation L

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 30
Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 12 Kidney Injury

	Creatinine	Low Creatinine
Creatinine	1.000	1.000
Low Creatinine	1.000	1.000

Table 31
Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 12 Kidney Injury

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 32
Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
 Group 1 Factor 13 Abuse

	Tobacco_ Abuse	alcoholol_ abuse	Illicit Drug (cocaine, heroin, etc., marijuana not included)	Illicit Drug (marijuana)
Tobacco_Abuse	1.000	.350	.291	.298
alcoholol_abuse	.350	1.000	.419	.390
Illicit Drug (cocaine, heroin, etc., marijuana not included)	.291	.419	1.000	.391
Illicit Drug(marijuana)	.298	.390	.391	1.000

Table 33
*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 13 Abuse*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
0.654	.689	4

Group 2 All Cases with Normal to High SBP at Presentation*Table 34. Group 2 Rotated Component Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation*

	Component													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
High Hemotocrit	.982													
Low Hemotocrit	.982													
HgB	.966													
Low Hemoglobin	.966													
Low RBC_Count	.916													
High RBC_Count	.916													
BUN		.912												
Low BUN		.912												
Creatinine		.887												
Low Creatinine		.887												
Mag		.541												
Low Eosinophils – Rel (Diff)			.963											
High Eosinophils - Rel (Diff)			.963											
High Eosinophils, Absolute			.962											
Low Eosinophils, Absolute			.962											
High PT				.990										
Low PT				.990										
Low INR				.985										
Chloride					.943									
Low Chloride					.943									
Low Sodium					.857									
Low Basophils – Abs (Diff)						.949								
High Basophils – Abs (Diff)						.949								
High Basophils - Rel (Diff)						.873								
Low CO2							.930							
Carbon Dioxide							.930							

DEVELOPMENT OF THE RISK MODEL FOR RESIDENT TRAINING 67

	Component														
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	
Anion_Gap							-								
							.642								
Mean_Corpuscular_Hemoglobin_Low							.973								
Mean_Corpuscular_Hemoglobin_Low_WBC_Count_High									.970						
WBC_Count_High									.970						
Platelet_Count									.529						
SBP										.763					
DBP										.750					
Co-morbidity										.582					
Pre-admission med – diuretic										.556					
Ethnicity										-.497					
Potassium											.952				
High Potassium											.952				
Discharge_ARB												.979			
Admit_ARB												.976			
Gender													.667		
Age														-.553	
Pain Score (Category List)														.539	
Anion Gap															.681
Race															-.641
RR															
Preadmit_Thiazide_Diuretic															
Low ALT(SGPT)															

Extraction Method: Principal Component Analysis.

Rotation Method: Varimax with Kaiser Normalization.

a. Rotation converged in 7 iterations.

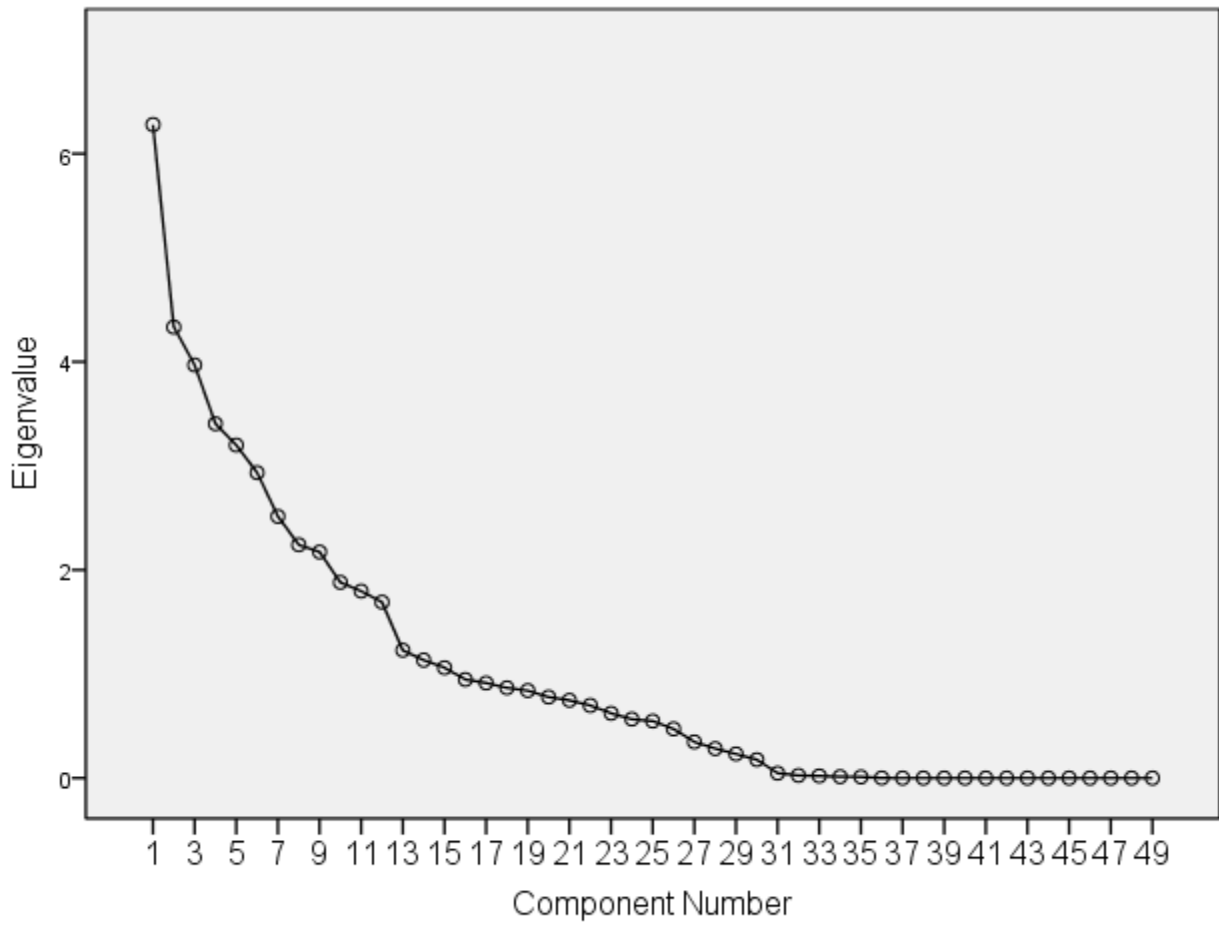


Figure 7 Scree Plot Cases with Normal to High Systolic Blood Pressure at Presentation

Table 35
Group 2 Total Variance Explained for Cases with Normal to High Systolic Blood Pressure at Presentation

Component	Total	Rotation Sums of Squared Loadings	
		% of Variance	Cumulative %
1	5.688	11.609	11.609
2	3.939	8.038	19.647
3	3.814	7.784	27.431
4	3.022	6.167	33.598
5	2.786	5.686	39.284
6	2.668	5.444	44.728
7	2.613	5.332	50.061
8	2.592	5.290	55.351
9	2.396	4.889	60.240
10	2.359	4.815	65.055
11	2.154	4.395	69.450
12	1.967	4.015	73.465
13	1.377	2.809	76.274
14	1.268	2.588	78.862
15	1.197	2.443	81.305

Note: Extraction Method: Principal Component Analysis.

Table 36

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 1 Anemia

	High Hemotocrit	Low Hemotocrit	HgB	Low Hemoglobin	Low RBC_Count
High Hemotocrit	1.000	1.000	.987	.987	.864
Low Hemotocrit	1.000	1.000	.987	.987	.864
HgB	.987	.987	1.000	1.000	.817
Low Hemoglobin	.987	.987	1.000	1.000	.817
Low RBC_Count	.864	.864	.817	.817	1.000

Table 37

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 1 Anemia

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.884	.985	5

Table 38

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 2 Kidney Injury

	BUN	Low BUN	Creatinine	Low Creatinine	Mag
BUN	1.000	1.000	.754	.754	.505
Low BUN	1.000	1.000	.754	.754	.505
Creatinine	.754	.754	1.000	1.000	.327
Low Creatinine	.754	.754	1.000	1.000	.327
Mag	.505	.505	.327	.327	1.000

Table 39

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 2 Kidney Injury

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.674	.910	5

Table 40

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 3 Heart Injury Lab Predictor

	Low Eosinophils - Rel (Diff)	High Eosinophils - Rel (Diff)	High Eosinophils, Absolute	Low Eosinophils, Absolute
Low Eosinophils - Rel (Diff)	1.000	1.000	.870	.870
High Eosinophils - Rel (Diff)	1.000	1.000	.870	.870
High Eosinophils, Absolute	.870	.870	1.000	1.000
Low Eosinophils, Absolute	.870	.870	1.000	1.000

Table 41

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 3 Heart Injury Lab Predictor

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.754	.977	4

Table 42

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 4 Coagulopathy

	High PT	Low PT	Low INR
High PT	1.000	1.000	.987
Low PT	1.000	1.000	.987
Low INR	.987	.987	1.000

Table 43

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 4 Coagulopathy

Cronbach's Alpha	Cronbach's Alpha Based on	
	Standardized Items	N of Items
.836	.997	3

Table 44

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor Electrolyte Imbalance

	Chloride	Low CHLORIDE	Low Sodium
Chloride	1.000	1.000	.729
Low Chloride	1.000	1.000	.729
Low Sodium	.729	.729	1.000

Table 45

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 5 Electrolyte Imbalance

Cronbach's Alpha	Cronbach's Alpha Based on	
	Standardized Items	N of Items
.932	.931	3

Table 46

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 6 Inflammation B

	Low Basophils _ Abs (Diff)	High Basophils – Abs (Diff)	High Basophils - Rel (Diff)
Low Basophils _ Abs (Diff)	1.000	1.000	.712
High Basophils – Abs (Diff)	1.000	1.000	.712
High Basophils - Rel (Diff)	.712	.712	1.000

Table 47
Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 6 Inflammation B

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	
		N of Items
.315	.927	3

Table 48
Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 7 Electrolyte Kidney Injury

	Carbon Dioxide	Low CO2	Anion Gap
Carbon Dioxide	1.000	1.000	-.160
Low CO2	1.000	1.000	-.160
Anion Gap	-.160	-.160	1.000

Table 49
Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 7 Electrolyte Kidney Injury

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	
		N of Items
.587	.468	3

Table 50
Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 8 Hemoglobin Kidney Injury

	High Mean_Corpuscular_Hemoglobin	Low Mean_Corpuscular_Hemoglobin
High Mean_Corpuscular_Hemoglobin	1.000	1.000
Low Mean_Corpuscular_Hemoglobin	1.000	1.000

Table 51

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 8 Hemoglobin Kidney Injury

Cronbach's Alpha	Cronbach's Alpha Based on	
	Standardized Items	N of Items
1.000	1.000	2

Table 52

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 9 Incidental Hypertension

	Low WBC_Count	High WBC_Count	High Platelet_Count
Low WBC_Count	1.000	1.000	.378
High WBC_Count	1.000	1.000	.378
High Platelet_Count	.378	.378	1.000

Table 53

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 9 Incidental Hypertension

Cronbach's Alpha	Cronbach's Alpha Based on	
	Standardized Items	N of Items
.089	.809	3

Table 54

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 10 Severity

	SBP	DBP	Co-morbidity	Preadmit_Thiazide_ Diuretic	Ethnicity
SBP	1.000	.637	.264	.126	-.245
DBP	.637	1.000	.221	.101	-.276
Co-morbidity	.264	.221	1.000	-.004	-.219
Preadmit_Thiazide_ Diuretic	.126	.101	-.004	1.000	-.103
Ethnicity	-.245	-.276	-.219	-.103	1.000

Table 55
Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 10 Severity

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.441	.209	5

Table 56
Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 11 RAAS Activation

	Potassium	High Potassium
Potassium	1.000	1.000
High Potassium	1.000	1.000

Table 57
Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 11 RAAS Activation

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 58
Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 12 Severity Treatment

	Discharge_ARB	Admit_ARB
Discharge_ARB	1.000	.943
Admit_ARB	.943	1.000

Table 59
Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 12 Severity Treatment

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.970	.970	2

Table 60

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 13 Patient Characteristics

	Gender	Age	Pain Score (Category List)
Gender	1.000	-.108	.047
Age	-.108	1.000	-.256
Pain Score (Category List)	.047	-.256	1.000

Table 61

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 13 Patient Characteristics

Cronbach's Alpha ^a	Cronbach's Alpha Based on Standardized Items	N of Items
-.208	-.402	3

Note: ^aThe value is negative due to a negative average covariance among items. This violates reliability model assumptions.

Table 62

Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 14 Blood Pressure Risk

	Anion Gap	Race
Anion Gap	1.000	-.168
Race	-.168	1.000

Table 63

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 14 Blood Pressure Risk

Cronbach's Alpha ^a	Cronbach's Alpha Based on Standardized Items	N of Items
-.236	-.403	2

Note: ^aThe value is negative due to a negative average covariance among items. This violates reliability model assumptions.

Table 64
Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 15 Diuresis

	RR	Preadmit_Thiazide_Diuretic	Low ALT (SGPT)
RR	1.000	-.079	.132
Preadmit_Thiazide_Diuretic	-.079	1.000	-.060
Low ALT (SGPT)	.132	-.060	1.000

Table 65
Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 15 Diuresis

Cronbach's Alpha ^a	Cronbach's Alpha Based on	
	Standardized Items	N of Items
-.036	-.007	3

Note: ^aThe value is negative due to a negative average covariance among items. This violates reliability model assumptions.

