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Examining the impact of chest tube-related factors on the risk of nosocomial infections in a community based hospital.

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EXAMINING THE IMPACT OF CHEST TUBE-RELATED FACTORS ON THE RISK OF NOSOCOMIAL INFECTIONS IN A COMMUNITY BASED HOSPITAL

by

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ABSTRACT

Purpose: The study was conducted to investigate the impact of chest tube-related factors on the risk for development of nosocomial infections (NI) and to examine the independent predictors of NI in a community based ICU

Significance: Critically ill patients are at high risk of developing NI, which then leads to higher mortality rates, prolonged hospitalization, and increased costs. Chest tubes have been associated with increased risk of nosocomial pneumonia, but only one study reported that the presence of a chest tube was an independent risk factor for the development of nosocomial bloodstream infection (NBSI). Little is known about the specific chest tube-related factors that contribute to NI.

Methods: A retrospective, case-control review of 120 medical records of ICU patients was conducted. Two groups were compared using *t*-test and chi square comparisons on each of the study variables. Hierarchical logistic regression was used to determine which chest tube-related factors were independent predictors of NI, while adjusting for other known risk factors.

Results: Of the 40 cases, 92.5% (n = 37) had pneumonia, while 7.5% (n = 3) had NBSI. Chest tube-related factors accounted for 7.4% to 10.2% of the explained variance. The variable *chest tube days* was the only chest tube-related factor to be an independent predictor of NI (*OR* 5.79, 95% CI, 1.459-23.015). *Mechanical ventilation (MV)* (OR, 4.88; 95%CI, 1.8-13.1) and *outcome length of stay (LOS)* (*OR*, .724; 95%CI, .624-.839) were also found to be independent predictors of NI.

Conclusions: Risk of infection among patients with chest tubes increases as the duration of chest tube use increases. Infection is likely to happen early during admission, which necessitates stringent adherence to infection control strategies from initial phases of treatment.

DEDICATION

Words are inadequate to express my deepest appreciation to three very special people in my life – my husband Charles, my son Jonathan, and my daughter Allison. Your practical assistance has made my studies possible. Your encouragement kept the momentum going. Your pride inspired my success. My accomplishment is a credit to each of you. Thanks from the bottom of my heart.

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TABLE OF CONTENTS

ABSTRACT iii

DEDICATION v

ACKNOWLEDGEMENTS vi

LIST OF TABLES ix

LIST OF FIGURES x

CHAPTER I: INTRODUCTION 1
- Problem Statement 2
- Significance 3
- Purpose 5
- Conceptual Framework 5
- Research Questions 14

CHAPTER II: REVIEW OF LITERATURE 15
- Infectious Complications of Chest Tubes 15
- Chest Tube-related Factors 17
- Microbial Factors 18
- Other Therapy Related Factors 20
- Circumstantial Factors 24

CHAPTER III: METHOD 31
- Design 31
- Protection of Human Subjects 31
- Inclusion/Exclusion Criteria 32
- Data Collection Procedures 32
- Variable Definitions 33
- Data Analysis Procedures 34

CHAPTER IV: RESULTS 36
- Data Cleaning Procedures 36
- Sample Characteristics 39
- Univariate Analysis 39
- Multivariate Analysis 44
- Sub-analysis 55

CHAPTER V: DISCUSSION 57
- Limitations 66
- Implications and Recommendations 66
- Conclusion 70

REFERENCES 71

APPENDICES 90
Research Ethics Board Approvals 90
Data Collection Sheet 93

VITA AUCTORIS 95

LIST OF TABLES

Table 1	Dichotomous Variables with Category Split of Less than 90-10	37
Table 2	Treatment of Skewness for Variables Which were not Normally Distributed	38
Table 3	*T*-test Comparisons of Continuous Variables for Patients With NI and Patients Without NI	40
Table 4	Chi Square Comparisons of Categorical Variables for Patients With NI and Patients Without NI	42
Table 5	Decision Regarding Inclusion of Variables in Multivariate Regression Analysis Resulting from Examination for Collinearity	46
Table 6	Dummy coding of Chest Tube Size	47
Table 7	First Logistic Regression Model	49
Table 8	Final Stepwise Logistic Regression Model	54
Table 9	Classification Index of Observed and Predicted Values of the final Regression Model	55

LIST OF FIGURES

Figure 1	El-Masri's model of NBSI among critically ill trauma patients	8
Figure 2	Harris's model of nosocomial pneumonia	10
Figure 3	Conceptual model utilized in the current study, including additional factors, which may be of significance in a general ICU population	13

EXAMINING THE IMPACT OF CHEST TUBE-RELATED FACTORS ON THE RISK OF NOSOCOMIAL INFECTIONS IN A COMMUNITY BASED HOSPITAL

CHAPTER I

INTRODUCTION

Intensive care patients are at high risk of developing nosocomial infections (NI) (Jarvis, Edwards, & Culver, 1991). The overall infection rate in critically ill patients is reported to be 40% and may be as high as 60% among patients who remain in the intensive care unit (ICU) for more than 5 days (Potgieter, Linton, Oliver, & Forder, 1987; Vincent, Bihari, & Suter, 1995). Pneumonia accounts for the largest number of these infections among ICU patients (Apostolopoulou, Bakakos, Katostaras, & Gregorakos, 2003; Hixson, Sole, & King, 1998; Ibrahim, Tracy, Hill, Fraser, & Kollef, 2001; Wallace, Cinat, Gornick, Lekawa, & Wilson, 1999), occurring predominantly in individuals requiring mechanical ventilation (MV) (Ibrahim, Ward, Sherman, & Kollef, 2000). Nosocomial bloodstream infection (NBSI) follows as one of the most common NI (Pittet, Harbarth, & Ruef, 1999; Rebollo, Bernal, Llorca, Rabasca, & Revuelta, 1996; Wallace et al.). Both pneumonia and NBSI are associated with higher mortality rates (Girou, Stephan, Novara, Safar, & Fagon, 1998; Ibrahim, Tracy et al; Jarvis, 1996; Pittet, Tarara, & Wenzel, 1994; Stone, Larson, & Kawar, 2002; Tablan et al., 1994), prolonged hospitalization (Bercault & Boulain, 2001; Digiovine, Chenoweth, Watts, & Higgins, 1999; Girou et al.; Ibrahim, Tracy et al.; Jarvis, 1996; Papia, McLellan, & El-Helou, 1999; Pittet et al.; Stone et al.; Tablan et al.), and increased costs (Digiovine et al.; Girou et al.; Grap & Munro, 1997; Harris, Majjari, Morton, & Soeken, 2000; Jarvis; Pittet et al.;

Stone et al.). Warren, Shukla, Olsen, Kollek, Hollenbeak, Cox et al. (2003) reported that the extra costs attributable to each case of nosocomial pneumonia averaged $11,887, while costs of treating NBSI averaged close to $40,000 (Jarvis, 1996; Stone et al.). Such costs impose a burden not only to the patient, but also to the health care system as a whole. Therefore, it is essential that every effort be made to identify and understand the factors that contribute to the development of such infections.

A recent study was the first to report that the presence of chest tubes is predictive of the development of NBSI. Other studies (Brunner, Vincent, Alexander, Laneve, & Fallon, 1990; LeBlanc & Tucker, 1985) reported that chest tubes were associated with increased risk of nosocomial pneumonia. However, little is known about the impact that various chest tube-related factors have on the risk for developing NI. In other words, no studies have reported on what it is about chest tubes that makes them risk factors for the development of NI. Therefore, this study aimed to investigate 1) the impact of chest tube-related factors on the development of NI, and 2) to examine the independent predictors of NI in a community based ICU. It is anticipated that findings of this study may provide guidance with respect to efforts made to minimize the risk of these infections in critically ill patients.

Problem Statement

Identifying the risk factors of NI is a first step to addressing the problem. A number of studies examined various risk factors across different types of NI (El-Masri, Hammad, McLeskey, Joshi, & Korniewicz, 2004; Harris, Joshi, Morton, & Soeken, 2000; Papia et al., 1999; Pittet et al., 1994; Walker, Kapelanski, & Weiland, 1985; Wallace et al., 1999). One risk factor for the development of NI is the insertion of invasive devices.

Much effort has been exerted on defining evidence-based guidelines geared to prevent NI related to central line insertion and MV. However, little attention has been paid to devices such as chest tubes. Although some studies have indicated that chest tubes were associated with increased risk of nosocomial pneumonia (Apostolopoulou et al., 2003; Brunner et al., 1990; LeBlanc & Tucker, 1985), El-Masri et al. were the first to report that chest tubes were independent risk factors for the development of NBSI. Despite these aforementioned reports, little is known about the chest tube-related factors that make them a risk for NI. Therefore, this study intended to examine factors such as the size, indication, number, and duration of chest tubes on the risk for the development of NI. In this study, NI was defined as pneumonia or bloodstream infection that develops more than 48 hours after hospital admission. While critically ill patients can develop other nosocomial infections, the selection of these two was based on the fact that other infections such as urinary tract and wound infections cannot be conceptually related to chest tubes.

Significance

Nosocomial pneumonia accounts for 17.8% of all NI, with the majority of cases occurring among critical care patients. Overall mortality attributed to pneumonia is estimated to be between 30% and 80%, making it the most common cause of death from NI (Craven & Regan, 1989). The estimated attributable cost associated with nosocomial pneumonia is reported to be $11,897 per patient (Warren et al., 2003).

The consequences of NBSI not only include adverse health complications and increased mortality rates, but also create significant financial costs that burden the patients and health care delivery systems (Digiovine et al., 1999; Pittet et al., 1994).

NBSI have been reported to account for an increased ICU length of stay of 2.86 days along with the associated costs of approximately $40,000 per patient (Jarvis, 1996; Stone et al., 2002).