Group 3 All Cases with Low SBP at Presentation

Group 3: Cases with Low Systolic Blood Pressure at Presentation

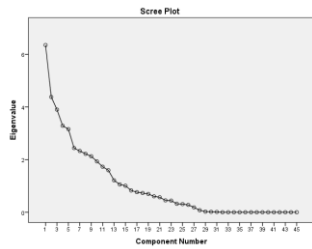


Figure 8 Scree Plot Cases with Low Systolic Blood Pressure at Presentation

Table 66
Total Variance Explained for Cases with Low Systolic Blood Pressure at Presentation

Component	Rotation Sums of Squared Loadings		
	Total	% of Variance	Cumulative %
1	5.654	12.564	12.564
2	3.781	8.402	20.966
3	3.769	8.375	29.341
4	3.261	7.246	36.587
5	2.669	5.931	42.518
6	2.449	5.443	47.962
7	2.428	5.396	53.358
8	2.257	5.017	58.374
9	2.127	4.726	63.101
10	2.039	4.530	67.631
11	2.028	4.508	72.139
12	1.868	4.151	76.290
13	1.818	4.040	80.330
14	1.427	3.171	83.501
15	1.127	2.504	86.005

Extraction Method: Principal Component Analysis.

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	Component															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
						.937										
LVEDd						.750										
Calcium							.950									
Low Calcium							.950									
Low Albumin							.535									
Low CO2								.965								
Carbon Dioxide								.965								
Low																
Platelet_Count										.970						
High																
Platelet_Count										.970						
High Sodium											.989					
Low Sodium											.989					
SBP												.857				
DBP												.825				
Co-morbidity													.751			
SYNCOPE													.710			
Readmission_12														.634		
Mos																
LOS															.846	
loop diuretic															.690	
Anion Gap																.800
Race																-
																.519

Extraction Method: Principal Component Analysis.

Rotation Method: Varimax with Kaiser Normalization.

a. Rotation converged in 7 iterations.

Table 68
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 1 Anemia

	Low Hemotocrit	High Hemotocrit	HgB	Low Hemoglobin	High RBC_Count	Low RBC_Count
Low Hemotocrit	1.000	1.000	.987	.987	.864	.864
High Hemotocrit	1.000	1.000	.987	.987	.864	.864
HgB	.987	.987	1.000	1.000	.817	.817
Low Hemoglobin	.987	.987	1.000	1.000	.817	.817
High RBC_Count	.864	.864	.817	.817	1.000	1.000
Low RBC_Count	.864	.864	.817	.817	1.000	1.000

Table 69
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 1 Factor 1 Anemia

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.872	.984	6

Table 70
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 2 Kidney Injury

	Low Creatinine	Creatinine	Low BUN	High Magnesium
Low Creatinine	1.000	1.000	.754	.327
Creatinine	1.000	1.000	.754	.327
Low BUN	.754	.754	1.000	.505
High Magnesium	.327	.327	.505	1.000

Table 71

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 1 Factor 2 Kidney Injury*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.199	.863	4

Table 72

*Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at
Presentation
Group 3 Factor 3 Heart Injury Lab Predictor*

	High Eosinophils - Rel (Diff)	High Eosinophils, Absolute	Low Eosinophils - Rel (Diff)	Low Eosinophils, Absolute
High Eosinophils - Rel (Diff)	1.000	.870	1.000	.870
High Eosinophils, Absolute	.870	1.000	.870	1.000
Low Eosinophils - Rel (Diff)	1.000	.870	1.000	.870
Low Eosinophils, Absolute	.870	1.000	.870	1.000

Table 73

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 1 Factor 3 Heart Injury Lab Predictor*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.754	.977	4

Table 74
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 4 Coagulopathy

	Low PT	High PT	High PTT	Low INR
Low PT	1.000	1.000	.403	.987
High PT	1.000	1.000	.403	.987
High PTT	.403	.403	1.000	.384
Low INR	.987	.987	.384	1.000

Table 75
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 1 Factor 4 Coagulopathy

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.682	.901	4

Table 76
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 5 Inflammation B

	High Basophils - Abs (Diff)	Low Basophils – Abs (Diff)	High Basophils - Rel (Diff)
High Basophils – Abs (Diff)	1.000	1.000	.712
Low Basophils – Abs (Diff)	1.000	1.000	.712
High Basophils - Rel (Diff)	.712	.712	1.000

Table 77
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 1 Factor 5 Inflammation B

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.315	.927	3

Table 78
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 6 Hemoglobin Kidney Injury

	High Mean_Corpuscular_ Hemoglobin	Low Mean_Corpuscular_ Hemoglobin
High Mean_Corpuscular_ Hemoglobin	1.000	1.000
Low Mean_Corpuscular_ Hemoglobin	1.000	1.000

Table 79
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 1 Factor 6 Hemoglobin Kidney Injury

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	
		N of Items
1.000	1.000	2

Table 80
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 7 Heart Injury

	High Troponin I	Tropinin	LVEDd
High Troponin I	1.000	.999	.558
Tropinin	.999	1.000	.559
LVEDd	.558	.559	1.000

Table 81
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 1 Factor 7 Heart Injury

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	
		N of Items
.069	.878	3

Table 82
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
 Group 3 Factor 8 Calcium-Albumin Complex

	Calcium	Low Calcium	Low Albumin
Calcium	1.000	1.000	.461
Low Calcium	1.000	1.000	.461
Low Albumin	.461	.461	1.000

Table 83
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
 Group 1 Factor 8 Calcium-Albumin Complex

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.847	.842	3

Table 84
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
 Group 3 Factor 9 Electrolyte Imbalance

	Low CO2	Carbon Dioxide
Low CO2	1.000	1.000
Carbon Dioxide	1.000	1.000

Table 85
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
 Group 3 Factor 9 Electrolyte Imbalance

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 86
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 10 Platelet Activation

	High Platelet_Count	Low Platelet_Count
High Platelet_Count	1.000	1.000
Low Platelet_Count	1.000	1.000

Table 87
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 10 Platelet Activation

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 88
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 All Factor 11 Natremia

	High Sodium	Low Sodium
High Sodium	1.000	1.000
Low Sodium	1.000	1.000

Table 89
Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 11 Natremia

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
1.000	1.000	2

Table 90
Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 12 Blood Pressure Measurement

	SBP	DBP
SBP	1.000	.637
DBP	.637	1.000

Table 91

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 12 Blood Pressure Measurement*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.761	.778	2

Table 92

*Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at
Presentation
Group 3 Factor 13 Severity*

	Co-morbidity	Syncopy	Readmission_12 Mos
Co-morbidity	1.000	.337	.423
Syncopy	.337	1.000	.183
Readmission_12 Mos	.423	.183	1.000

Table 93

*Reliability Statistics for Cases with Low Systolic Blood Pressure At Presentation
Group 3 Factor 13 Severity*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.288	.579	3

Table 94

*Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at
Presentation
Group 3 Factor 14 Treatment*

	LOS	loop diuretic
LOS	1.000	.353
loop diuretic	.353	1.000

Table 95

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 14 Treatment*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.015	.522	2

Table 96

Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation

Group 3 Factor 15 Blood Pressure Risk

	Anion Gap	Race
Anion Gap	1.000	-.168
Race	-.168	1.000

Table 97

Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 15 Blood Pressure Risk

Cronbach's Alpha ^a	Cronbach's Alpha Based on Standardized Items	N of Items
-.236	-.403	2

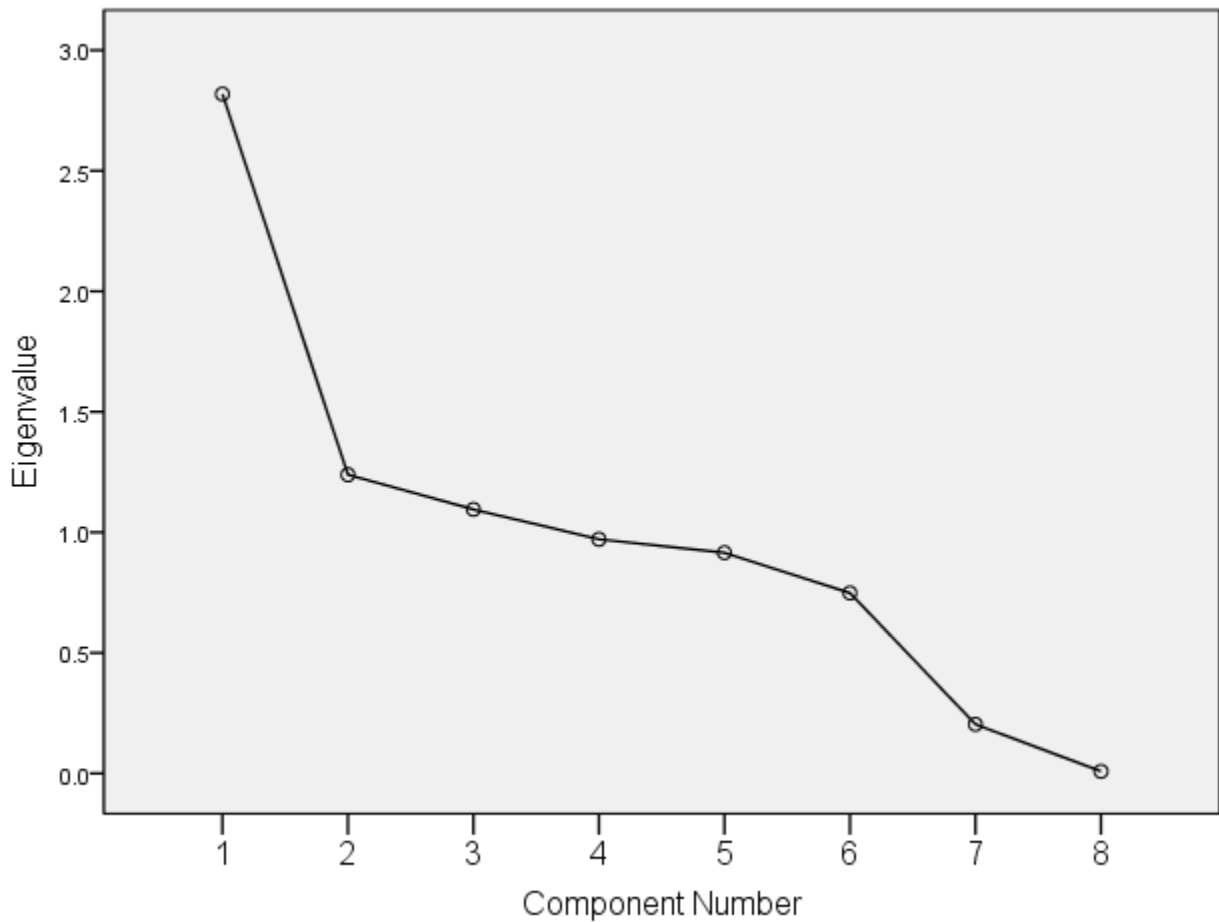
Note: ^aThe value is negative due to a negative average covariance among items. This violates reliability assumptions.

Second Analysis

Table 99

*Group 1: KMO and Bartlett's Test for All Cases Regardless of Systolic Blood Pressure at Presentation
Second Analysis*

Kaiser-Meyer-Olkin Measure of Sampling Adequacy.		.607
Bartlett's Test of Sphericity	Approx. Chi-Square	1598.70
	Df	9
	Sig.	.000



*Figure 9 Scree Plot of All Cases Regardless of Systolic Blood Pressure at Presentation
Second Analysis*

Table 100

Second Analysis for Total Variance Explained for All Cases Regardless of Systolic Blood Pressure at Presentation
Second Analysis

Component	Extraction Sums of Squared Loadings			Rotation Sums of Squared Loadings		
	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
	1	2.818	35.229	35.229	2.786	34.827
2	1.239	15.489	50.718	1.264	15.801	50.628
3	1.095	13.689	64.407	1.102	13.779	64.407

Extraction Method: Principal Component Analysis.

Table 101

Rotated Component Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Second Analysis

	Component		
	1	2	3
Low Hemotocrit	.988		
HgB	.972		
High RBC_Count	.922		
Co-morbidity		.790	
Tobacco_Abuse		.769	
High Basophils - Rel (Diff)			.690
Low Eosinophils - Rel (Diff)			.558
LVEDd			.558

Note: Extraction Method: Principal Component Analysis. Rotation Method: Varimax
 a. Rotation converged in 4 iterations.