The cost of NI to individuals and society makes it imperative that every effort be made to reduce its incidence. El-Masri et al. (2004) reported that the presence of a chest tube is a risk factor of NBSI among trauma patients. This is a significant finding because of the frequency with which chest tubes are used in the ICU. Blunt chest trauma, second only to head and spinal cord trauma, is the leading cause of death among trauma victims (Keough, 2001). Almost 25% of all trauma patients who die will have required treatment with one or more chest tubes (Keough; Sharma, Mullins, & Trunkey, 1996). However, trauma patients account for only a small portion of patients who require the insertion of one or more chest tubes during the course of their medical treatment in a community ICU (Gilbert, Mcgrath, & Soberman, 1993). Any surgical procedure in which the chest cavity is entered, whether done electively or emergently, requires a chest tube in order to facilitate re-expansion of the lung and maintain effective respiration. Critically ill patients may also require the insertion of one or more chest tubes to drain fluid collections such as pleural effusion and blood. Similarly, critically ill patients may require chest tube placement to treat a pneumothorax, which may occur spontaneously or iatrogenically. It is not clear whether the presence of a chest tube in non-trauma critically ill patients also poses a risk for the development of NBSI.

While physicians are responsible for inserting chest tubes, the ongoing care of the patient including the maintenance and monitoring of these devices falls into the domain of nursing. Therefore, nurses play a vital role in infection control activities and

monitoring for early signs of infections. Attention to infection control techniques during handling of chest tubes is of paramount importance to limiting the risk of NI. Nurses must also be vigilant about observing the patient for early signs of infection, so that it can be treated before developing into more serious systemic infections or sepsis. It is essential that critical care nurses participate in determining the factors that expose patients to increased risk for NI, so that prevention efforts can be properly implemented.

Purpose

The purposes of this study are to:

- Examine chest tube-related factors that contribute to development of NI in a community hospital
- Identify other independent predictors of NI in a community hospital setting

Conceptual Framework

Indication for chest tube insertion

An appreciation of the anatomy and physiology of the lungs helps to explain how the presence of a chest tube could be related to the development of NBSI or pneumonia. Tiny alveolar sacs are essentially wrapped with a network of pulmonary capillaries such that the basement membranes of the capillary walls often fuse with the basement membranes of the alveolar septa. This results in very little separation between blood in the capillary and gas in the alveolus. Gas exchange occurs across the alveolar-capillary membrane (Brashers, 2002).

The chest wall (skin, ribs, intercostal muscles) protects the lungs from injury, and its muscles, in conjunction with the diaphragm, perform the work of breathing. A serous

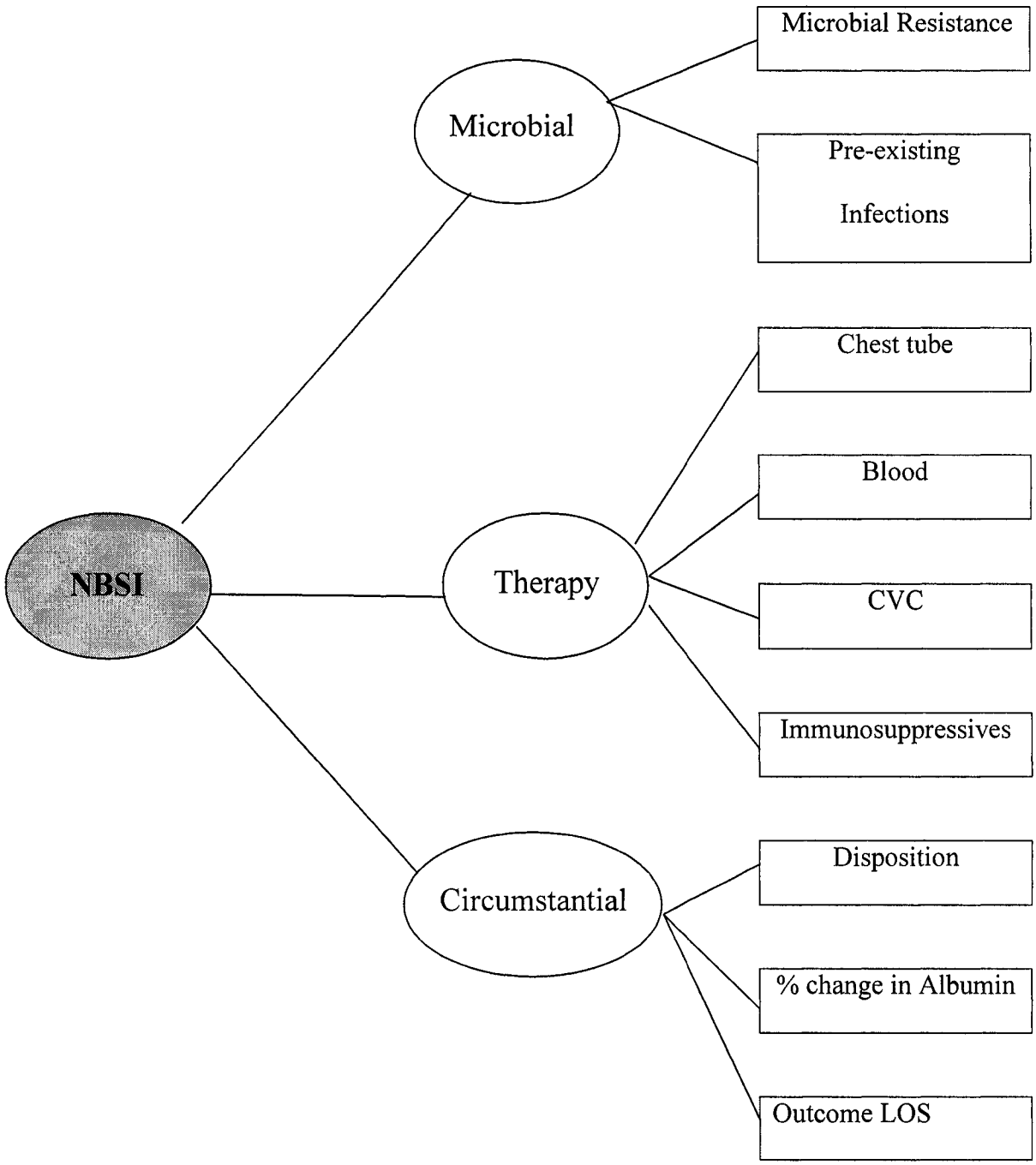
membrane called the pleura adheres firmly to the lungs (visceral pleura). It then folds over itself and attaches firmly to the chest wall (parietal pleura). The area between the two pleurae is called the pleural space. There is a thin layer of lubricating fluid between the two pleurae, allowing them to slide effortlessly over each other. Normally there is a negative pressure between the two layers, keeping them closely adhered to each other (Brashers, 2002).

In the act of breathing, muscular action expands the chest wall, causing the lungs to expand. The negative pressure that is created then draws air into the lungs. Gas exchange occurs. The relaxation of the chest wall then compresses the lungs, expelling air containing the products of respiration. Any breach in either of the pleural membranes impacts ventilation, because of the loss of negative pressure. The presence of air in the pleural space is called a pneumothorax. It is considered "open", when there is an external opening in the chest wall, and "closed" when there is no opening to the outside. An open pneumothorax can result from a stab wound, gunshot wound or surgical procedure. Closed pneumothorax may be caused by a number of factors, including rupture of alveoli related to MV, blunt chest trauma, ruptured emphysematous bullae, chest wall defects and central line insertions. As air accumulates in the pleural space, it compresses the adjacent lung tissue, resulting in its collapse. If the collapse is small, no treatment is indicated, but if the collapse is greater than 25% of the lung volume, treatment consists of insertion of a chest tube (Thelan, Davie, Urden, & Lough, 1994) that will be connected to suction by way of an underwater seal, to re-establish the intrapleural negative pressure and facilitate ventilation. The chest tube is left in place until the breach in the pleural membrane has healed itself and the lungs are re-expanded.

In a similar fashion, the accumulation of fluid in the intrapleural space, such as blood as a result of trauma or surgery, or of other fluids secondary to disease, compresses the lung and impedes ventilation. A chest tube is often required to drain the accumulated fluid and re-establish adequate lung expansion.

Only a part of the chest tube is inserted into the body and the rest remains outside, to be connected to a drainage device. Such devices could easily serve as a conduit for bacteria to enter the body and become a source of infection. Because of the close proximity of both the alveolar and capillary membranes to the intrapleural space, any infection involving the intrapleural space could easily migrate to either the lungs and or the bloodstream, and vice versa. The body may treat the chest tube as a foreign object, and initiate an inflammatory response to minimize its impact. The inflammatory response itself makes blood vessels more permeable, enhancing the possibility of infection spreading into the circulatory system.

Many factors are involved in the development of NBSI. El-Masri (2004) presented a model that depicted nine predictors of NBSI that were explained by three categories: therapy, microbial and circumstantial. The predictors were *"use of immunosuppressives, presence of microbial resistance, outcome length of stay, presence of pre-existing infections, percent change of albumin levels, patient disposition, transfusion of 10 or more units of blood, and the number of central venous catheters"* (Figure 1).



CVC = central venous catheter; LOS = length of stay

Figure 1. El-Masri's model of NBSI among critically ill trauma patients.

Another model was developed by Harris, Joshi, et al. (2000) depicting the factors that contribute to the development of nosocomial pneumonia in the trauma population (Figure 2). Harris et al. identified groups of factors known as *host related factors*, *treatment related factors*, and *infection control related factors*. While chest tubes were not specifically reported in this model, it reported the presence of thoracic surgery that would likely require the insertion of chest tube(s). Harris et al. depicts *treatment related factors* as contributing to *lung defences overwhelmed*, which directly leads to pneumonia. The model makes an interesting connection between *bacteremia* and *lung defences overwhelmed.* The relationship is identified as bi-directional, indicating that bacteremia contributes to the overwhelming of the lung defenses, and hence the development of pneumonia, and that pneumonia contributes to the development of bacteremia.