Table 102

Group 1 Factor 1 for Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Second Analysis

	High Hemotocrit	HgB	High RBC_Count
High Hemotocrit	1.000	.987	.864
HgB	.987	1.000	.817
High RBC_Count	.864	.817	1.000

Table 103

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 1 Anemia
Second Analysis*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.680	.960	3

Table 104

*Group 1 Factor 1 for Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 2 Abuse
Second Analysis*

	Co-morbidity	Tobacco_Abuse
Co-morbidity	1.000	.251
Tobacco_Abuse	.251	1.000

Table 105

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 2 Abuse
Second Analysis*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.396	.401	2

Table 106

*Group 1 Factor 3 for Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation
Second Analysis*

	High Basophils - Rel (Diff)	Low Eosinophils - Rel (Diff)	LVEDd
High Basophils - Rel (Diff)	1.000	.059	.061
Low Eosinophils - Rel (Diff)	.059	1.000	.029
LVEDd	.061	.029	1.000

Table 107

*Reliability Statistics for All Cases Regardless of Systolic Blood Pressure at Presentation
Group 1 Factor 3 Inflammation
Second Analysis*

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.024	.136	3

Table 108

Group 2: Cases with Normal to High Systolic Blood Pressure at Presentation

Kaiser-Meyer-Olkin Measure of Sampling Adequacy.		.471
Bartlett's Test of Sphericity	Approx. Chi-Square	2388.33
	Df	5
	Sig.	.000

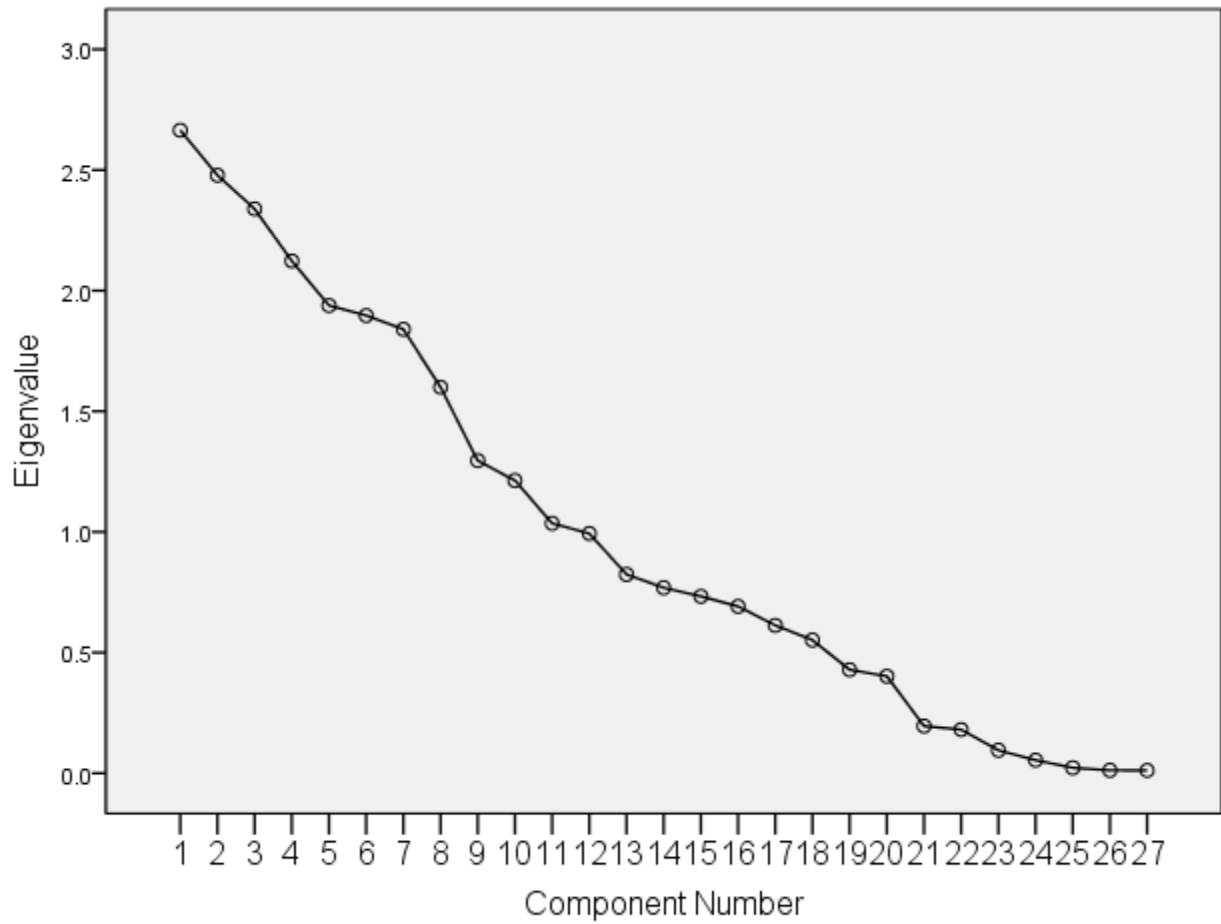


Figure 10 Scree Plot of Cases with Normal to High Systolic Blood Pressure at Presentation

Table 109

Total Variance Explained for Cases with Normal to High Systolic Blood Pressure at Presentation

Component	Extraction Sums of Squared Loadings			Rotation Sums of Squared Loadings		
	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	2.664	9.868	9.868	2.329	8.625	8.625
2	2.478	9.179	19.047	2.224	8.238	16.862
3	2.339	8.662	27.709	2.120	7.851	24.713
4	2.123	7.863	35.572	2.022	7.488	32.201
5	1.938	7.179	42.751	1.966	7.280	39.481
6	1.897	7.026	49.776	1.956	7.246	46.727
7	1.840	6.814	56.590	1.908	7.066	53.793
8	1.600	5.924	62.515	1.761	6.523	60.315
9	1.295	4.798	67.312	1.606	5.949	66.265
10	1.213	4.493	71.805	1.323	4.900	71.165
11	1.036	3.835	75.641	1.208	4.476	75.641

Note: Extraction Method: Principal Component Analysis.

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Table 110

Rotated Component Matrix for Total Variance Explained for Cases with Normal to High Systolic Blood Pressure at Presentation

	Component											
	1	2	3	4	5	6	7	8	9	10	11 12	
High PT	.953											
Low INR	.947											
High PTT	.605											
High WBC_Count		.958										
Low Neutrophils - Abs(Diff)		.932										
Low Monocytes - Abs(Diff)		.545										
Bun			.876									
Creatinine			.826									
Mag			.707									
Abnormal_Labs				.760								
CV				.730								
SBP				.708								
Discharge_HTN				.515								
Discharge_ARB					.977							
Admit_ARB					.976							
High RBC_Count						.954						
High Hemotocrit						.922						
High Eosinophils, Absolute							.962					
High Eosinophils - Rel (Diff)							.958					

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High Basophils - Rel (Diff)													.919
Component													
	1	2	3	4	5	6	7	8	9	10	11	12	
High Basophils - Abs (Diff)													.902
LVEDd													.883
High Troponin I													.858
Highmean_Corp uscular_ Hemoglobin													.850
Low Albumin													.506
Carbon Dioxide													.680
High Sodium													.618

Extraction Method: Principal Component Analysis.

Rotation Method: Varimax with Kaiser Normalization. (converged in 7 iterations)

Table 111
Group 2 Factor 1 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation Second Analysis

	High PT	Low INR	High PTT
High PT	1.000	.987	.403
Low INR	.987	1.000	.384
High PTT	.403	.384	1.000

Table 112
*Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 1 Coagulopathy
Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.024	.136	3

Table 113
Group 2 Factor 2 Inter-Item Correlation Matrix for C Cases with Normal to High Systolic Blood Pressure at Presentation
Second Analysis

	High WBC_Count	Low NEUTROPHILS – Abs (Diff)	Low MONOCYTES – Abs (Diff)
High WBC_Count	1.000	.964	.341
Low Neutrophils – Abs (Diff)	.964	1.000	.255
Low Monocytes – Abs (Diff)	.341	.255	1.000

Table 114
Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation
Group 2 Factor 2 Incidental Hypertension
Second Analysis

Cronbach's Alpha	Cronbach's Alpha Based on Standardized Items	N of Items
.458	.813	3

Table 115
Group 2 Factor 3 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation
Second Analysis

	BUN	Creatinine	Mag
BUN	1.000	.754	.505
Creatinine	.754	1.000	.327
Mag	.505	.327	1.000

Table 116

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 3 Kidney Injury

Second Analysis

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.128	.771	3

Table 117

Group 2 Factor 4 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Second Analysis

	Abnormal_:Labs	CV	SBP	Discharge_HTN
Abnormal_Labs	1.000	.481	.376	.220
CV	.481	1.000	.369	.193
SBP	.376	.369	1.000	.209
Discharge_HTN	.220	.193	.209	1.000

Table 118

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 4 Severity

Second Analysis

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.069	.640	4

Table 119

Group 2 Factor 5 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Second Analysis

	Discharge_ARB	Admit_ARB
Discharge_ARB	1.000	.943
Admit_ARB	.943	1.000

Table 120

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 5 Severity Treatment

Second Analysis

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.970	.970	2

Table 121

Group 2 Factor 6 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Second Analysis

	High RBC_Count	High Hemotocrit
High RBC_Count	1.000	.864
High Hemotocrit	.864	1.000

Table 122

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 6 Anemia

Second Analysis

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.342	.927	2

Table 123

Group 2 Factor 7 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Second Analysis

	High Eosinophils, Absolute	High Eosinophils - Rel (Diff)
High Eosinophils, Absolute	1.000	.870
High Eosinophils - Rel (Diff)	.870	1.000

Table 124

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 7 Heart Injury Lab Predictor

Second Analysis

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.263	.930	2

Table 125

Group 2 Factor 8 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Second Analysis

	High Basophils - Abs (Diff)	High Basophils - Rel (Diff)
High Basophils – Abs (Diff)	1.000	.712
High Basophils - Rel (Diff)	.712	1.000

Table 126

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 8 Inflammation B

Second Analysis

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.224	.832	2

Table 127

Group 2 Factor 9 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Second Analysis

	LVEDd	High Troponin I
LVEDd	1.000	.558
High Troponin I	.558	1.000

Table 128

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 2 Factor 9 Heart Injury

Second Analysis

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.046	.717	2

Table 129

Group 2 Factor 10 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Second Analysis

	High Mean_Corpuscular_Hemoglobin	Low Albumin
High Mean_Corpuscular_Hemoglobin	1.000	.214
Low Albumin	.214	1.000

Table 130

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

Group 1 Factor 10 Hemoglobin Kidney Injury

Second Analysis

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.138	.353	2

Table 131

Group 2 Factor 11 for Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation

Second Analysis

	Carbon Dioxide	High Sodium
Carbon Dioxide	1.000	.071
High Sodium	.071	1.000

Table 132

Reliability Statistics for Cases with Normal to High Systolic Blood Pressure at Presentation

*Group 2 Factor11 Electrolyte Kidney Injury
Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.263	.930	2

3: Cases with Low Systolic Blood Pressure at Presentation

Table 133

KMO and Bartlett's Test for Cases with Normal to High Systolic Blood Pressure at Presentation

Kaiser-Meyer-Olkin Measure of Sampling Adequacy.		.568
Bartlett's Test of Sphericity	Approx. Chi-Square	1491.29
	Df	4
	Sig.	.000

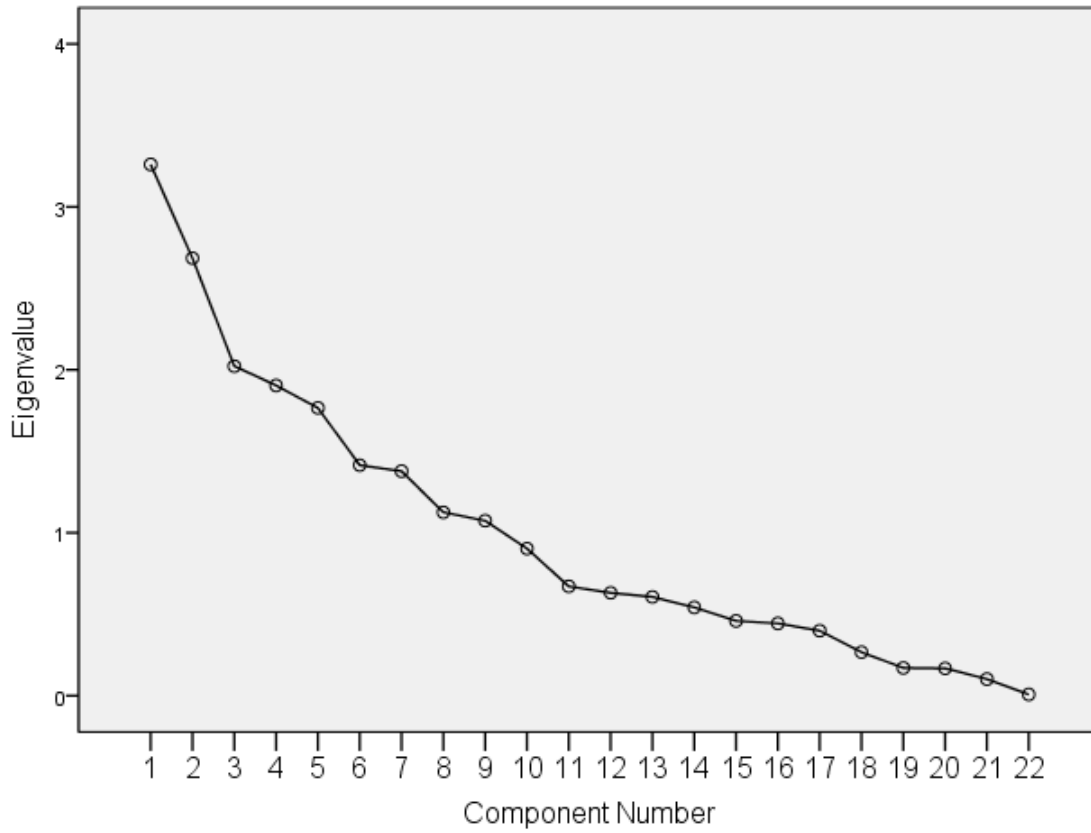


Figure 11 Scree Plot of Cases with Low Systolic Blood Pressure at Presentation

Table 134

Total Variance Explained for Cases with Low Systolic Blood Pressure at Presentation

Component	Extraction Sums of Squared Loadings			Rotation Sums of Squared Loadings		
	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	3.260	14.819	14.819	2.855	12.979	12.979
2	2.685	12.206	27.025	2.376	10.801	23.780
3	2.023	9.194	36.219	2.155	9.796	33.576
4	1.904	8.654	44.872	1.899	8.633	42.210
5	1.766	8.027	52.899	1.739	7.904	50.114
6	1.415	6.431	59.330	1.592	7.236	57.350
7	1.378	6.264	65.593	1.458	6.627	63.977
8	1.126	5.117	70.710	1.399	6.358	70.335
9	1.074	4.881	75.591	1.156	5.256	75.591

Extraction Method: Principal Component Analysis.