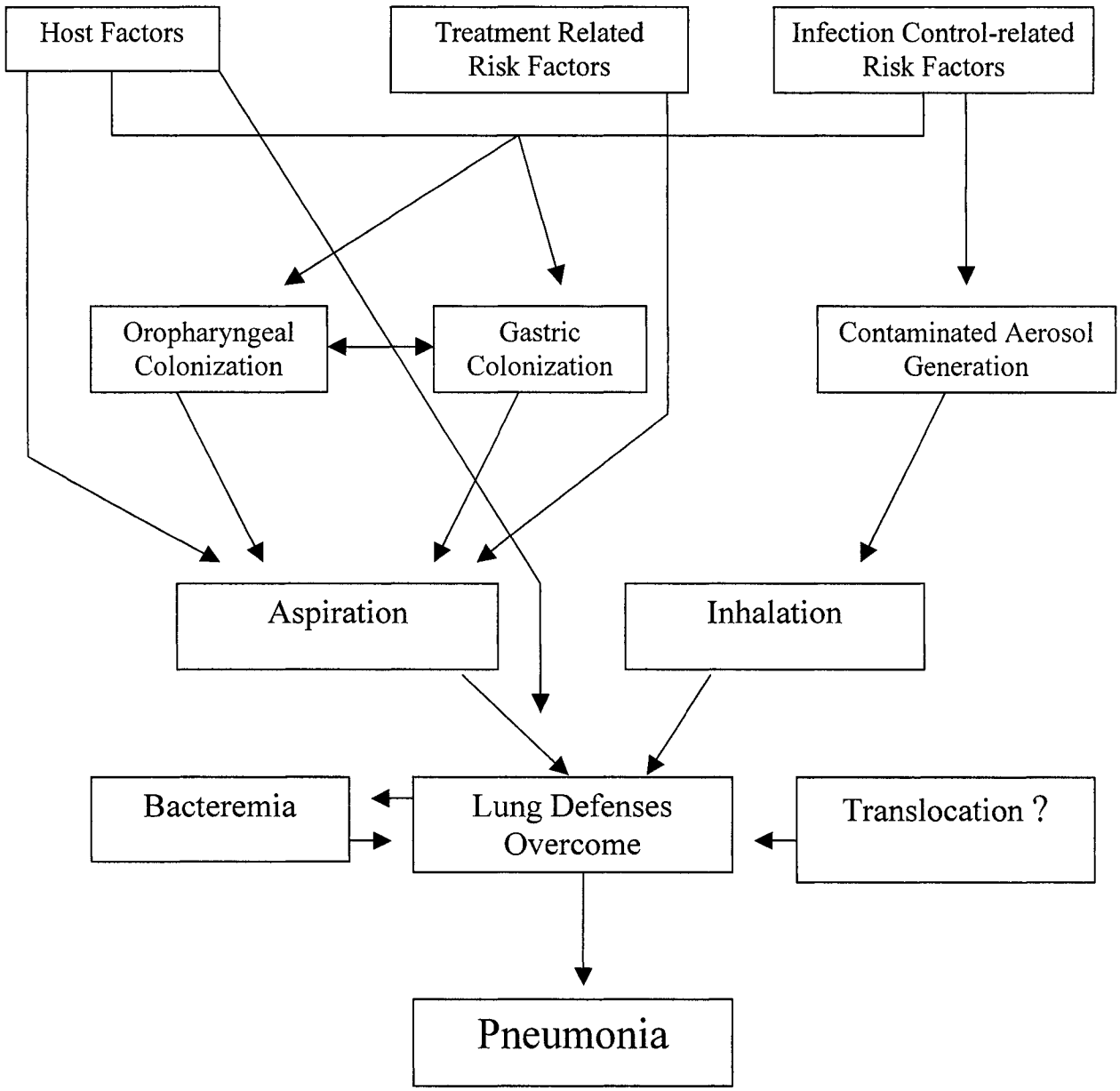


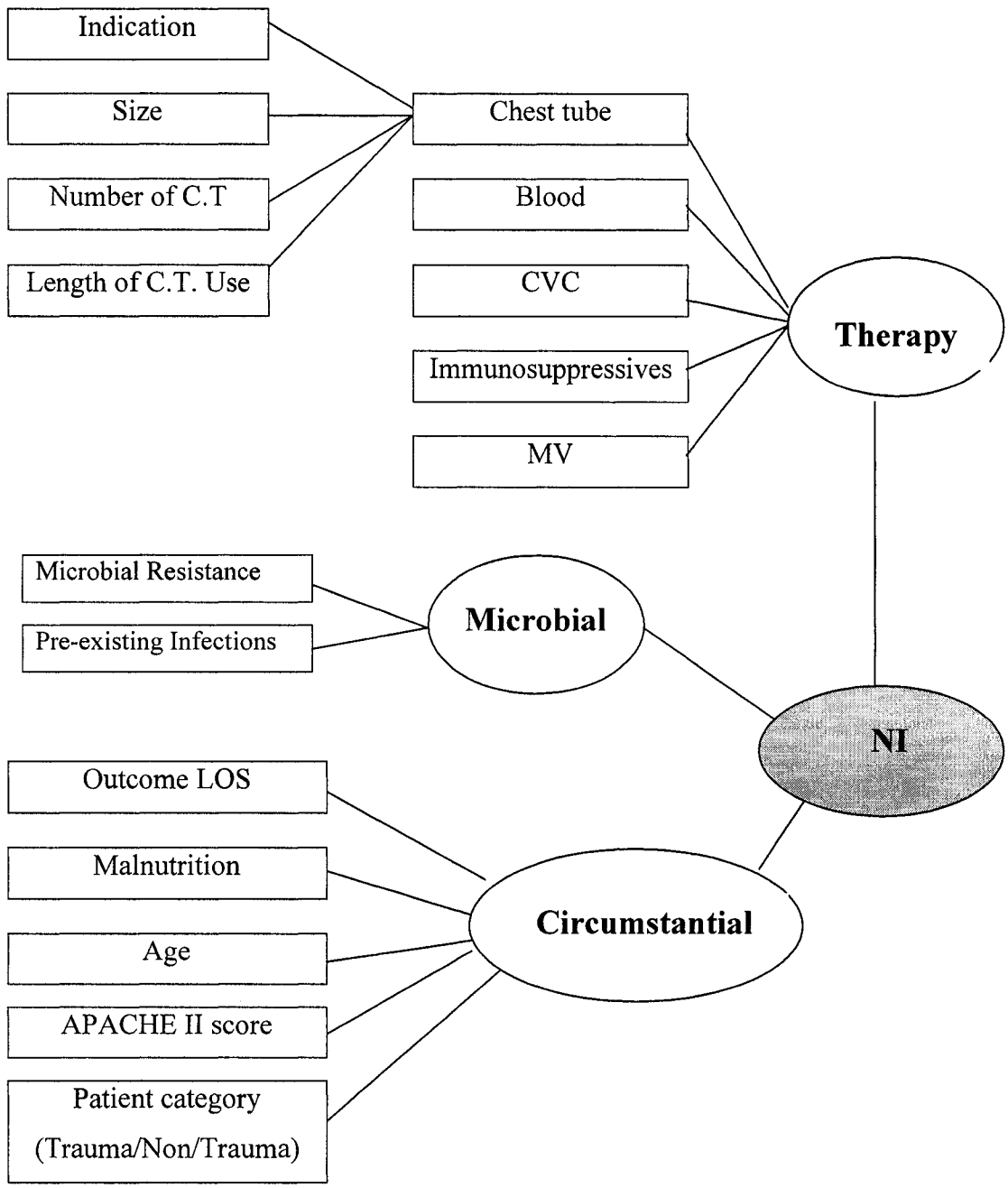
Figure 2. Harris's model of nosocomial pneumonia

The relationship between NBSI and pneumonia is well established in the literature which suggests that primary pneumonia may complicate into secondary bacteremia and vice versa (Antonelli et al., 1996; Gordon et al., 1998; Harris, Joshi et al., 2000; Scheckler, Bobula, Beamsley, & Hadden, 2003; Tablan et al., 1994; G. D. Taylor, Buchanan-Chell, Kirkland, McKenzie, & Wiens, 1994, 1995; Wallace et al., 1999). Therefore, in this study, NI will be defined as a case that has either NBSI or pneumonia.

The focus of this study was to examine the impact of chest tubes on the risk of developing NI, specifically NBSI and pneumonia. Because the models proposed by El-Masri (2003) and Harris, Joshi et al. (2000) have relatively similar groupings of factors, and because of the suggested relationship between bacteremia and pneumonia, a modified version of El-Masri's model was used as a basis to examine the impact of chest tube-related factors on the development of NI. These factors include size, duration and indication of chest tube use, and number of chest tubes used.

El-Masri's model (2004) was based on a study conducted in a sample of critically ill trauma patients, among whom age and injury severity were found not to be independent risk factors for NBSI. This may be because of the homogeneity of age and injury severity within this patient population. Most trauma patients are healthy young adults prior to their injury. Patients in a community ICU setting may tend to be diverse in age, and have comorbidities. In addition, El-Masri's model was based on a study that measured injury severity in terms of an anatomic injury severity index known as the Injury Severity Score (ISS). It is not known whether injury severity as measured by a physiological injury scale, such as the Acute Physiology and Chronic Health Evaluation

(APACHE II) score, would be associated with NBSI. Therefore, in this study, age, and APACHE II score were included in the modified model (Figure 3) and were investigated to examine whether there is a difference in using an anatomic versus a physiologic injury severity scale. *Patient disposition* was one factor that was included in El-Masri's model, but will not be included in the modified model. This factor identified whether the patient was transferred to the trauma centre where the study was being conducted, from other facilities within the area. It was not included in the current study, because patients admitted to HDGH are rarely received as transfers from other institutions. The model was also modified to include *mechanical ventilation* (MV) as a therapy associated factor, to control for the high impact that it has on the development of nosocomial pneumonia.



CT = chest tube, CVC = central venous line; MV = mechanical ventilation;
LOS = length of stay.

Figure 3. Conceptual model utilized in the current study, including additional factors, which may be of significance in a general ICU population.

Research Questions

1. What are the chest tube-related factors that contribute to the development of NI among critically ill patients in a community hospital?
2. What are the independent predictors of NI among critically ill patients in a community hospital?

CHAPTER II

REVIEW OF LITERATURE

The modified version of El-Masri's model (Figure 3) is used as a framework to organize the literature review of this study. A brief review of the infectious complications of chest tubes will be presented, along with the chest tube-related factors that may contribute to the development of NI. Literature on MV, age, APACHE II scores and patient category (trauma or non-trauma) will be reviewed in addition to the factors identified by El-Masri (2004) to examine the impact that these factors have on the risk for the development of NI among patients in a community ICU. Each of these factors will be discussed as it relates to the development of NBSI and nosocomial pneumonia. Since 90% of the cases of nosocomial pneumonia in a critical care setting are considered to be ventilator associated pneumonia (VAP), references to VAP will often be used to represent the broader diagnosis of nosocomial pneumonia.