Table 135
Rotated Component Matrix for Cases with Low Systolic Blood Pressure at Presentation

	Component								
	1	2	3	4	5	6	7	8	9
High Hemotocrit	.973								
HgB	.956								
High RBC_Count	.915								
Abnormal_Labs		.791							
CV		.766							
Co-morbidity		.744							
Readmission_12 Mos		.742							
BUN			.878						
Creatinine			.846						
High Magnesium			.641						
High Eosinophils - Rel (Diff)				.966					
High Eosinophils, Absolute				.954					
High Basophils – Abs (Diff)					.927				
High Basophils - Rel (Diff)					.907				
LVEDd						.879			
High Troponin I						.866			
High PT							.829		
High PTT							.811		
Low Albumin								.808	
Calcium								.792	
LOS									.688
Carbon Dioxide									.673

Extraction Method: Principal Component Analysis.

Rotation Method: Varimax with Kaiser Normalization.^a

a. Rotation converged in 6 iterations.

Table 136

*Group 3 Factor 1 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Second Analysis*

	High Hemotocrit	HgB	High RBC_Count
High Hemotocrit	1.000	.987	.864
HgB	.987	1.000	.817
High RBC_Count	.864	.817	1.000

Table 13

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 1 Anemia
Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.263	.930	2

Table 138

*Group 3 Factor 2 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Second Analysis*

	Abnormal_Labs	CV	Co-morbidity	Readmission_12 Mos
Abnormal_Labs	1.000	.483	.443	.459
CV	.483	1.000	.436	.439
Co-morbidity	.443	.436	1.000	.423
Readmission_12 Mos	.459	.439	.423	1.000

Table 139
*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
 Group 3 Factor 2 Severity
 Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.595	.764	4

Table 140
*Group 3 Factor 3 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
 Second Analysis*

	BUN	High Magnesium	High Eosinophils - Rel (Diff)
BUN	1.000	.505	.036
High Magnesium	.505	1.000	.107
High Eosinophils - Rel (Diff)	.036	.107	1.000

Table 141
*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
 Group 3 Factor 3 Kidney Injury
 Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.034	.453	3

Table 142
*Group 3 Factor 4 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
 Second Analysis*

	High Eosinophils - Rel (Diff)	High Eosinophils, Absolute
High Eosinophils - Rel (Diff)	1.000	.870
High Eosinophils, Absolute	.870	1.000

Table 143

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 4 Heart Injury Lab Predictor
Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.263	.930	2

Table 144

*Group 3 Factor 5 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Second Analysis*

	High Basophils - Abs (Diff)	High Basophils - Rel (Diff)
High Basophils - Abs (Diff)	1.000	.712
High Basophils - Rel (Diff)	.712	1.000

Table 145

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 5 Inflammation B
Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.224	.832	2

Table 146

*Group 3 Factor 6 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Second Analysis*

	LVEDd	High Troponin I
LVEDd	1.000	.558
High Troponin I	.558	1.000

Table 147

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 6 Heart Injury
Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.046	.717	2

Table 148

*Group 3 Factor 7 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Second Analysis*

	High PT	High PTT
High PT	1.000	.403
High PTT	.403	1.000

Table 149

*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
Group 3 Factor 7 Coagulopathy
Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.504	.575	2

Table 150

*Group 3 Factor 8 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
Second Analysis*

	Low Albumin	Calcium
Low Albumin	1.000	.461
Calcium	.461	1.000

Table 151
*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
 Group 3 Factor 8 Albumin-Calcium Complex
 Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.630	.631	2

Table 152
*Group 3 Factor 9 for Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation
 Second Analysis*

	LOS	Carbon Dioxide
LOS	1.000	.057
Carbon Dioxide	.057	1.000

Table 153
*Reliability Statistics for Cases with Low Systolic Blood Pressure at Presentation
 Group 3 Factor 9 Electrolyte Kidney Injury
 Second Analysis*

Cronbach's Alpha		
Cronbach's Alpha	Based on Standardized Items	N of Items
.107	.108	2

Findings

After the study site's Crystal Report approval was granted by the Helios Administrative Group, data were obtained from discreet fields identified in Epic. Because Epic was recently installed, all of the elements identified required investigation by the principal performance management analyst in the Operational Analytics department at the study site. Some variables were stored in notes that are not easily queryable because the report was stored as an Adobe pdf file imported from other software programs such as Syngo, a Siemens imaging software. The major challenge for accessing patients coming to the emergency room was the electronic medical record change from EMStat®, a system that was well mature and robust with many of the data elements laid out, to the new system, EPIC where the data elements had to be found and validated for accuracy. All cases for patients admitted for decompensated heart failure (ICD 428.xx) for the 2014 year were selected. All cases were screened for entry criteria and the first 300 chronologically dated cases were identified.

EFA was conducted on the data abstracted from 300 electronic medical records stored in EPIC software and entered in an Excel spreadsheet. Patient inclusion was limited to those patients with decompensated heart requiring intravenous diuretic treatment. Data were abstracted via report analysis by the IT administrator and missing variables were manually abstracted by the author. All factors were reviewed by the Program Director of Advanced Heart Failure and Transplantation at the study site, for clinical meaningfulness.

Initial Analysis

Group 1 All Cases Regardless of SBP at Presentation

EFA conducted on cases regardless of SBP at presentation resulted in fourteen factors with explaining 87.292% of the variance (Table 6). Fourteen factors were also resulted in the Cattell scree test which plots the components on the X axis and the corresponding eigenvalues as the Y-axis (Figure 6). Reliability indices for each factor were high (i.e., > 0.7) as measured by Cronbach Alpha (α) (Tables 9, 11, 13, 15, 17, 19, 21, 23, 25, 27, 29, 31, and 33), a measure of internal consistency reliability, except for factor 8 with $\alpha=0.687$ (Table 23) and factor 9 with $\alpha=0.628$ (Table 25). Tables 8, 10, 12, 14, 16, 18, 20, 22, 24, 26, 28, 30, and 32 indicate correlation of each item with the sum of all remaining items in a matrix (Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation). Factors derived from this initial analysis of all cases regardless of SBP at presentation were evaluated and named based on clinical criteria. The factor names were carried throughout the remaining analyses and new factors were named with each analysis.

Factor 1 named Anemia consists of laboratory values for high and low hemotocrit, hemoglobin and low hemoglobin, and high and low red blood cells (RBC) Count. Factor 2 named Heart Injury Lab Predictor consists of laboratory values for high and low relative and absolute eosinophils. Factor 3 named Coagulopathy consists of laboratory values for high and low prothrombin time (PT), partial thromboplastin time (PTT) and international normalized ratio (INR). Factor 4 name Severity consists of SBP, grouping of abnormal laboratory results, DBP, grouping of co-morbidity, 12 month hospital readmission rate, and ethnicity (as defined in the site's electronic medical

record). Factor 5 named Hemoglobin Kidney Injury consists of laboratory values for high and low mean corpuscular hemoglobin. Factor 6 named Heart Injury consists of laboratory values for troponin and high troponin and echocardiogram measure of left ventricular diastolic diameter (LVEDd). Factor 7 named Electrolyte Kidney Injury consists of laboratory values for carbon dioxide and low carbon dioxide. Factor 8 named Inflammation B consists of laboratory values for high and low basophils. Factor 9 named Incidental Hypertension consists of laboratory values for high and low white blood Count (WBC). Factor 10 named Natremia consists of laboratory values for high and low sodium. Factor 11 named Inflammation B consists of laboratory values for high and low absolute lymphocytes. Factor 12 named Kidney Injury consists of laboratory values for creatinine and low creatinine. Factor 13 named Abuse consists of abuse of current or past illicit drug, alcohol or tobacco use.

Group 2 All Cases with Normal to High SBP at Presentation

EFA conducted on cases with normal to high SBP at presentation resulted in fifteen factors with explaining 81.305% of the variance (Table 35). Fifteen factors were also resulted in the Cattell scree test (Figure 7). Reliability indices for each factor resulted in significance as measured by Cronbach α of ≥ 0.7 (Tables 37, 39, 41, 43, 45, 47, 49, 51, 53 and 55, except for factor 8 with $\alpha = 0.689$ (Table 51) and factor 13 with $\alpha = 0.053$ (Table 61). Tables 36, 38, 40, 42, 44, 46, 48, 50, 52, 54, 56, 58, 60, 62, and 64 indicate correlation of each item with the sum of all remaining items in a matrix (Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation). The factors derived from the initial analysis of all cases regardless of SBP at presentation were all found except for the Severity Factor consisting of group of abnormal labs, grouping of comorbidity, 12 month readmission rate and implantable cardioverter defibrillator / Cardiac Resynchronization Therapy (ICD/CRT) implant, the Heart Injury Factor consisting of laboratory values for troponin, high troponin and echocardiogram measurement of left ventricular end diastolic diameter (LVEDd), Electrolyte Kidney Injury Factor consisting of laboratory values for low carbon dioxide and carbon dioxide, Natremia consisting of laboratory values for high and low sodium, Inflammation L consisting of laboratory values for high and low absolute lymphocytes, and Abuse consisting of abuse of current or past illicit drug, alcohol or tobacco use. Additional factors were revealed and these are Electrolyte Imbalance, Severity, renin-angiotensin-aldosterone system (RAAS), Severity of Treatment, Patient Characteristics, Blood Pressure Risk, and Diuresis.

Group 3 All Cases with Low SBP at Presentation

EFA conducted on cases with low SBP at presentation resulted in fifteen factors with explaining 86.005% of the variance (Table 67). Fifteen factors were also resulted in the Cattell scree test (Figure 8). Reliability indices for each factor resulted in significance as measured by Cronbach α of >0.7 (Tables 69, 71, 73, 75, 77, 79, 81, 83, 85, 87, 91, 93, 95, and 97) except for factor 13 with $\alpha = .579$ (Table 93), factor 14 with $\alpha=0.522$, and factor 15 with $\alpha= -.403$ (Table 97). Cronbach α will be negative whenever the sum of the individual item variances is greater than the scale variance and can be contributed to a large measurement error, low sample size, reverse coding, or variables that do not measure the same factor (as seen with negative covariance). Anion gap is related to race and race value obtained was -0.519 (Table 66). In this case, race may be inversely related to anion gap value. Tables 68, 70, 72, 74, 75, 78, 80, 82, 84, 86, 88, 90, 92, 94, and 96 indicate correlation of each item with the sum of all remaining items in a matrix (Inter-Item Correlation Matrix for Cases with Low Systolic Blood Pressure at Presentation). The factors derived from the initial analysis of all cases regardless of SBP at presentation were all found except for Electrolyte Kidney Injury Factor consisting of laboratory values for carbon dioxide and high sodium, Incidental hypertension Factor consisting of laboratory values for high WBC Count, low neutrophils (absolute) and low monocytes (absolute), Inflammation L Factor consisting of lab values for high and low lymphocytes, and Abuse Factor consisting of current or past illicit drug, alcohol or tobacco use.

Second Analysis

Sampling adequacy was tested with Kaiser-Meyer-Olkin (KMO) Measure of Sampling Adequacy. Bartlett's Test of Sphericity tests the hypothesis that the correlation matrix is an identity matrix indicating uncorrelated variables and is accepted if >0.5 . Significance levels less than the alpha value set at 0.05 allows rejection of the null hypothesis that the population matrix is an identity matrix. Rejection of the null hypothesis allows one to conclude that there are correlations in the data set appropriate for factor analysis.

Group 1 All Cases Regardless of SBP at Presentation

EFA conducted on cases regardless of SBP at presentation resulted in three factors with explaining 64.407% of the variance (Table 100). Three factors were also resulted in the Cattell scree test (Figure 9). Sampling adequacy was tested with Kaiser-Meyer-Olkin (KMO) Measure of Sampling Adequacy was 0.607 and Bartlett's Test of Sphericity was significant at 0.000 (Table 99). Reliability indices for each factor resulted in significance as measured by Cronbach α of >0.7 in only Factor 1 (Tables 103, 105 and 107). The three factors that resulted were the Anemia Factor consisting of laboratory values for low hematocrit, hemoglobin and high red blood cells (RBC) Count, Severity consisting of a group of comorbidity and tobacco use, and Inflammation consisting of laboratory values for high basophils, low eosinophils and echocardiogram measure of left ventricular diastolic diameter (LVEDd). Tables 102, 104, and 106 indicate correlation of each item with the sum of all remaining items in a matrix (Inter-Item Correlation Matrix for All Cases Regardless of Systolic Blood Pressure at Presentation). The variables for inflammation, laboratory values for high basophils and

low relative eosinophils, and echo measurement of left ventricular end diastolic diameter (LVEDd) in this group differ significantly from other groups in the previous analyses and resulted in a poor α of .103.