Infectious Complications of Chest Tubes

A number of studies examined infectious complications as they relate to the presence of chest tubes (Brunner et al., 1990; Daly, Mucha, Pairolero, & Farnell, 1985; Eddy, Luna, & Copass, 1989). Eddy et al. reported that infectious complications may develop in 2% to 25% of patients who undergo thoracostomy tube placement. Complications of chest tube insertion include pneumonia (Brunner et al.) and positive bacterial cultures (Daly et al.). El-Masri et al. (2004) were the first to report on the relationship between the use of chest tubes and NBSI among critically ill trauma patients.

Pneumonia

Pneumonia was reported as a common complication associated with chest tube insertion (Brunner et al., 1990; LeBlanc & Tucker, 1985). A study of risk factors for VAP (Apostolopoulou et al., 2003) was the first to identify thoracostomy tubes as an independent risk factor for VAP. Apostolopoulou et al. suggested that interventions near the lung parenchyma might play a role in the development of VAP. While the incidence of pneumonia as a result of chest tube insertion has been reported, none of the studies explored chest tube-related factors that may have had a causative role. Helling, Gyles & Einstein (1989) concluded that pneumonia should not be considered a complication of the presence of a chest tube, because pneumonia occurred no more often in blunt injuries than it did in penetrating wounds. Other investigators examining complications related to thoracostomy tubes did not specifically survey the incidence of pneumonia, because there were multiple factors responsible for pneumonia in patients with multiple injuries (Etoch, Bar-Natan, Miller, & Richardson, 1995).

Bloodstream Infection

El-Masri et al (2004) suggested that the odds of developing NBSI among patients who had a chest tube were 4.32 times higher than the odds of patients who did not have a chest tube. This finding is of important clinical significance because of the proximity of chest tube insertion sites to the central circulation. Thus, any break in the continuity of the major blood vessels that may result from intentional or unintentional trauma could lead to migration of microorganisms from the insertion site to the central circulation. Despite the significance of this finding, El-Masri did not provide details concerning the

unique impact of chest tube-related factors on the risk of developing NI. The following highlights some of these potential factors that will be further investigated in this study.

Chest Tube-Related Factors

Indication for Chest Tube

None of the studies reviewed that were related to the development of NI differentiated between chest tubes inserted to treat pneumothoraces as opposed to those inserted to treat hemothoraces. Helling et al. (1989) examined patients whose need for a chest tube was due to blunt trauma as opposed to penetrating trauma and reported that despite longer requirement for MV and ICU stay, patients with blunt trauma were not at any higher risk of infectious complications than patients who had penetrating injuries. Blood in the pleural cavity is an excellent culture medium for bacteria (Hix, 1984), so an undrained hemothorax has the potential to develop into an empyema. Gilbert (1993) indicated that empyema occurs more frequently in trauma patients with incomplete drainage, penetrating injury or prolonged use. There is no indication however, whether empyema is related in any way to the development of either NBSI or nosocomial pneumonia.

Size of Chest Tube

There is a trend toward use of smaller bore chest tubes following cardiac surgery because of increased patient comfort. A comparison of smaller bore and traditionally-sized chest tubes (Farhat et al., 2003) reported no post-operative infections in the small bore group and a 5% incidence of wound and orifice infection in the regular-sized chest tube group. There was no report of bloodstream infection in the study. However, sample size was limited to 20 patients in each group, so further investigation may be required.

The British Thoracic Society recommends the use of small bore tubes (10-14F) in the management of pneumothoraces (Henry, Arnold, & Harvey, 2003). However, there is no evidence to suggest that either size is therapeutically superior (Farhat et al.; Laws, Nevill, & Duffy, 2003). Large bore chest tubes are recommended for management of acute hemothorax in order to achieve adequate drainage of the thoracic cavity and to assess the extent of blood loss (Laws et al.).

Number of Chest Tubes and Duration of Use

Little is reported in the literature about the impact of either the number of chest tubes inserted or the duration of use. In relation to injury caused by gunshot wounds, Fallon (1994) reported that the extent of the injury and its secondary damage may hamper complete re-expansion, predisposing to longer periods of chest tube drainage with its increased risk of infectious sequelae – either as a portal of entry or as a foreign body. Several studies (Brunner et al., 1990; Fallon, 1994; Gonzalez & Holevar, 1998) recommend continuing antibiotics as long as a chest tube is in place. These recommendations may indicate that chest tubes pose a risk for infection.

Microbial Factors

Microbial Resistance

Microbial resistance to antibiotics has been a growing problem in health care. A three-fold increase in rates of antibiotic resistant bacteremia in recent years has been reported (Castillo, Rickman, Brodine, Ledbetter, & Kelly, 2000). However, the literature has primarily focused on the trends and epidemiology of resistant pathogens more than whether microbial resistance is a risk factor for NI (Edmond et al., 1999; Gales et al., 2001; Pfaller, Jones, Doern, & Kugler, 1998; Stokes, 2002). Several studies have reported

that microbial resistance was associated with NI (El-Masri et al., 2004; Fierobe et al., 2001; Maschmeyer, Hoskin, Ribaud, & Sepkowitz, 2001; McGowan, Hall, & Parrott, 1989; Pfaller et al.; Raad et al., 1997). Other studies suggest that patients who are colonized with an antibiotic resistant microbe have a higher frequency of colonization or infection with other nosocomial pathogens than non-colonized patients (Fierobe et al.; Hsueh et al., 2002). El-Masri et al. reported that the frequency of NBSI was higher in trauma patients with microbial resistance than patients who did not have microbial resistance (*OR* of 9.25; 95% CI, 1.84-46.42). However, El-Masri et al. cautioned that the generalizability of this finding might be limited because of the small number of resistant cases in their study.

Pre-existing Infections

The presence of other infections such as pneumonia and wound infections has been reported as a potential risk for the development of secondary NBSI (El-Masri, 2003; El-Masri et al., 2004; Garner, Jarvis, Emori, Horan, & Hughes, 1988; Pittet, Li, Woolson, & Wenzel, 1997). In fact, El-Masri et al. reported that patients with a pre-existing infection were 5.46 times more likely to develop NBSI than those who did not have a pre-existing infection (95% CI, 2.28-13.06). The mechanism by which a pre-existing infection increases the risk of NBSI is not fully understood, although it is believed that microbes break into the circulation through the infection-damaged walls of blood vessels and lymphatic system by a process called bacterial translocation (Berg, 1995; Deitch & Berg, 1987). Translocation was originally described as a process by which bacteria indigenous to the gastrointestinal tract pass through the mesenteric lymphatic system to

the bloodstream. It has been suggested that a compromised immune system, as occurs in critically ill trauma patients, further facilitates the process (Berg).

Antonelli et al. (1996) reported that the risk for late onset bacteremia among trauma patients was significantly higher among patients with pneumonia. The association between presence of another NI and increased NBSI has been examined in other populations (Antonelli et al.; Gordon et al., 1998; Leibovici, Greenshtain, Cohen, Mor, & Wysenbeek, 1991; G. D. Taylor et al., 1994, 1995). A study of cardiothoracic intensive care patients (Gordon et al.) reported that a considerable percentage of patients developed NBSI as a complication to another infection. Another study (G. D. Taylor et al., 1994) reported that urinary tract infections, gastrointestinal tract infections and respiratory tract infections were important sources of NBSI (11%, 8%, and 7% respectively). Similar results were reported in a 25-year follow-up in a community teaching hospital (Scheckler et al., 2003). Taylor et al. (1995) reported that 8.4% of all NBSI cases in a tertiary care setting were pneumonia-related. Little is reported about the impact of pre-existing infections on the development of nosocomial pneumonia.

Other Therapy Related Factors

Blood Transfusion

Several authors have suggested that blood transfusion is an independent risk factor for the development of NI, particularly bloodstream infections (Agarwal, Murphy, Cayten, & Stahl, 1993; Rebollo et al., 1996; Ryan, Carthy, & Rady, 1997; R. W. Taylor et al., 2002). Studies have documented that immunosuppression as a result of blood transfusion may play a significant role in the development of NI (Bordin, Heddle, & Blajchman, 1994; van de Watering, Hermans, & Houbiers, 1998). El-Masri et al. (2004)

reported that 96.4% of NBSI patients had received blood transfusion and that those who received 10 or more units of blood were approximately 5 times more likely to acquire NBSI. Papia et al. (1999) reported that patients who acquire a NI were likely to have had multiple blood transfusions. It has been further reported that the infection rate in patients who received at least one transfusion was higher than that of patients receiving no blood transfusion (Claridge, Sawyer, Schulman, McLemore, & Young, 2002). Both Papia et al. and Claridge et al. indicated that there is a dose-dependent relationship between infection and the number of units of blood transfused. In fact, Leal-Noval et al. (2001) reported that patients who receive four or more units of blood are at higher risk for NBSI than patients who receive less than 4 units of blood.

Shorr (2004) reported that transfusion of red blood cells independently increased the risk for VAP (*OR*, 1.89; 95% CI, 1.33-2.68). He further reported that the effect was more evident for late-onset VAP, and demonstrated a positive dose-response relationship ($p = 0.02$). In a review of more than 30 factors related to the development of nosocomial pneumonia, there was no suggestion that blood transfusion plays a significant role (Harris, Joshi et al., 2000).

Central Venous Catheter

The use of a central venous catheter (CVC) is an essential part of the care of critically ill patients. Unfortunately, the use of such catheters poses a major risk for NI, particularly NBSI. Breaches in aseptic techniques during insertion, microbial colonization, poor maintenance of insertion sites, multiple accesses for medication administration, and infusion of potentially contaminated solutions contribute to the development of CVC-related infections.