Group 2 All Cases with Normal to High SBP at Presentation

EFA conducted on cases with normal or high SBP at presentation resulted in eleven factors with explaining 75.641% of the variance (Table 109) Sampling adequacy was tested with Kaiser-Meyer-Olkin (KMO) Measure of Sampling Adequacy was 0.471 and Bartlett's Test of Sphericity was significant at 0.000 (Table 108). Reliability indices for each factor resulted in significance as measured by Cronbach α of >0.7 except for the Coagulopathy Factor, the Severity Factor, and the Hemoglobin Kidney Injury Factor (Tables 112, 118, and 130, respectively). The eleven factors that resulted were Coagulopathy consists of laboratory values for high and low prothrombin time (PT), partial thromboplastin time (PTT) and international normalized ratio (INR), Incidental hypertension consisting of laboratory values for high WBC Count, low neutrophils (absolute) and low monocytes (absolute), Kidney Injury consisting of laboratory values for blood urea nitrogen (BUN), creatinine, and magnesium, Severity consisting of a group of abnormal labs, cardiovascular disease, SBP and hypertension at discharge, angiotensin II receptor blocker (ARB) treatment consisting of use of angiotensin II receptor blocker (ARB) at admission and at discharge, Anemia consisting of laboratory values for high red blood cells (RBC) Count and high Hemotocrit, Heart Injury Laboratory Predictory consisting of laboratory values for high Eosinophils (absolute) and high Eosinophils (relative), Inflammation B consisting of laboratory values for high basophils (relative) and high basophils (absolute), Heart Injury consisting of laboratory

value for troponin and echocardiogram measure of left ventricular end diastolic diameter (LVEDd), Hemoglobin Kidney Injury consisting of laboratory values for high mean corpuscular hemoglobin and low albumin, and Electrolyte Kidney Injury consisting of laboratory values for carbon dioxide and high sodium. Tables 111, 113, 115, 117, 119, 121, 123, 125, 127, 129, and 131 indicate correlation of each item with the sum of all remaining items in a matrix (Inter-Item Correlation Matrix for Cases with Normal to High Systolic Blood Pressure at Presentation). Although the Coagulopathy Factor was found in previous analyses as significant, a weak α of .136 was found.

Group 3 All Cases with Low SBP at Presentation

EFA conducted on cases with low SBP at presentation resulted in nine factors with explaining 75.591% of the variance (Table 134). Sampling adequacy was tested with Kaiser-Meyer-Olkin (KMO) Measure of Sampling Adequacy was 0.568 and Bartlett's Test of Sphericity was significant at 0.000 (Table 133). Reliability indices for each factor resulted in significance as measured by Cronbach α of >0.7 except for the Kidney Injury, Coagulopathy Factor, Albumin-Calcium Factor and Electrolyte Kidney Injury Factor (Tables 141, 149, 151, and 153, respectively). The nine factors that resulted were Anemia consisting of laboratory values for high Hemotocrit, hemoglobin and high red blood cells (RBC) Count, Severity consisting of grouping of abnormal labs, cardiovascular disease, grouping of co-morbidity and 12 month readmission rate, Kidney Injury consisting of laboratory blood urea nitrogen (BUN), creatinine and high magnesium, Heart Injury Laboratory Predictor consisting of laboratory values for high Eosinophils (relative) and high Eosinophils (absolute), Inflammatory B for high basophils (absolute) and high basophils (relative), Heart Injury consisting of laboratory

value for high troponin and echocardiogram measure of left ventricular end diastolic diameter (LVEDd). The variables for inflammation, laboratory values for high basophils and low relative eosinophils, and echo measurement of left ventricular end diastolic diameter (LVEDd) in this group differ significantly from other groups in the previous analyses and resulted in a poor α of .103. Coagulopathy consisting of laboratory values with high prothrombin time (PT), high partial thromboplastin time (PTT), Calcium-Albumin Complex consisting of laboratory values for low albumin and calcium, and Treatment consisting of length of stay and laboratory value for carbon dioxide.

CHAPTER 5 DISCUSSION

The purpose of this study was to characterize patients hospitalized with acute decompensated heart failure with and without low systolic blood pressure using EFA. Direct and surrogate measurements are measured and dictate treatment. Medical laboratory results are key in the role in detecting, diagnosis and treatment. Blood, urine and imaging test results help to determine the presence, extent, or absence of disease and its effects and monitor the effectiveness of treatment. Although the Mayo Clinic, the American Clinical Laboratory Association (an industry organization), and the Center for Disease Control and Prevention (CDC) all cite that 70% of treatment decisions are based on laboratory test results (<http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5413a1.htm>; <https://web.archive.org/web/20070320220017/http://www.clinical-labs.org/issues/value/index.shtml>; <http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5413a1.htm>) no original source was found after an exhaustive review of the literature. The earliest reference to this statistic was found in an article by the Mayo clinic but no reference was provided (Forsman, 1996). Interestingly, this article focused on financial aspects of laboratory testing by managed care organizations rather than the source of the finding that laboratory results direct clinical decision making. The writer also consulted the Instruction Services Coordinator who is the Liaison Librarian to the College of Education and Department of Anthropology at the Wayne State University Library System and no original source was found for this widely used statistic. The discussion and impact of using such a statistic to drive patient care and issues with laboratory reporting errors is beyond the scope of this paper (Bonini, 2002).

Safety and cost-effective management is paramount, but there is a deluge of clinical data. The question investigated was could the method of data reduction using EFA elicit a parsimonious group of factors to summarize the relationship between these variables? A better understanding of the characteristics and outcomes of patients presented with acute decompensated heart failure with and without low systolic blood pressure could potentially lead to individualized treatment modalities tailored to effectively and economically improve care.

Interpretation of Findings

The major findings of the study found using EFA were that two factors, Anemia and Kidney Function were seen across the three groups. Group 1 consists of All Cases Regardless of SBP at Presentation. Group 2 consists of All Cases with Normal to High SBP at Presentation. Group 3 consists of All Cases with Low SBP at Presentation. EFA was conducted in a second analysis with the same 3 groups. The second analysis was run since the initial analysis resulted in correlation matrices that were not positive definitive. All variables with linear dependency defined as 1.0 on the inter-item correlation matrix when each of the resulting factors were run for a reliability analysis using the alpha model were removed. Variables that loaded on more than one factor, with a factor loading less than $|.4|$, or did not load were removed. Missing data were replaced with the mean which may have contributed to linear dependency. Again, Group 3, consisting of All Cases with Low SBP at Presentation factors were nearly the same as for the other two groups. There were two factors that were not found in Group 3. One is by definition, Incidental Hypertension since this group was defined as presenting with low systolic blood pressure. The other is abuse defined as illicit drug, alcohol or tobacco use. Group 3

cases are patients who are excluded from clinical trial enrollment as described in the review of the literature. However, the resulting factors from this EFA indicate that except for hypertension and abuse, the clinical characteristics of patients with low blood pressure encountered in the real-world clinical practice do not significantly differ.

Several individual factors that affect kidney function were also found. These factors include Heart Injury Lab Predictor, Kidney Injury, Coagulopathy, HgB Kidney Injury, Incidental HTN and Calcium-Albumin Complex. Anemia and Kidney Function factors affect blood pressure. Anemia decreases blood pressure and inversely, poor kidney function increases blood pressure (hypertension). Low blood pressure is a cause of poor kidney function but patients on dialysis were excluded from the study. As discussed in the review of the literature, anemia and kidney function are associated with poor outcomes including death in patients admitted with acute decompensated heart failure.

Recommendations for Future Research

The factors identified in each group using EFA can be tested in a future confirmatory factor analysis study. Once these factors are confirmed, an Acute Decompensated Heart Failure Risk Model can be developed for Emergency Room Resident Training within the context of evidence-based medicine. Over 20 years ago, articles appeared describing evidence-based medicine indicating the following:

A NEW paradigm for medical practice is emerging. Evidence-based medicine de-emphasizes intuition, unsystematic clinical experience, and pathophysiologic rationale as sufficient grounds for clinical decision making and stresses the examination of evidence from clinical research. Evidence-based medicine requires new skills of the physician, including efficient literature searching and the application of formal rules of evidence evaluating the clinical literature. (Guyatt, 1992, p. 2420)

However, there is criticism stating, “There are several reasons why the quality of the evidence for teaching EBM [evidence-based medicine] is so weak” (Hatala, 2002, p.1110). That is, the evidence for evidenced-based medicine is observational, the weakest form of evidence as espoused by the definition of evidence-based medicine. The pedagogical approach in medical education where instruction is provided by the experienced physician to the novice, namely the medical resident, is in conflict of adult learning theory leading to a contributing factor to the success or failure of teaching evidence-based medicine. The shift to student centered learning is based on adult learning theory (Spencer, 1999). Knowles’ (1970) exposure to the term andragogy was from, “a Yugoslavian adult educator [Dusan Savicevic] in the mid-sixties and printed in an article in *Adult Leadership* in 1968.” (p. 42). Andragogy is a Greek word referring to man or adult as opposed to a boy in pedagogy. (andra – meaning man and agogos – meaning learning). Knowles, Holton, and Swanson (2005) reported five underlying assumptions in the andragogy model:

1. The learner is self-directing. Adult learners want to take responsibility for their own lives, including the planning, implementing, and evaluating of their learning activities.
2. The learner enters an educational situation with a great deal of experience. This experience can be a valuable resource to the learner as well as to others. It needs to be valued and used in the learning process.
3. Adults are ready to learn when they perceive a need to know or do something in order to perform more effectively in some aspect of their lives. Their readiness to learn may be stimulated by helping them to assess the gaps between where they are now and where they want and need to be.
4. Adults are motivated to learn after they experience a need in their life situation. For that reason, learning needs to be problem-focused or task-centered. Adults want to apply what they have learned as quickly as possible. Learning activities need to be clearly relevant to the needs of the adult.

5. Adults are motivated to learn because of internal factors, such as self-esteem, recognition, better quality of life, greater self-confidence, the opportunity to self-actualize, and so forth. External factors, such as pressure from authority figures, salary increases, and the like, are less important. (p 299-300)

After an exhaustive literature review, articles discussing the use of teaching techniques based on findings using any particular statistical method, including EFA were not found. However, based on adult learning theory, specifically transformational learning, and this writer's nursing experience with both the target group, heart failure patients with or without low systolic blood pressure and extensive experience teaching medical residents in structured programs, the following information was used to support the use of the andragogical approach to teaching emergency room residents to use the risk model to be developed. The use of the andragogical technique is based on the results of the EFA conducted. If other results were found, then it is plausible an alternate teaching style may be appropriate and would have been explored.

The underlying assumptions in the andragogy model by Knowles, Holton, and Swanson (2005, p 294-295) are matched below to the method of learning of a risk model by emergency room residents. The second and third assumptions directly relate to the factors found in the EFA conducted since the literature reports that patients with and without low systolic pressure are different. It also important that the two common factors shared by patients regardless of blood pressure at presentation are directly related to blood pressure. Risk models are powerful tools for assessing biomedical significance but the importance of how to teach and use a risk model cannot be underestimated. The purpose of this study was to compare patients who presented with acute decompensated heart failure and to determine if patients with low systolic blood pressure share common

factors with patients with normal to high blood pressure at presentation. There is paucity in the literature based on the exclusion of patients with low systolic blood pressure in randomized clinical trials. Currently, treatment of patients with low systolic blood pressure is approached differently, yet both groups share common factors. Building on what emergency room residents may know, or determining whether there is a knowledge deficit is extremely important. Unlike problem-based learning which takes the pedagogical approach, emergency room residents already have the training and understanding to treat the target group of patients.

1. *“The learner is self-directing”* (Knowles, Holton, &Swanson, 2005, p. 299).

Emergency room residents are practicing under an attending physician to be able to independently treat patients including those who present with acute decompensated heart failure with or without low systolic blood pressure. The focus of their training is to become independent.

2. *“The learner enters an educational situation with a great deal of experience”* (Knowles, Holton, &Swanson, 2005, p. 299). Emergency room residents have not only completed medical school and criteria for residency matching but several other medical rotations including internal medicine in their training in order to practice in the emergency room. However, a step-by-step process layering information on what is already known (present level of understanding) by the learner or the required knowledge level is needed.