The use of CVC in critical care patients has been reported to be associated with the development of NBSI (Antonelli et al., 1996; El-Masri, 2003; Papia et al., 1999). CVC catheters account for about 70% - 90% of all NBSI cases (Adal & Farr, 1996; Banerjee et al., 1991; Edgeworth, Treacher, & Eykyn, 1999; Pearson & Abrutyn, 1997). Some authors have suggested that skin colonization at the site of catheter insertion is a major factor in the development of NBSI (Moro, Vigano, & Cozzi Lepri, 1994; Safdar & Maki, 2004; Saint, Veenstra, & Lipsky, 2000; Tennenberg et al., 1997). Others suggest that multiple lumens in a CVC increase the likelihood of NBSI more than single lumen catheters (Charalambous, Swoboda, Dick, Perl, & Lipsett, 1998; Dezfulian, Lavelle, Nallamothu, Kaufman, & Saint, 2003; Hilton et al., 1988; Tokras et al., 1999). However, El-Masri found that the number of catheter lumens was not associated with increased risk for NBSI, despite reporting that the number of CVC lines used in a patient increased the risk for NBSI.

Researchers have also suggested that the duration of CVC use (Gil, Kruse, Thill-Baharozian, & Carlson, 1989; Richet, Hubert, & Nitemberg, 1990; Ullman, Gurevich, Schoch, & Cunha, 1990), the location of the CVC (Brun-Buisson, Doyon, & Carlet, 1996; Charalambous et al., 1998; Horowitz, Dworkin, Savino, Byrne, & Pecora, 1990; McKinley, MacKenzie, Finfer, Ward, & Penfold, 1999; Richet et al., 1990), and the frequency with which the CVC is changed (Cook et al., 1997; El-Masri, 2003; Manglano & Martin, 1991; Uldall, Merchant, Woods, Yarworski, & Vas, 1981) to be risk factors for NBSI. However, when El-Masri et al. (2004) included all CVC related factors in one regression model, the number of CVC was found to be the only risk factor for NBSI. This finding may suggest that other CVC related factors were essentially confounders.

Although central venous catheters appear to pose a greater risk for the development of NBSI than pneumonia, Ibrahim et al. (2001) reported that insertion of multiple central venous lines was an independent predictor of VAP (*OR* 4.20; 95% CI, 2.72 to 6.48; $p < 0.001$) occurring within the first 96 hours of MV. In another study, patients with CVC were more likely to develop VAP than patients without a CVC (Warren, 2003).

Immunosuppressives

Immunosuppression can be either pathologic such as in neutropenia (Carlisle, Gucalp, & Wiernik, 1993; van Burik & Weisdorf, 1999) and HIV (Craven, Steger, & Hirshhorn, 1996), or drug induced (Arruda et al., 1999; Engelhard et al., 1995; Horl, Schmitz, Ivens, & Grabensee, 2002). Patients with compromised immune functions often experience higher risk of NI (Charalambous et al., 1998; El-Masri et al., 2004; Maki, McCormick, Uman, & Wirtanen, 1979). While several authors (Arruda et al.; Garibaldi, 1983; Horl et al.) have reported that the use of immunosuppressive medications increases the risk of NI, no reports are available on the incidence of NBSI associated with such medications. Use of corticosteroids has been associated with an increased risk of pneumonia in critically ill trauma (Harris, Joshi et al., 2000) and nontrauma patients (Shukla, 2003). Immunosuppressive medications have also been associated with an increased risk for NI in transplant (Garibaldi; Horl et al.) and HIV patients (Petrosillo et al., 1999). Only one study (Rojo, Pinedo, Clavijo, Garcia-Rodrigues, & Garcia, 1999) reported that an immunocompromised state was not significantly related to the risk of developing NBSI in a general hospital population.

Mechanical Ventilation

The incidence of nosocomial pneumonia related to mechanical ventilation (MV) ranges from 8% to 50% (Chastre & Fagon, 2002; Dodek et al., 2004), with the risk increasing by 1-3% for each MV day (Apostolopoulou et al., 2003; George, 1995). A number of mechanisms contribute to the development of pneumonia in the mechanically ventilated patient. Many patients have nasogastric tubes that predispose them to gastric reflux and increase the potential for aspiration (Kollef, 1999). The presence of an endotracheal tube (ETT), required for MV, bypasses normal physiological defence mechanisms, making aspiration of larger quantities of bacteria from the oropharynx possible, and increasing the likelihood of infection (Harris, Joshi et al., 2000). Inhalation of contaminated aerosols also contributes to the development of VAP.

Circumstantial Factors

Malnutrition

Malnutrition occurs when there is insufficient nutritional intake to meet bodily requirements, and results in a catabolic state. It is estimated to occur in as many as 50% of ICU patients (Quirk, 2000), due to delay in delivering nutrition to critically ill patients and due to the increased caloric requirements associated with critical illness. Trauma itself results in a depletion of protein stores as a result of injured muscles. Consequently, critically ill trauma patients often experience a state of severe protein breakdown, and thus carry a high risk for malnutrition. Clinically significant malnutrition negatively affects the immune system by impacting the body's ability to synthesize the needed protein to defend against infection (Pepe & Barba, 1999). Nutritional deficiency does not

only impair the immune response, but the resultant infection can itself precipitate malnutrition. In trauma patients, the impact of malnutrition is further compounded by the immune depression that occurs as a result of the trauma itself (Bhaskaram, 2002; Catania & Chaudry, 1999; Field, Johnson, & Schley, 2002).

Several authors (El-Masri, 2003; El-Masri et al., 2004; Leibovici et al., 1991; Ryan et al., 1997) have examined the risk of NBSI associated with malnutrition as reflected by decreased serum albumin levels in various patient populations. Leibovici et al. found that low serum albumin was a strong predictor of bacteremia in febrile patients admitted to an internal medicine service. El-Masri quantified malnutrition as a percent-change in the final albumin level from baseline and reported that for each 10% decrease in the albumin levels from baseline, there was a 20% increase in the risk of NBSI. Another study (Ryan et al.) arrived at a similar conclusion, but the use of simple descriptive comparisons compromises the validity of his results. A study relating to major thoracic trauma (Walker et al., 1985) found that there was no significant association between malnutrition and general NI.

Garrard (1996) indicated that poor nutritional status is associated with a high incidence of nosocomial pneumonia. Harris, Joshi, et al. (2000) examined decreased albumin levels in relationship to the development of VAP among trauma patients, but found no significant relationship. The author did suggest that malnutrition may be predictive of pneumonia in critically ill non-trauma patients, and indicated that further research was warranted using other populations. Ibrahim et al. (2002) examined the risk for NI associated with early versus late enteral feeding of mechanically ventilated patients. One would expect that early feeding would reduce the likelihood of the

development of malnutrition and consequently result in fewer NIs. However, Ibrahim et al. reported that the administration of more aggressive early enteral nutrition is associated with greater infectious complications and prolonged lengths of stay in hospital. The author suggested that clinicians balance the potential for complications resulting from early enteral feeding with the expected benefits of such therapy.

Outcome LOS

Several studies have examined the relationship between LOS and NBSI or other infections (Bochicchio, Joshi, Bochicchio, Tracy, & Scalea, 2004; Bochicchio, Joshi, Knorr, & Scalea, 2001; El-Masri, Joshi, Hebden, & Korniewicz, 2002; Hurr, Hawley, Czachor, Markert, & McCarthy, 1999; Laupland, 2002; Laupland et al., 2004; Papia et al., 1999). All reported that longer ICU and hospital LOS were associated with the development of NI. It appears that LOS in these studies was regarded as a consequence of NI, rather than a risk factor for developing NI. Hurr reported that NI were related to LOS, but did not provide an explicit operational definition of LOS. Others (Bochicchio et al.; Laupland; Papia et al.) appear to be referring to the relationship between NI and the total LOS. It is not clear whether the development of NI is the result of a long LOS, or whether prolonged LOS is a function of NI. ICU LOS, which includes the time before and after the development of infection, can be misleading when trying to determine predictors of NI. To avoid this, Hurr suggested recording the number of days from admission to the first NI as an endpoint. This concept was used by El-Masri et al. (2004) in examining risk factors for NBSI in critically ill trauma patients. The author defined *Outcome LOS* as the number of days from admission to either the development of NBSI or discharge without NBSI, whichever occurred first. The author reported that the mean hospital LOS and ICU

LOS were significantly higher among patients with NBSI than in patients without NBSI. However, patients with NBSI had a significantly lower outcome LOS than patients who did not (*OR*, 0.89: 95%CI, 0.83-0.95), indicating that patients who developed NBSI were more likely to develop it early in their admission.

Age

A number of studies relating to critically ill patients examined the impact of age on the development of NI. Two studies (Bochicchio et al., 2001; Pories et al., 1991) reported that age was an independent predictor of NBSI and general NI in trauma patients. A study of over 27,000 trauma patients reported that complications including respiratory failure and pneumonia were more common in elderly patients (over 65 years) than in younger patients (Stawicki, Grossman, Hoey, Miller, & Reed, 2004). It further reported that older patients experienced longer ICU and hospital stay. However, El-Masri et al.(2002) reported that there was no significant association between the age of trauma patients and the development of NBSI. Similar findings were reported (Hurr et al., 1999; Offner, Moore, Biffl, Johnson, & Silliman, 2002; Papia et al., 1999) concerning unstratified infections in trauma patients.

There are also conflicting results among trauma patients. A three-year chart review of tertiary care patients (Pittet et al., 1997) suggested that age was an independent predictor of NBSI. The odds ratio of 1.014 was relatively small considering the sample size (n = 64,281), so age cannot be assumed to be a strong predictor of NBSI. Rebollo et al. (1996) reported that age was a predictor of NBSI, and indicated that the relative risk of infections for patients older than 65 years was two-times greater than that for those who were younger. Other studies (Craven et al., 1988; Emori et al., 1991; Schneider, 1983)

have indicated that older patients are more likely to develop NI than younger patients. One study reported that elderly patients experience an increased daily rate of NI, especially after 7 days of hospitalization. Results of a 15-year follow-up of bloodstream infections showed an increased proportion of NBSI among the Medicare age group, and that this age group accounted for 33-55% of the cases with bloodstream infections (Scheckler, Scheibel, & Kresge, 1991). Follow-up by the same investigator 10 years later (Scheckler et al., 2003) continued to report that the incidence of bloodstream infections among patients older than 65 years remains approximately twice the overall incidence. Similar results were reported in a study of patients with severe bloodstream infections (Laupland et al., 2004), with those 65 years of age or older having a relative risk of 7.0 (95%CI, 5.6 - 8.7) of developing BSI. Several authors (Applegren et al., 2001; Jerome et al., 2000; Pittet et al., 1999; Rojo et al., 1999) however, reported no association between age and NI.