3. *“Adults are ready to learn when they perceive a need to know or do something in order to perform more effectively in some aspect of their lives. Their readiness to learn may be stimulated by helping them to assess the gaps between where they are now and where they want and need to be”* (Knowles, Holton, &Swanson, 2005,

p. 299). There is a paucity of data characterizing the patient with low systolic blood pressure and heart failure in the literature and thus, this was the basis of this retrospective study. Clinical trials exclude patients with heart failure and low systolic blood pressure and these patients are often treated differently than those who were included in randomized clinical trials testing medication in those with normal or high blood pressure. However, the results of the EFA conducted indicates that patients with and without low systolic blood pressure share common factors. These factors, anemia and kidney function also directly affect blood pressure. If emergency room residents do not know that these factors are shared, then the first step would be to educate them about this finding. Despite extensive education, training, and experience, the emergency physician will not have seen every variation of every condition that a patients presents with in the emergency department and they cannot be expected to have vast knowledge of every possibility or to have access to that knowledge readily. Having access and time to analyze all of the data to compare outcomes is not expected of a clinical provider and is left to researchers. That is why a risk model would be extremely important for the emergency room physician to access to assess patients quickly and safely prior to initiating, stopping or containing a treatment. Using statistical approaches such as EFA can elicit surprising new associations in the clinical data that a clinical treating physician would not expect. If emergency room residents do know from prior knowledge (underlying assumption 1), then the teacher would be adding to their knowledge base when teaching the residents the use of the risk model. Expanding on Knowles' contribution, Mezirow's (1991) transformational learning theory provides for an improved, meaningful learning experience for the adult learner and, "...the educator must actively encourage reflective

discourse through which learners can examine the justification for their meaning schemes and perspectives as well as focusing on the new data presented” (p. 201).

4. *“Adults are motivated to learn after they experience a need in their life situation”* (Knowles, Holton, & Swanson, 2005, p. 299). Decisions are made quickly in the emergency room and thus using a tool like a risk model would enhance the emergency room physician’s decision making abilities. Emergency workflow is based on expedient triage and treatment for either hospital admission or discharge. One problem of overcrowding seen in the emergency room is that patients leave without being seen or fully evaluated (Chan, Killeen, Kelly, & Guss, 2005). The factors resulting from this EFA will be tested in a confirmatory fashion and then used to develop a risk model which can be used as a biomedical model for treatment of the target group.

5. *“Adults are motivated to learn because of internal factors, such as self-esteem, recognition, better quality of life, greater self-confidence, the opportunity to self-actualize, and so forth”* (Knowles, Holton, & Swanson, 2005, p. 299). It is vital to determine if the treating emergency room resident is exposed to transformational learning in order to incorporate new information in their current practice. The practice of emergency medicine retains its own unique processes that are generally followed during the course of patient care and can be found in the institution’s policy and procedure guidelines. At each step, barriers can increase risk and threaten patient safety. From a physician’s standpoint, these may be potential medical-legal pitfalls. Learning opportunities to provide the quickest, safest medical care allows emergency room physicians more autonomy in their practice. Accurate detailed history taking, assessment of risk factors, test results and the patient’s individualized emergency room course are used to formulate

the medical treatment plan that can be expected by any reasonable physician and understood by any juror. Reflection back to prior learning to better understand the present circumstance is perhaps more fundamental to adult learning than explaining established meaning schemes.

Expanding on Knowles' contribution, Mezirow's (1991) transformational learning theory provides for an improved, meaningful learning experience for the adult learner and, "the educator must actively encourage reflective discourse through which learners can examine the justification for their meaning schemes and perspectives as well as focusing on the new data presented" (p.201). Teaching in a learning environment that allows for trial and error without the risk of negative criticism from both the physician teacher and colleagues encourages learner participation and willingness to participate in learning opportunities.

A discussion of how the brain learns is warranted at this point. The following figure (Figure 12) illustrates the Triune Brain. Tri refers to three, i.e., the reptilian (R-complex), paleomammalian (limbic system) and neomammalian (neocortex) brains and Une to one, i.e., one brain.

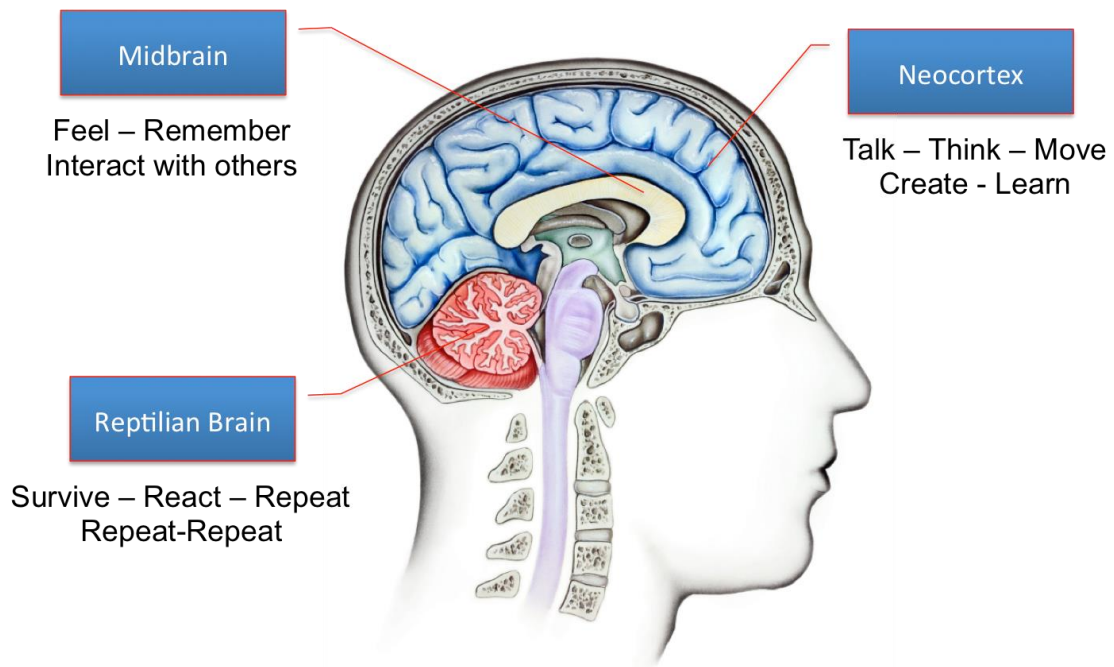


Figure 12: Triune Brain

<http://www.mini.ca/settings?ReturnUrl=http%3A%2F%2Fwww.mini.ca%2F>

The primitive, reptilian part of the brain dominates when there is perceived threat or under conditions of negative stress (distress as opposed to positive stress or eustress) and the response is reactionary without reasoning resulting in a downshifting. There is a literal shift down from the higher thinking of the neocortex to the social bond of the limbic and to the survival of the reptilian part. Learners who perceive a threat feel helpless and their thinking or emotions shift to behavior characterized as reptilian. For effective learning to occur, there must be a dynamic balance between the challenge of learning (i.e., stress) and a safe environment (Caine & Caine 1991). Physician leaders who create an environment of negative feelings using ridicule or belittling by making comparisons or unhealthy competition when training medical residents build a defensive and acquiescent cohort. Alternatively, a positive learning environment which provides for relaxed learning

and feelings of acceptance allows the learning to function in the neocortex where feeling associated with thinking is logical, sophisticated and thinking, creativity and reasoning is performed. This is especially important when introducing a new concept such as a new Acute Decompensated Heart Failure Risk Model. A break in the routine or a change in the curriculum may evoke emotion that may be upsetting to both the teacher and learner (MacLean, 1978). Teaching the use of a new Acute Decompensated Heart Failure Risk Model to Emergency Room Residents will be most successful if a transformative approach is utilized by the physician educator.

Methods for, and approaches to, medical education and general education have most significant ties dating back to the Flexnor report. The Carnegie Foundation for the Advancement of Teaching brought in Abraham Flexner to visit and evaluate medical school education in the U.S. and Canada at the turn of the 20th century. Flexor, a teacher from Louisville, Kentucky attended graduate school at Harvard from 1905-1906 and the following year studying the university-based medical education in Germany. In 1908, Flexner visited 155 medical schools in the U.S. and Canada over a 13 month period to write a report of his evaluation and assessment of physician training resulting in the closure of one third of the schools. The main concentrations written in the Flexnor report are to establish an association between medical schools and universities thus eliminating proprietary medical schools, mandate the basic science laboratory rotation, and ensure a progressive medical education as seen in elementary and secondary school education (Duffy, 2011). Competency-based instruction is built-in the Flexnerian program since medical residents are adult learners who tend to be self-directed and willing to assume responsibility in the learning process. As a supporter of the educator John Dewey, Flexor

understood medical education has analogies to elementary and secondary school education in, “that ‘the initiative lies with the learner’ and that education involves more than an accumulation of facts but a method of inquiry, thinking and problem solving.” (Arky, 2007, p. 91).

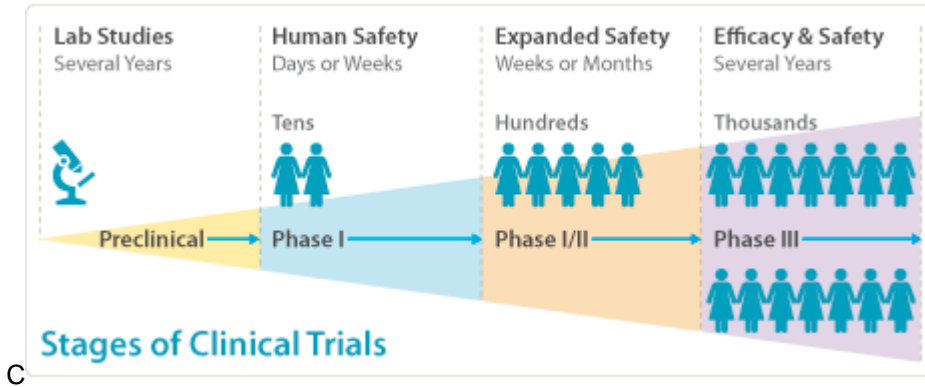
APPENDIX A**New York Heart Association (NYHA) Classification**

Functional Capacity	Objective Assessment
Class I. Patients with cardiac disease but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea, or anginal pain.	A. No objective evidence of cardiovascular disease.
Class II. Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.	B. Objective evidence of minimal cardiovascular disease.
Class III. Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnea, or anginal pain.	C. Objective evidence of moderately severe cardiovascular disease.
Class IV. Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.	D. Objective evidence of severe cardiovascular disease.

*The Criteria Committee of the New York Heart Association. *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels*. 9th ed. Boston, Mass: Little, Brown & Co; 1994:253-256.

APPENDIX B

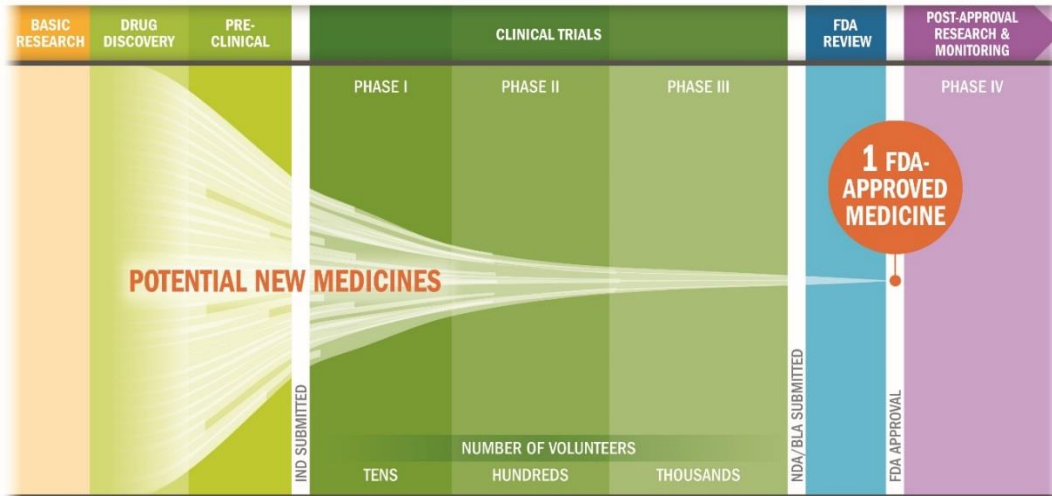
Clinical Trial Phases



<http://columbiasciencereview.com/2015/04/18/a-promising-cure-for-alzheimers-disease/>

THE BIOPHARMACEUTICAL RESEARCH AND DEVELOPMENT PROCESS

From drug discovery through FDA approval, developing a new medicine takes at least 10 years on average and costs an average of \$2.6 billion.* Less than 12% of the candidate medicines that make it into Phase I clinical trials will be approved by the FDA.



Key: IND: Investigational New Drug Application, NDA: New Drug Application, BLA: Biologics License Application

* The average R&D cost required to bring a new, FDA-approved medicine to patients is estimated to be \$2.6 billion over the past decade (in 2013 dollars), including the cost of the many potential medicines that do not make it through to FDA approval.

Source: PhRMA adaptation based on Tufts Center for the Study of Drug Development (CSDD) Briefing: "Cost of Developing a New Drug," Nov. 2014. Tufts CSDD & School of Medicine., and US FDA Infographic, "Drug Approval Process," <http://www.fda.gov/downloads/Drugs/ResourcesForYou/Consumers/UCM284393.pdf> (accessed Jan. 20, 2015).

<http://www.phrma.org/innovation/clinical-trials>

APPENDIX C

Henry Ford Hospital Internal Review Board (IRB) Yearly Initial IRB Approval



New Protocol Application

All submissions must be complete and typewritten. Investigators are responsible for utilizing the most current versions of IRB forms and the IRB has the authority to refuse out of date forms.

Section 1: GENERAL INFORMATION

HFHS Principal Investigator (PI): Zora Cvetkovski-Injic Department (select from drop downs):

Hematology/Oncology Division: Hematology/Oncology

Phone or pager: 313-352-0901

PI E-mail address: zcvetko1@hfhs.org

Are you employed by HFHS or a HFH Medical Group physician?