Age has been reported to be a factor in the development of pneumonia as well. Harris et al. (2000) reported that trauma patients 40 to 55 years of age have a 2 to 3.7-fold increased risk of development of pneumonia. The author further reported that each 10-year increase in age from a base-age of 20, results in a 1.28-fold increase in the risk of pneumonia. Among non-trauma patients, age greater than 60 years was found to be independently associated with VAP (Kollef, 1993). In a study examining factors related to the early onset of VAP, patients with VAP were younger (41 ± 21 years) than patients who did not develop pneumonia (50 ± 20 years) (Akca, 2000). Thus, with varying evidence relating to the impact of age on the development of NI, it is worthwhile including age in this study.

Injury Severity

Another factor that has been examined in other studies with respect to the development of pneumonia and NBSI concerns the extent of injury sustained at the time of admission. Researchers have hypothesized that the more severely injured patients will be more prone to the development of NI. Two scores are frequently mentioned with respect to measuring acuity of patients in a critical care setting. APACHE II combines a numeric score derived from values of 12 routine physiologic measurements, age and previous health history to provide a general measure of severity of disease (Knaus, Draper, Wagner, & Zimmerman, 1985). Injury Severity Score (ISS) used with trauma patients provides a measure of the overall condition of the patient based on the number of body parts that have been impacted by trauma. It is calculated by summing the squares of the highest three Abbreviated Injury Severity scores obtained over six regions of the body. The scores range from 0 to 75, with 0 indicating no injury and 75 indicating a non-survivable injury. In a study of the effectiveness of APACHE II and ISS scores as predictors of NI in trauma patients, Hurr et al. (1999) reported that these scores had no value in predicting NI. However, studies (Bohnen, Mustard, Oxholm, & Schouten, 1988; Chevret, Hemmer, & Claret, 1993; Fernandez-Crehuet et al., 1997) showed a predictive value for the APACHE II in determining the occurrence of NI. Increasing APACHE II scores are reported to be associated with the development of nosocomial pneumonia, particularly early-onset nosocomial pneumonia (Ibrahim et al, 2000, Warren, 2003). While the APACHE II score was not used as the measuring scale, Rello (2002) reported that underlying illness severity is one of three independent risk factors for development of VAP.

In a 25-year follow-up study, severity of underlying illness was reported to be one of three risk factors associated with the acquisition of NBSI in a community hospital (Scheckler et al., 2003). Another study of patients who developed NBSI following cardiac bypass surgery (Ryan et al., 1997) reported that patients with preoperative comorbidities had a higher incidence of NBSI. A review of patients with gram-negative rod (GNR) infections among patients admitted to general, transplant and trauma surgery services (Raymond et al., 2003) revealed that antibiotic-resistant GNR infections were associated with increased APACHE II scores as well as multiple comorbidities. The same patients were also more likely to have pneumonia and catheter infections, coexistent infection with antibiotic-resistant gram-positive cocci and fungi and high mortality by both univariate and logistic regression analysis.

Patient Category (Trauma versus non-trauma patients)

Trauma patients are reported to be at increased risk for NI because of unclean penetrating injuries, trauma-related depression of humoral and cell-mediated responses (El-Masri et al., 2004; Meert, Long, Kaplan, & Sarnaik, 1995; O'Mahony, Palcer, & Wood, 1984; Stillwell & Caplan, 1989), massive blood loss and blood transfusion (Agarwal et al., 1993; El-Masri et al.; Papia et al., 1999; Ryan et al., 1997), inadequate nutritional support (Bochicchio et al., 2001; El-Masri et al.; Papia et al.; Pittet et al., 1994) and the extensive use of invasive devices. A study examining the differences in NI in trauma and surgical patients (Wallace et al., 1999) reported that the overall infection rate among trauma patients was 11.64 % compared with 6.43% for surgical patients ($p <$ .001). Trauma patients were reported to have higher rates of VAP (6.13% vs 2.50%; $p <$ 0.001 and NBSI (2.52% vs 1.27%; $p <$0.01).

CHAPTER III

METHOD

Design

A retrospective, case-control chart review was conducted to compare chest tube-related factors among patients who had chest tubes and developed NI, and patients who had chest tubes but did not develop NI. The data was collected using the medical records of patients who were admitted to the ICU of Hotel Dieu Grace Hospital (HDGH). The ICU at HDGH is a 20 bed medical surgical ICU that provides care to patients with a broad range of critical trauma and non-trauma related conditions. Considering the limited number of patients who had chest tubes in this ICU population, traditional matching procedures of cases and controls were not followed. Instead, all patients with chest tubes who were admitted to the ICU over the last 3 years (from January 2002 to April 2005) were identified. These patients were assigned to one of two groups, either positive for NI (cases group) or negative for NI (control group). To provide statistical control for the proposed risk factors of NI, data was collected on each of the variables identified in the study's model. Data was also collected on other factors such as indication for chest tube, number of chest tubes, length of use, size of chest tube, age, APACHE II score, and patient category (trauma vs. non-trauma).

Protection of Human Subjects

Ethical approval (Appendix A) was obtained from both the University of Windsor and HDGH prior to data collection. Based on the fact that the study is very low risk, involving no patient contact, and requiring information gathered from their medical

records, a waiver of patient consent was obtained. Each patient was identified by a study code that was used in the data collection and data analysis phases. The log linking the patient's identity to the study code has been maintained in a locked cabinet in the investigator's office. Only the investigator had access to this information. Completed data collection sheets were stored in a locked cabinet in the investigator's office at HDGH. Completed data sheets were entered into a computerized data file that was assigned a secure password. To prevent disclosure of patients' identities, data collection sheets and the computerized data entries are identified only by their assigned codes

Inclusion/Exclusion Criteria

Inclusion

Patients whose charts were examined were:

1. 18 years or older
2. Had one or more chest tube(s) during their ICU stay
3. Required admission to the Intensive Care Unit.

Exclusion

Patients were excluded if an infection was diagnosed within the first 48 hours of admission to HDGH.

Data Collection Procedures

A data collection sheet (Appendix B) was developed for the purpose of this study. The Health Records Department was asked to identify the charts of all patients who were in the ICU and who had one or more chest tubes during their ICU stay for the period extending from January 2002 to April 2005. The researcher reviewed the medical records pertaining to the patients' hospitalizations in the computerized chart repository, and

recorded the required information on the data collection sheets. Records of the entire ICU stay were reviewed.

Variable Definitions

NI was defined in a patient if he/she had either BSI or pneumonia manifesting 48 hours or more after admission to ICU. NBSI is defined as having a clinically positive blood culture for bacteria and/or fungus that is obtained more than 48 hours after hospital admission (Garner et al, 1988). If the isolated organism is a common skin contaminant (coagulase-negative staphylococci, Bacillus species, diptheroids, viridans streptococci or micrococcus species), one or more clinical signs of NBSI such as fever, chills or hypotension must be present. Pneumonia is defined by the criteria proposed by the Centers for Disease Control and Prevention as the presence of new, persistent pulmonary infiltrates not otherwise explained, appearing on chest radiographs. In addition, at least two of the following criteria are required: (a) temperature > 38^0 C; (b) leukocytosis > 10,000 cells per mm^3; and (c) purulent respiratory secretions (Houston, 2002).

The size of each chest tube was recorded as either large bore (26 French or larger), medium bore (size 14 – 24 French) or small bore (size 12 French or smaller). Number of chest tubes was defined as the total number of chest tubes inserted from admission to the ICU until the last chest tube was removed. Length of use was measured as the number of days during which a chest tube was in place. Dose relationship is a derived factor, called number of chest tube days, determined by summing the number of days that each chest tube was in place.

Microbial resistance was defined as the presence of methicillin-resistant Staphyloccus aureus or vancomycin-resistant enterococci documented by culture. Pre-

existing infection refers to any infection that was present or developing at the time of admission, and was defined as any confirmed infection prior to the development of NBSI or pneumonia.

Outcome LOS was measured by recording the number of days from the date that the first chest tube was inserted to either the development of a NI or removal of the last chest tube without having developed NI, whichever occurred first. Data relating to the total number of packed red blood cells (PRBC) and total number of CVCs were recorded during the period from admission to the endpoint. Transfusion of PRBC and the use of one or more CVC was recorded as a dichotomous variable and coded as (yes = 1 and no = 0). Total number of units of PRBC transfused and the total number of CVCs were recorded as continuous variables. Use of an immunosuppressive agent was recorded as a dichotomous variable and coded as (yes = 1 and no = 0). Use of MV was recorded as (yes = 1 and no = 0), and the number of days during which the patient was connected to a ventilator was recorded as MV days. Albumin level was recorded as percentage change between final and baseline values.

The age of the patient in years on the date of admission was recorded. The APACHE II score was based on information in the patient's chart relating to the first 24-hour period during the ICU stay, as calculated electronically by the data collection nurse in the ICU. Patients were identified as trauma or non-trauma based on the reason for their hospital admissions.

Data Analysis Procedures

Data was analyzed using SPSS 13.0 statistical software. Data was screened for appropriateness of data entry, outliers, assumption of normality for continuous variables,

collinearity and missing data. Data was treated according to the procedures outlined in Tabachnick and Fidell (2001). Basic descriptive statistics including general frequencies of discrete and categorical variables, as well as means and standard errors of continuous variables are reported.

The research questions were addressed as follows:

Research Question 1. What are the chest tube-related factors that contribute to the development of NI among critically ill patients in a community hospital?

Bivariate *t*-test and chi square comparisons were performed to examine unadjusted differences between the two groups to identify the crude comparisons between those who developed NI and those who did not. Then, each variable identified as having a significant relationship with NI ($p \leq .25$) in the univariate analysis, was included in a multivariate logistic regression analysis to identify which chest tube-related factors were independent predictors of NI, and to estimate the odds ratio associated with each. The use of a liberal *p* value was chosen to avoid unnecessary deletion of potentially significant independent predictors from the final multivariate regression model. To examine the unique impact of C.T. related factors, a 2-step hierarchical regression approach was used. In step 1, all variables, except C.T. related variables were entered into the model. In step 2, C.T.-related variables were entered to examine their impact on NI, above and beyond that explained by other variables. A 95% confidence interval that does not include 1 was used as the criterion to establish significance.

Research Question 2. What are the independent predictors of NI among critically ill patients in a community hospital?

This question could be answered as part of answering research question #1.

CHAPTER IV

RESULTS

Data Cleaning Procedures

Missing Data

Upon completion of data collection procedures, the computerized database was proof read against the original data. Physicians did not consistently order measurement of serum albumin level, so the variable *albumin level* (both initial and final) was missing in more than 20% of the sample and was therefore deleted from the analysis. The variable *chest tube size* was not recorded in 17.5 % (n = 21) of the charts. Rather than eliminating the variable, an additional category, *size not specified,* was added to the other size categories. The remaining variables had no missing data.

Univariate Outliers and Normality

Discrete variables. General frequencies were performed on all dichotomous and categorical variables in the sample and no outliers that could have resulted from inappropriate data labels were found. Table 1 however, shows that the dichotomous variables *microbial resistance, pre-existing infection* and *use of immunosuppressive medication* had their categories split at greater than 90-10 and their cases had a quasi-complete separation when cross-tabulated with the outcome variable (NI). Therefore, these variables were considered outliers and were deleted from any further analysis because their regression coefficients would not be stable and scores in the smaller categories of variables would be more influential than those of the larger categories (Hosmer & Lemeshow, 2000; Tabachnick & Fidell, 1996).

Table 1

Dichotomous Variables with Category Split of Less than 90-10

Variable		NI			
		No	Yes	Total	Split %
Microbial Resistance	No	79	38	117	97.5%
	Yes	1	2	3	2.5%
Pre-existing Infection	No	78	39	117	97.5%
	Yes	2	1	3	2.5%
Use of immunosuppressives	No	72	36	108	90.0%
	Yes	8	4	16	10.0%

Continuous variables. Since logistic regression analysis involves grouped data, the search for univariate outliers was sought separately within each group (cases and controls) using z score statistics, histograms and box plots. The variables *APACHE II score, outcome LOS,* and *number of chest tube days* had 1 to 5 outlier cases as indicated by a *z* score value >3.29 for those cases. While statistical transformation of the data was an option to treat the extreme scores of outliers, another option was to bring the value of the outlier cases to match the next acceptable extreme score in their distributions (Kline, 1998; Tabachnick & Fidell, 1996). The latter technique was used to treat *APACHE II* and *outcome LOS* to avoid potential difficulties that would be encountered during the interpretation of the odds ratios of transformed data.

The variable *chest tube days* was not normally distributed and was therefore recoded into a categorical variable by transforming the data into categories based on the 10-day intervals as seen in Table 2.

Table 2

Treatment of Skewness for Variables Which were not Normally Distributed

Variable	Pre-treatment Skewness / SE	Treatment
Chest tube days	12.90	Changed to categorical data, grouped as follows; 1-10 days 11-20 days > 20 days

Singularity

Singularity causes data redundancy that may lead to artificial inflation of regression coefficients. It results when the multivariate analysis includes a continuous variable that is a composite of two or more constituent variables (Kline, 1998; Tabachnick & Fidell, 1996). In this study, no variables were derived from or were constituents of other variables, and therefore singularity was not an issue.

Sample Characteristics

The mean age of patients was 60.26 years (range 19 to 87 years), with men comprising 73.3% (n = 76). Surgical patients comprised 76.7% (n = 92) of the sample. Trauma patients accounted for 15.0% (n = 18) of the patients in the sample. The mean APACHE II score for the first 24 hours of admission was 13.7 (range 3 to 32). Eighty-nine (74.2%) patients had a chest tube inserted during the performance of a surgical procedure. The remaining chest tubes were inserted for treatment of pneumothorax (n = 11; 9.2%), pleural effusion (n = 9; 7.5%), pneumohemothorax (n = 7; 5.8%) and hemothorax (n = 4; 3.3%). Generally, a large sized chest tube was inserted as 72.5% (n = 87) of the patients had tubes sized 26 or greater. Patients had a mean number of chest tube days of 18.8 (range 1 to 120 days). The majority of patients required a CVC (n = 89; 74.2%), while13.3% (n = 16) required transfusion of at least one unit of blood. Those who had blood transfusions (n = 16) received a mean of 6.8 units. 39.2% (n = 47) were mechanically ventilated, for an overall mean of 1.88 days (range 0 to 22 days). Those who required MV (n = 47) were intubated for a mean of 4.81 days. Of the 40 cases with NI, 7.5% (n = 3) had BSI, while 92.5% (n = 37) had pneumonia. The mean outcome LOS was 7.7 days (range 1 to 59 days).

Univariate Analysis

Univariate analysis is organized according to the level of variable measurement (categorical vs. continuous). Tables 3 and 4 provide *t*-test statistics and chi square comparisons between patients who developed NI and those who did not on each of the study variables.

Table 3 suggests that the two groups differed with respect to *APACHE II* score, *number of CVC* catheters used, number of days spent on a ventilator, and *outcome LOS.* The results suggest that infected patients had higher *APACHE II* scores (15.3 versus 12.7; $p = .023$), required more CVC catheters (1.18 versus .79; $p = .010$) and spent more time on a mechanical ventilator (4.5 days versus 6 days; $p < .001$) than those who did not have NI. The infected group also had a shorter outcome LOS (4.9 days versus 9.1 days; $p < .001$), which indicates that those who developed infection did so early during admission. While the infected group tended to be older, the difference in *age* was not statistically different (63.8 years versus 58.5; $p = .083$). The two groups did not differ in terms of the number of *units of blood* transfused.

Table 3

T-test Comparisons of Continuous Variables for Patients With and Without NI

Variable	Infected (M ± SE) n = 40	Not infected (M ± SE) n = 80	*t*	*p*
Age in years	63.80 ± 2.348	58.49 ± 1.798	-1.749	.083
Apache II	15.28 ± 6.465	12.65 ± 5.585	-2.301	.023
Units of blood	1.23 ± .415	.66 ± .218	-1.323	.188
Number of CVC	1.18 ± .147	.79 ± .075	-2.614	.010
Ventilator days	4.50 ± .851	.57 ± .156	-6.146	<.001
Outcome LOS	4.88 ± .443	9.09 ± .824	3.486	.001

CVC = central venous line; LOS = length of stay

Table 4 shows that patients with NI were not significantly different from patients without NI in terms of the type of medical problem (medical versus surgical) (p = .222) and the need for *blood transfusion* (p = .117). There was no significant difference between the groups in terms of various chest tube-related factors such as the *indication for chest tube use* (p = .192), *chest tube size* (p = .249), *chest tube location* (p = .105), and *chest tube days* (p = .211). The two groups did differ however, in terms of *gender* (p = .007), the use of *CVC catheters* (p = .018), and the need for *MV* (p = <.001)). The difference between patients with NI and those without is approaching significance in *patient category* (trauma/non-trauma) (p = .055).

Table 4

Chi Square Comparisons of Categorical Variables for Patients With NI and Patients Without NI

Variable	Infected n (%) n = 40	Not infected n (%) n = 80	χ^2	*p*
Gender			7.177	.007
Male	32 (80%)	44 (55%)		
Female	8 (20%)	36 (45%)		
Classification			1.491	.222
Medical	12 (30%)	16 (20%)		
Surgical	28 (70%)	64 (80%)		
Patient category			4.706	.055
Trauma	10 (25%)	8 (10%)		
Non-trauma	30 (75%)	72 (90%)		
Received blood transfusion			2.455	.117
No	28 (70%)	66 (82.5%)		
Yes	12 (30%)	14 (17.5%)		

(table continues)

Table 4. (continued)

Variable	Infected n (%) (n = 40)	Not infected n (%) (n= 80)	χ^2	*p*
Required CVC			5.567	.018
No	5 (12.5%)	26 (32.5%)		
Yes	35 (87.5%)	54 (67.5%)		
Mechanically ventilated			16.806	<.001
No	14 (35%)	59 (73.8%)		
Yes	26 (65%)	21 (25.2%)		
Reason for chest tube use			6.103	.192
Pneumothorax	5 (12.5%)	6 (7.5%)		
Hemothorax	1 (2.5)	3 ((3.8%)		
Post-op planned	26 (65.0)	63 (78.8%)		
Pleural effusion	3 (7.5)	6 (7.5%)		
Pneumohemothorax	5 (12.5%)	2 (2.5%)		
Chest tube size			5.394	.249
Small	0 (0)	4 (5.0%		
Medium	1 (2.5%)	1 (1.3%)		
Large	26 (65.0%)	61 (76.3%)		
More than 1 size	3 (7.5%)	3 (3.8%)		
Size not specified	10 (25.0%)	11 (13.8%)		

CVC = central venous line

(table continues)

Table 4. (continued)

Variable	Infected n (%) n = 40	Not infected n (%) n = 80	χ^2	p
Chest tube location			4.502	.105
Right	17 (42.5%)	43 (53.8%)		
Left	14 (35.0%)	30 (37.5%)		
Bilateral	9 (27.5%)	7 (8.8%)		
Chest tube days			3.111	.211
1-10 days	14 (35.0%)	28 (35.0%)		
11-20 days	10 (25.0%)	31 (38.8%)		
> 20 days	16 (40.0%)	21 (26.3%)		

Multivariate Analysis

As explained in the methods section, a liberal alpha of ≤ .25 was selected as the cut-off point for the inclusion of a variable in the multivariate model. All variables met this criterion and were considered for the regression model. Prior to the analysis, the data were checked for multivariate outliers and multicollinearity.

Multicollinearity results from a high correlation between two or more of the independent variables. It affects the stability of the regression coefficients and causes inflation of the error terms (Nunnally & Bernstein, 1994; Tabachnick & Fidell, 1996). Although several approaches could be used to assess for multicollinearity (Tabachnick &

Fidell), a correlation of ($\geq$ 0.7) was used as a cut-off point to suggest multicollinearity in this study. Several pairs of variables were examined for possible collinearity. Table 5 depicts the correlations between the suspected collinear pairs along with the decisions that were made regarding which variable was to be included in the regression model. The pair of variables relating to blood transfusion was multicollinear and so the decision was made to use the categorical variable *blood transfusion.* This variable is dichotomous, and can be included in a multivariate regression analysis without concern about its distribution, and because its odds ratio is easy to interpret. The other two pairs of variables (*CVC used and number of CVC, MV and number of MV days*) did not have a correlation of $\geq$ .7, but their correlations were considerably high (Table 5). Therefore, the decision was made to use only the categorical variables of these two pairs to avoid possibility of data redundancy. This decision was made because these pairs of variables essentially represented different methods of measuring the same variable.