[X] Yes [] No (if not, you will need to contact Research Administration for assistance)

Entire Project Title (no acronyms): Development of the Acute Decompensated Heart Failure Risk Model for Emergency Room Resident Training

Contact Person: Zora Cvetkovski-Injic Contact phone #: 313-352-0901 Contact E-mail address: zcvetko1@hfhs.org

Location to send correspondence or pick-up (required): pick-up please

Grant title & Project Director (if different):

Sponsor/Funding source (name of agency, company, NIH or internal committee): n/a

Is this study PI initiated (the original idea of the HFHS PI)? [X] Yes [] No

Is this study federally funded (if so, attach grant proposal)? [] Yes [X] No Date submitted to funding agency:

If sponsor's grant number known, please supply: Multi-center study? [] Yes [] No

Performance Sites: [] Cottage [X] Detroit/Main [] Macomb [] W. Bloomfield [] Wyandotte [] All [] Other:

Research conducted: [X] Inpatient [] Outpatient [] In & Outpatient [] No direct patient contact [] Other:

What is submitted with this application?: [X] Protocol (version # 1) [] Investigator Brochure (version #) []

Consent Form (version) Other documents: data collection chart

THE REST OF THIS PAGE IS FOR IRB USE ONLY

IRB #: 9330

Review Type:

- [] Full Board
[X] Expedited: meets category/s [45 CFR 46.110(a)(b)]: S
[X] Approve
[] Withheld pending response:
[] member review [] administrative review
[] Expedited approval denied (requires full board review)
[] Exempt: meets category/s [45 CFR 46.101(b)]:

Consent/patient authorization:

- [] Required [X] Waived

APPROVAL STAMP
APPROVAL PERIOD
NOV 12 '14 NOV 11 '15
Institutional Review Board

The HFHS IRB has read & reviewed this protocol & finds this research is appropriate in design and meets the requirements of the Federal Guidelines, 45 CFR Part 46 and 21 CFR Part 50.

Chairperson or designee - Henry Ford Health System IRB
Abstentions:

Date: 11-12-14

APPENDIX D


Wayne State University Human Investigation Committee (HIC) Initial IRB Approval



IRB Administration Office
87 East Canfield, Second Floor
Detroit, Michigan 48201
Phone: (313) 577-1628
FAX: (313) 993-7122
<http://irb.wayne.edu>

NOTICE OF EXPEDITED APPROVAL

To: Zora Cvetkovski-Injic
College of Education
DRH/UHC Annex 2T

From: James Chinarian, M.D. or designee 
Chairperson, Medical Pediatric Institutional Review Board (MP2)

Date: November 25, 2014

RE: IRB #: 116014A
Protocol Title: Development of the Acute Decompensated Heart Failure Risk Model for Emergency Room Resident Training

Funding Source:
Protocol #: 1412013620

Expiration Date: November 24, 2015

The above-referenced Administrative Application request for you to use another IRB as the IRB of Record was APPROVED on 11/25/2014. The IRB Administration Office is in receipt of 1) the IRB Approval Letter from the IRB of Record Institution indicating that Wayne State University has been approved as an additional site under their approved protocol 2) Wayne State University's IRB Authorization to Use Another IRB for Protocol Approval Agreement with appropriate signatures; and 3) an Administrative Application appropriately completed and signed by the principal investigator.

- Administrative Application dated 11/12/2014) received in the IRB Office 11/17/2014)
- Authorization to Use Another IRB of Record
- Name of IRB of Record: Henry Ford Health System

- Please forward a copy of this approval memo and attached agreement to the collaborating institution's IRB upon receipt.
- All amendments and correspondence should be directed to the collaborating institution's IRB, unless instructed by that institution to send a copy to the WSU IRB Administration Office.
- Yearly Continuation approval from the collaborating institution must be submitted to the WSU IRB Administration Office along with an updated Administrative Application (check the continuation box at the top of the form). This submission should be received at least six weeks before the expiration date.
- Please reference the Protocol # (above) on all communication to the IRB Administration Office related to this research.
- Failure to receive approval for continuation before the expiration date will result in the automatic suspension of the approval of this protocol on the expiration date. Information collected following suspension is unapproved research and can never be reported or published as research data.

Enc: Copy of signed Agreement

APPENDIX E

Henry Ford Hospital Continuation Approval

APPENDIX F



Continuation/Final Report

All submissions must be sent electronically to: research_admin@hfhs.org
Investigators are responsible for utilizing the most current versions of IRB forms and the IRB has the authority to refuse out of date forms.

Please Indicate: [X] Continuation [] Final Report

SECTION 1 INVESTIGATOR INFORMATION

Principal Investigator (PI): Zora Cvetkovski-Injic
Department (select from the drop downs): Hematology/Oncology Division: Hematology/Oncology
Entire Project Title (no acronyms): Development of the Acute Decompensated Heart Failure Risk Model for Emergency Room Resident Training

IRB #: 9330 Current IRB Approval Period: 11/12/2014 – 11/11/2015

Location to send correspondence (required): zcvetko1@hfhs.org

Contact Person: Zora Cvetkovski-Injic Contact phone #: 313-352-0901 Contact e-mail: zcvetko1@hfhs.org

Current source of funding: none Is this study currently NIH funded? [X] No [] Yes (grant #)

If yes, was it originally submitted as such? [] No (if no, submit copy of grant) [] Yes

Current budget period (if federally funded): - Title of NIH grant (if different):

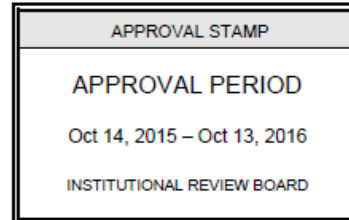
THE REST OF THIS PAGE IS FOR IRB USE ONLY

Type of IRB Review:

- [] Full Board
[X] Expedited (all expedited continuation & final reports are reviewed as informational items at fully convened IRB meetings)

Result of IRB Review:

- [X] Continuation Approved
[] Approval Withheld (see reason below)
[] Final Report Approved (Closure #)



The HFHS IRB has read & reviewed this protocol & finds this research is appropriate in design and meets the requirements of the Federal Guidelines, 45 CFR Part 46 and 21 CFR Part 50.

[Signature]
Chairperson or designee - Henry Ford Health System IRB

Date: 10/14/2015

Abstentions:
Comments:
Action required:

Wayne State University Human Investigation Committee (HIC) Continuation


Approval



IRB Administration Office
87 East Canfield, Second Floor
Detroit, Michigan 48201
Phone: (313) 577-1628
FAX: (313) 993-7122
<http://irb.wayne.edu>

NOTICE OF EXPEDITED CONTINUATION APPROVAL

To: Zora Cvetkovski-Injic
College of Education
IRH/UHC Annex 2T

From: James Paxton, M.D. or designee 
Chairperson, Medical Pediatric Institutional Review Board (MP2)

Date: October 27, 2015

RE: IRB #: 116014A

Protocol Title: Development of the Acute Decompensated Heart Failure Risk Model for Emergency Room Resident Training

Funding Source:

Protocol #: 1412013620

Expiration Date: October 26, 2016

Continuation for the above-referenced Authorization to Use Another IRB Program protocol and items listed below (if applicable) were APPROVED following Expedited Review by the Chairperson/designee of the Wayne State University Institutional Review Board (MP2) for the period of **10/27/2015 through 10/26/2016**. This approval does not replace any departmental or other approvals that may be required.

- Administrative Application
- Authorization to Use Another IRB of Record
- Name of IRB of Record: Henry Ford Health System

-
- ° Federal regulations require that all research be reviewed at least annually. You may receive a "Continuation Renewal Reminder" approximately two months prior to the expiration date; however, it is the Principal Investigator's responsibility to obtain review and continued approval **before** the expiration date. Data collected during a period of lapsed approval is unapproved research and can **never** be reported or published as research data. All changes or amendments to the above-referenced protocol require review and approval by the IRB **BEFORE** implementation.
 - ° Adverse Reactions/Unexpected Events (AR/UE) must be submitted on the appropriate form within the timeframe specified in the IRB Policy (<http://irb.wayne.edu>).

NOTE:

1. Upon notification of an impending regulatory site visit, hold notification, and/or external audit the IRB office must be contacted immediately.
2. Forms should be downloaded from the IRB website (<http://irb.wayne.edu>) at **each** use.

APPENDIX G

Collaborative Institutional Training Initiative (CITI) Certification

COLLABORATIVE INSTITUTIONAL TRAINING INITIATIVE (CITI PROGRAM) COURSEWORK REQUIREMENTS REPORT*

* NOTE: Scores on this Requirements Report reflect quiz completions at the time all requirements for the course were met. See list below for details. See separate Transcript Report for more recent quiz scores, including those on optional (supplemental) course elements.

- **Name:** Zora Injic (ID: 228337)
- **Email:** aj0418@wayne.edu
- **Institution Affiliation:** Detroit, MI-553 (ID: 142)
- **Institution Unit:** Advanced Heart Failure and Cardiac Transplantation
- **Phone:** 313-916-3520

- **Curriculum Group:** Human Research
- **Course Learner Group:** VA Human Subjects Protection
- **Stage:** Stage 1 - Basic Course

- **Report ID:** 616505
- **Completion Date:** 01/17/2008
- **Expiration Date:** 01/17/2008
- **Minimum Passing:** 80
- **Reported Score*:** 81

REQUIRED AND ELECTIVE MODULES ONLY	DATE COMPLETED
Good Research Practices for Protection of Human Subjects (With Special Emphasis on GCP), Module 1 (ID: 822)	01/17/08
Good Research Practices for Protection of Human Subjects, Module 2: Conducting Ethical Research (ID: 844)	01/17/08
Good Research Practices for Protection of Human Subjects, Module 3: Good Clinical Practice and VA Research (ID: 845)	01/17/08
Good Research Practices for Protection of Human Subjects, Module 4: Informed Consent (ID: 846)	01/17/08
Good Research Practices for Protection of Human Subjects, Module 5: Monitoring Subject Safety (ID: 847)	12/09/05
Good Research Practices for Protection of Human Subjects, Module 6: Records and Reports (ID: 849)	01/17/08
Good Research Practices for Protection of Human Subjects, Module 7: Managing Investigational Products (ID: 850)	12/09/05
Good Research Practices for Protection of Human Subjects, Module 8: Patient Privacy and Confidentiality (ID: 848)	01/17/08
Good Research Practices for Protection of Human Subjects, Module 9: Quality in VA Research- Past, Present, Future (ID: 851)	01/17/08

For this Report to be valid, the learner identified above must have had a valid affiliation with the CITI Program subscribing institution identified above or have been a paid Independent Learner.

CITI Program
 Email: citisupport@miami.edu
 Phone: 305-243-7970
 Web: <https://www.citiprogram.org>

Collaborative Institutional
 Training Initiative
 at the University of Miami

APPENDIX H**Collaborative Institutional Training Initiative (CITI) Refresher Certification****COLLABORATIVE INSTITUTIONAL TRAINING INITIATIVE (CITI PROGRAM)
COURSEWORK REQUIREMENTS REPORT***

* NOTE: Scores on this Requirements Report reflect quiz completions at the time all requirements for the course were met. See list below for details. See separate Transcript Report for more recent quiz scores, including those on optional (supplemental) course elements.

- **Name:** Zora Injic (ID: 228337)
- **Email:** aj0418@wayne.edu
- **Institution Affiliation:** Detroit, MI-553 (ID: 142)
- **Institution Unit:** Advanced Heart Failure and Cardiac Transplantation
- **Phone:** 313-916-3520

- **Curriculum Group:** Human Research
- **Course Learner Group:** VA Human Subjects Protection
- **Stage:** Stage 2 - Refresher Course

- **Report ID:** 807418
- **Completion Date:** 11/27/2007
- **Expiration Date:** 11/27/2010
- **Minimum Passing:** 80
- **Reported Score*:** 90

REQUIRED AND ELECTIVE MODULES ONLY	DATE COMPLETED
Biomed Refresher 1 – History and Ethical Principles (ID: 975)	11/27/07
Biomed Refresher 1 – Regulations and Process (ID: 981)	11/27/07
Biomed Refresher 1 – Informed Consent (ID: 990)	11/27/07
Biomed Refresher 1 – SBR Methodologies in Biomedical Research (ID: 982)	11/27/07
Biomed Refresher 1 – Records-Based Research (ID: 983)	11/27/07
Biomed Refresher 1 – Genetics Research (ID: 984)	11/27/07
Biomed Refresher 1 – Populations in Research Requiring Additional Considerations and/or Protections (ID: 985)	11/27/07
Biomed Refresher 1 – FDA-Regulated Research (ID: 987)	11/27/07
Biomed Refresher 1 – Human Subjects Research at the VA (ID: 988)	11/27/07
GCP Update Course: Module 1, Good Clinical Practices for VA Staff (ID: 1026)	11/27/07
GCP Update Course: Module 2, Accountability (ID: 1027)	11/27/07
GCP Update Course: Module 3, Informed Consent (ID: 1028)	11/27/07
GCP Update Course: Module 4, Safety Reporting (ID: 1029)	11/27/07
GCP Update Course: Module 5, Documentation (ID: 1030)	11/27/07
GCP Update Course: Module 6, Privacy and Confidentiality (ID: 1031)	11/27/07
GCP Update Course: Module 7, Application of GCP Concepts (ID: 1265)	11/27/07

For this Report to be valid, the learner identified above must have had a valid affiliation with the CITI Program subscribing institution identified above or have been a paid Independent Learner.

CITI Program
 Email: citisupport@miami.edu
 Phone: 305-243-7970
 Web: <https://www.citiprogram.org>

Collaborative Institutional Training Initiative (CITI) Refresher Certification

COLLABORATIVE INSTITUTIONAL TRAINING INITIATIVE (CITI PROGRAM)

COURSEWORK TRANSCRIPT REPORT**

** NOTE: Scores on this Transcript Report reflect the most current quiz completions, including quizzes on optional (supplemental) elements of the course. See list below for details. See separate Requirements Report for the reported scores at the time all requirements for the course were met.

- **Name:** Zora Injic (ID: 228337)
- **Email:** aj0418@wayne.edu
- **Institution Affiliation:** Detroit, MI-553 (ID: 142)
- **Institution Unit:** Advanced Heart Failure and Cardiac Transplantation
- **Phone:** 313-916-3520

- **Curriculum Group:** Human Research
- **Course Learner Group:** VA Human Subjects Protection
- **Stage:** Stage 2 - Refresher Course

- **Report ID:** 807418
- **Report Date:** 02/16/2016
- **Current Score**:** 93

REQUIRED, ELECTIVE, AND SUPPLEMENTAL MODULES	MOST RECENT
Biomed Refresher 1 – History and Ethical Principles (ID: 975)	11/27/07
Biomed Refresher 2 – History and Ethical Principles (ID: 511)	05/27/11
Biomed Refresher 1 – Regulations and Process (ID: 981)	11/27/07
Biomed Refresher 2 – Regulations and Process (ID: 512)	05/27/11
Biomed Refresher 1 – Informed Consent (ID: 980)	11/27/07
Biomed Refresher 2 – Informed Consent (ID: 514)	09/13/14
Biomed Refresher 1 – SBR Methodologies in Biomedical Research (ID: 982)	11/27/07
Biomed Refresher 1 – Records-Based Research (ID: 983)	11/27/07
Biomed Refresher 2 – Records-Based Research (ID: 516)	09/13/14
Biomed Refresher 1 – Genetics Research (ID: 984)	11/27/07
Biomed Refresher 2 – Genetics Research (ID: 518)	09/13/14
Biomed Refresher 1 - Populations in Research Requiring Additional Considerations and/or Protections (ID: 985)	11/27/07
Biomed Refresher 2 - Populations in Research Requiring Additional Considerations and/or Protections (ID: 519)	05/27/11
Biomed Refresher 1 – FDA-Regulated Research (ID: 987)	11/27/07
Biomed Refresher 1 – Human Subjects Research at the VA (ID: 988)	11/27/07
GCP Update Course: Module 1, Good Clinical Practices for VA Staff (ID: 1026)	11/27/07
GCP Update Course: Module 2, Accountability (ID: 1027)	11/27/07
GCP Update Course: Module 3, Informed Consent (ID: 1028)	11/27/07
GCP Update Course: Module 4, Safety Reporting (ID: 1029)	11/27/07
GCP Update Course: Module 5, Documentation (ID: 1030)	11/27/07
GCP Update Course: Module 6, Privacy and Confidentiality (ID: 1031)	11/27/07
GCP Update Course: Module 7, Application of GCP Concepts (ID: 1265)	11/27/07

For this Report to be valid, the learner identified above must have had a valid affiliation with the CITI Program subscribing institution identified above or have been a paid Independent Learner.

CITI Program
 Email: citisupport@miami.edu
 Phone: 305-243-7970
 Web: <https://www.citiprogram.org>

APPENDIX I

International Classification of Diseases, Ninth Revision, Clinical Modification

1. ▶ 428 Heart failure
2. ▶ 428.0 Congestive heart failure, unspecified convert 428.0 to ICD-10-CM
3. ▶ 428.1 Left heart failure convert 428.1 to ICD-10-CM
4. ▶ 428.2 Systolic heart failure
5. ▶ 428.20 Systolic heart failure, unspecified convert 428.20 to ICD-10-CM
6. ▶ 428.21 Acute systolic heart failure convert 428.21 to ICD-10-CM
7. ▶ 428.22 Chronic systolic heart failure convert 428.22 to ICD-10-CM
8. ▶ 428.23 Acute on chronic systolic heart failure convert 428.23 to ICD-10-CM
9. ▶ 428.3 Diastolic heart failure
10. ▶ 428.30 Diastolic heart failure, unspecified convert 428.30 to ICD-10-CM
11. ▶ 428.31 Acute diastolic heart failure convert 428.31 to ICD-10-CM
12. ▶ 428.32 Chronic diastolic heart failure convert 428.32 to ICD-10-CM
13. ▶ 428.33 Acute on chronic diastolic heart failure convert 428.33 to ICD-10-CM
14. ▶ 428.4 Combined systolic and diastolic heart failure
15. ▶ 428.40 Combined systolic and diastolic heart failure, unspecified convert 428.40 to ICD-10-CM
16. ▶ 428.41 Acute combined systolic and diastolic heart failure convert 428.41 to ICD-10-CM
17. ▶ 428.42 Chronic combined systolic and diastolic heart failure convert 428.42 to ICD-10-CM
18. ▶ 428.43 Acute on chronic combined systolic and diastolic heart failure convert 428.43 to ICD-10-CM
19. ▶ 428.9 Heart failure, unspecified convert 428.9 to ICD-10-CM

<http://www.icd9data.com/2012/Volume1/390-459/420-429/428/>

Appendix J

Epic Report Information Technology Request

The screenshot shows an Outlook email interface. The title bar reads "Report request for 'HFHS HF Admissions' received. - AfterMail (HTML)". The ribbon includes "FILE" and "AFTERMAIL" tabs. The ribbon contains various icons for actions like Ignore, Delete, Reply, Forward, Meeting, and more. The email header shows: "From: HFHS ServiceNow System", "To: Cvetkovski - Injic, Zora", "Subject: Report request for 'HFHS HF Admissions' received.", and "Sent: Mon 12/29/2014 3:54 PM".

Dear Zora Cvetkovski - Injic

Thank you for using the HFHS Report Request Form. This email is to confirm that we have received your recent request for "HFHS HF Admissions".

Please note that current status of your request is "waiting for Approval". Once the report request has been approved, you will be notified via email. A member of the Reporting team will contact you regarding next steps.

Order Details:
RITM330181: HFHS Report Request
Stage: Waiting for Approval
Short description: Report request for 'HFHS HF Admissions'

Description of the report (what?):
Add admissions for heart failure (ICD [428.XX](#)) for 2013.

Purpose of the Report (Why you need this report?):
Group patient characteristics (blood/urine labs, ejection fraction, drugs, etc.) to see if there are any differences between patients with low and without systolic blood pressure. Variables in report include dates (admission, discharge), in ER: age, BMI, blood pressure, heart rate, O2 sats, blood (BNP, creatinine, sodium, glucose, potassium, BUN, hemoglobin, eGFR, troponin, TSH, LDH, Calcium, magnesium, chloride, CO2, protein, ALT, AST, bilirubin, albumin, total protein), other diagnoses (co-morbidity history), current home medications, total number of days hospitalized.

Yours Sincerely,
HFHS Reporting Team

HFHS Report Request Created
Ref:MSG15617678

APPENDIX K

Epic Report Information Technology Approval

Report request for "HFHS HF Admissions" has been approved. - Message (HTML)

FILE **MESSAGE**

Ignore, Delete, Reply, Forward, Meeting, COVERAGE, To Manager, Done, Reply & Delete, Create New, Move, OneNote, Actions, Mark Unread, Categorize, Follow Up, Translate, Find, Related, Select, Zoom

Wed 5/20/2015 8:29 AM

HFHS ServiceNow System

Report request for "HFHS HF Admissions" has been approved.

To: Cvetkovski - Injic, Zora

You forwarded this message on 5/26/2015 9:37 PM.

Dear Zora Cvetkovski - Injic

This email is to confirm that your recent request for "HFHS HF Admissions" has been approved and has been authorized for development. A member of the Reporting team will contact you regarding next steps.

Order Details:
RITM330181: HFHS Report Request
Stage: Completed
Short description: Report request for 'HFHS HF Admissions'

Description of the report (what?):
Add admissions for heart failure (ICD [428.XX](#)) for 2013.

Purpose of the Report (Why you need this report?):
Group patient characteristics (blood/urine labs, ejection fraction, drugs, etc.) to see if there are any differences between patients with low and without systolic blood pressure.
Variables in report include dates (admission, discharge), in ER: age, BMI, blood pressure, heart rate, O2 sats, blood (BNP, creatinine, sodium, glucose, potassium, BUN, hemoglobin, eGFR, troponin, TSH, LDH, Calcium, magnesium, chloride, CO2, protein, ALT, AST, bilirubin, albumin, total protein), other diagnoses (co-morbidity history), current home medications, total number of days hospitalized.

Yours Sincerely,
HFHS Reporting Team

HFHS Report Request Approved

Ref:MSG17301224

HFHS ServiceNow System No Items

REFERENCES

- Abraham, W. T., Fonarow, G. C., Albert, N. M., Stough, W. G., Gheorghiade, M., Greenberg, B. H., . . . Young, J. B. (2008). Predictors of in-hospital mortality in patients hospitalized for heart failure: insights from the Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF). *J Am Coll Cardiol*, 52(5), 347-356. doi: 10.1016/j.jacc.2008.04.028
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ABSTRACT

**DEVELOPMENT OF THE ACUTE DECOMPENSATED HEART FAILURE RISK
MODEL FOR EMERGENCY ROOM RESIDENT TRAINING**

by

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May 2016

Advisor: Shlomo Sawilowsky, Ph.D.

Major: Education Evaluation and Research

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The purpose of this study was to characterize patients hospitalized with acute decompensated heart failure with and without low systolic blood pressure using exploratory factor analysis (EFA). Direct and surrogate measurements were measured. The aim was to use EFA for data reduction to elicit a parsimonious set of factors summarizing the relationships between variables by measuring intercorrelations of the clinical variables collected as part of standard care, and abstracted from electronic medical records.

A better understanding of the characteristics and outcomes of the target group could potentially lead to individualized treatment modalities tailored to effectively and economically improve care. Patients hospitalized are at a high risk for adverse outcomes after discharge.

Prospectively collected new data is expensive, labor-, and time- intensive while the use of existing data allows a quicker, more efficient and less expensive source. A large urban, academic teaching hospital was the study site. Wayne State University Human

Investigation Committee and Henry Ford Internal Review expedited review approval was obtained.

Eligible cases were patients hospitalized with a primary diagnosis of acute decompensated heart failure for the 2014 year. Variables collected were identified based on review of the literature, Framingham criteria, clinical relevance, and were routinely availability.

As is the case in empirical studies, determining sample size in EFA, a large sample size technique, is based on the minimum necessary to obtain reliable results from the analysis. Guidelines or a rule of thumb by expert opinions such as Gorsuch (1983) and Kline (1994) include absolute numbers of at least 100 cases.

Dimension reduction of factors via SPSS (ver 23) was conducted on all cases regardless of presenting systolic blood pressure (Group 1), cases with normal to high systolic blood pressure (Group 2) and cases with low systolic blood pressure (Group 3) separately, for a total of groups. All cases were screened for entry criteria and the first 300 chronologically dated cases were identified.

EFA was conducted on the data abstracted from 300 electronic medical records. The major findings of the study were that two factors, Anemia and Kidney Function were seen across the three groups. Several individual factors that affect kidney function were found. Data reduction using EFA is a highly pragmatic function. Computer software programs such as SPSS® allow for quick and easy computations and a large number of variables can be directly imported from databases such as Excel®. However, EFA is a complex procedure with fewer absolute guidelines or rules for selecting options compared to other statistical approaches. The steps taken were detailed, justified by the literature

reviewed and alternate choices were discussed. The seven stages in factor analysis design as outlined by Hair et al. (2006) were employed in this analysis.

The factors identified in each group using EFA can be tested in a future confirmatory factor analysis study. Once these factors are confirmed, an Acute Decompensated Heart Failure Risk Model can be developed for Emergency Room Resident Training within the context of evidence-based medicine.

The pedagogical approach in medical education where instruction is provided by the experienced physician to the novice, namely the medical resident, is in conflict of adult learning theory leading to a contributing factor to the success or failure of teaching evidence-based medicine. Risk models are powerful tools for assessing biomedical significance but the importance of how to teach and use a risk model cannot be underestimated. Building on what emergency room residents may know, or determining whether there is a knowledge deficit is extremely important. A step-by-step process layering information on what is already known (present level of understanding) by the learner or the required knowledge level is needed. The results of the EFA conducted indicates that patients with and without low systolic blood pressure share common factors. These factors, anemia and kidney function also directly affect blood pressure. If emergency room residents do not know that these factors are shared, then the first step would be to educate them about this finding. If emergency room residents do know from prior knowledge, then the teacher would be adding to their knowledge base when teaching the residents the use of the risk model as is described by Knowles, Holton, and Swanson (2005) as the first underlying assumption. The shift to student centered learning

is based on adult learning theory (Spencer, 1999) and transformational learning should be employed.

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