Table 5

Decision Regarding Inclusion of Variables in Multivariate Regression Analysis Resulting from Examination for Collinearity

Variables	Correlation		Decision
	Pearson's *r*	*p.* (2 tailed)	
Blood transfusion	.737	<.001	Use categorical variable
Number of units of blood			
CVC used	.693	<.001	Use categorical variable
Number of CVC			
Mechanically ventilated	.625	<.001	Use categorical variable
Number of MV days			

CVC = central venous line; MV = mechanical ventilation

Logistic regression analysis can be performed using independent variables that are either continuous or dichotomous. Categorical variables measured at more than two levels must be recoded in order to be included in the regression model. Thus dummy codes were created for categorical variables that had more than two categories. Table 6 illustrates how *size of chest tube* was dummy coded for regression analysis. The variables *chest tube days, indication for chest tube use* and *chest tube location* were similarly dummy coded, but are not displayed in the table.

Table 6.

Dummy coding of Chest Tube Size

Original codes		Dummy codes	
Label	Code	Label	Code
	Each code is a category of Chest tube size		Each code is a separate dichotomous variable
Small	1	Reference	
Medium	2	CT Medium	1 = medium; 0 = else
Large	3	CT Large	1 = large; 0 = else
Mixed	4	CT Mixed	1 = mixed; 0 = else
Not specified	5	CT Notspec	1 = not specified; 0 = else

CT = *Chest tube*

Logistic Regression Analysis

Table 7 displays the results of the two-step hierarchical modeling approach that was conducted to examine the impact of chest tube-related factors, while adjusting for other factors. A total of 24 variables that had significant associations with NI at an alpha of $\leq .25$ were considered for the regression modeling process. In step one of the hierarchical regression analysis, all variables, except for chest tube-related variables, were allowed to enter the regression model in a stepwise approach. A stepwise approach, which identifies independent predictors of NI, was selected to avoid inflation of regression coefficients and the explained variance that could result from the inclusion of non-significant variables in the model. In step two, all chest tube-related variables were

allowed to enter the model using a standard regression approach (variables were forced into the model). This selection of a standard regression approach was chosen to elicit specific information with regard to the unique impact of each chest tube-related variable, regardless of its level of significance. The Hosmer and Lemeshow goodness of fit statistic for the first step of the regression model was insignificant ($\chi^2 = 6.166$; $p = .629$) indicating that the model has good fit of the data. The Cox and Snell R^2 for the first step was 0.307 and the Nagelkerke R^2 was 0.427, indicating that the four variables (*age, gender, MV and, outcome LOS)* could explain between 30.7% and 42.7% of the total variance in NI. In the second step, the Hosmer and Lemeshow goodness of fit statistic remained insignificant ($\chi^2 = 9.158$; $p = .329$). However, at this step, *gender* was no longer significant. The inclusion of chest tube factors increased the Cox and Snell R^2 to .381 and the Nagelkerke R^2 to .529. These results suggest that chest tube-related variables explained between 7.4% and 10.2% of the final variance. However, of all chest tube-related variables, only *chest tube days* was significant.

Table 7

First Logistic Regression Model

Variable	Step 1 (Stepwise)				Step 2 (Standard)			
	B	*SE*	*OR*	*p*	*B*	*SE*	*OR*	*p*
Age	.036	.016	1.037	.025	.040	.021	1.040	.066
Gender	-1.059	.535	.347	.048	-.730	.609	.482	.230
MV	1.645	.483	5.183	.001	1.140	.609	3.127	.061
Outcome LOS	-.258	.070	.773	<.001	-.393	.097	.675	< .000
Chest tube days								
1 - 10							(Reference = 1)	
11-20					.988	.777	2.686	.203
>20					2.659	1.035	14.277	.010

(table continues)

Table 7 (continued)

Variable	Step 1 (Stepwise)				Step 2 (Standard)			
	B	*SE*	*OR*	*p*	*B*	*SE*	*OR*	*p*
Indication								
Post-op planned							(Reference = 1)	
Pneumothorax					.885	1.025	2.422	.388
Hemothorax					-2.66	1.276	.767	.835
Pleural effusion					1.302	1.269	3.676	.305
Pneumohemothorax					1.832	1.238	6.249	.139
Chest tube location								
Left sided							(Reference = 1)	
Right sided					-.197	.570	.821	.729
Bilateral					-.024	1.130	.977	.983

(table continues)

Table 7 (continued)

	Step 1 (Stepwise)				Step 2 (Standard)			
Variable	*B*	*SE*	*OR*	*p*	*B*	*SE*	*OR*	*p*
Chest tube size								
Small							(Reference = 1)	
Medium					.506	1.611	1.659	.753
More than 1 sized					-.128	1.804	.880	.943
Size not specified					.416	.828	1.515	.616

B = unstandardized regression coefficient; *SE* = standard error; *OR* = odds ratio

Step 1 Cox and Snell R^2 = .307 ; Nagelkerke R^2 = .427

Step 2 Cox and Snell R^2 = .381; Nagelkerke R^2 = .529

Table 7 suggests that only two variables were independent predictors of the development of NI: *outcome LOS* (*OR*, .675; p <.001) and *chest tube days* > 20 (*OR*, 14.277; p = .010). However, it is important to mention that prior to adjusting for chest tube-related factors in step 1, age (*OR*, 1.037; p = .025), gender (*OR*, .347; p = .048) and MV (*OR*, 5.183; p = .001) were all suggested to be independent predictors of NI. In step 2, all variables from step 1 and CT-related factors were allowed in the regression model. The results suggested that of all chest tube-related factors, only *chest tube days* was an independent predictor.

The aforementioned analysis was re-performed, conducting both steps of the hierarchical regression model in a stepwise fashion so that only significant variables were included in the final model. This approach was done in order to determine variance in NI that is unique to the significant variables. Table 8 shows that results of Step 1 were unchanged from those reported in Table 7. The Hosmer and Lemeshow goodness of fit statistic for the regression model at step 1 was insignificant (χ^2 = 7.326; p = .502), indicating that the model has a good fit with the data. Step 2 of the regression model suggests that of all chest tube-related factors, only *chest tube days* was a significant predictor of NI. Overall, the analysis suggests that only three variables were independent predictors of NI: *MV, outcome LOS, and chest tube day.*

The data suggest that mechanically ventilated patients have a higher risk of developing NI (*OR*, 4.88; 95% *CI*, 1.8-13.1). The results show that patients with NI have significantly lesser *outcome LOS* than patients who did not develop NI. The data also show that the odds of developing NI are 5.79 times higher among patients who had more than 20 chest tube days. Patients who had chest tubes for 11 – 20 days were not included

in the final model, indicating that they were not significantly different from those who had chest tubes for 1 – 10 days. Thus, the final reference for chest tube days was considered to be 1 – 20 days.

Using this method of regression analysis, the change in the explained variance was less than was observed in the first regression analysis as a result of including only the one significant chest tube factor, *chest tube days >20*. The difference in the Cox and Snell R^2 and the Nagelkerke R^2 between Steps one and two suggest that *chest tube days > 20* explains between 4.1% and 5.6% of the variance in NI in this sample.

Table 9 presents a classification index of the observed and predicted values of the final regression model (presented in Table 8) based on a cut-off probability criterion of 0.5. The data suggests that the specificity of the model (i.e. the proportion of patients who did not have NI and were correctly classified as such by the model: TN/[TN+FP]) was 92.5%. The sensitivity of the model (i.e. patients who had NI and were correctly identified as such by the model; TP/[TP+FN]) was 65.0%. The positive predictive value (PPV) of the model (i.e., if a patient is predicted to have NI, the probability that this patient truly has NI; TP/[TP+FP]) was 81.3%. The negative predictive value (NPV) of the model (i.e., if a patient is predicted by the model to be non-NI patient, the probability that this patient truly does not have NI; NT/[TN+FN]) was 84.1%. The overall precision of the model (i.e., the model's ability to accurately classify a patient as either NI or non-NI) was 83.3%. Thus, the model not only has a good fit of the data, but is also a reliable and valid model.

Table 8

Final Stepwise Logistic Regression Model

Variable	Step 1 (Stepwise)				Step 2 (Stepwise)			
	B	*SE*	*OR*	*p*	*B*	*SE*	*OR*	*p*
Age	.036	.016	1.037	.025	.025	.021	1.026	.129
Gender	-1.059	.535	.347	.048	-.926	.538	.386	.091
MV	1.645	.483	5.183	.001	1.585	.504	4.879	.002
Outcome LOS	-.258	.070	.773	<.00	-.323	.076	.724	<.00
Chest tube days								
1 – 20 days							(Reference = 1)	
> 20					1.757	.704	5.795	.013

B = unstandardized regression coefficient; *SE* = standard error; *OR* = odds ratio

Step 1 Cox and Snell R^2 = .307 ; Nagelkerke R^2 = .427

Step 2 Cox and Snell R^2 = .348; Nagelkerke R^2 = .483

Table 9

Classification Index of Observed and Predicted Values of the final Regression Model

		Predicted		Percentage correct
		No	Yes	
Observed	No	74 (TN)	6 (FP)	92.5
	Yes	14 (FN)	26 (TP)	65.0

The cut-off probability criterion of 0.5; TN = true negative, FP = false positive, FN = false negative, TP = true positive

Sub-analysis

It was initially planned to stratify the data based on the type of NI (BSI vs. pneumonia) to examine the unique impact of chest tube-related factors on each of these infections. However, such an analysis was meaningless because only three of the 40 patients with NI had BSI and therefore no comparisons were made

Because this study was based on a model derived from El-Masri's (2004) model, tested in a critically ill trauma population, it was of interest to examine whether trauma patients in the current study population differed from the non-trauma patients. The results of this analysis suggested no significant difference between the two groups in terms of NI, although trauma patients tended to develop NI more than non-trauma patients (55.6% versus 29.4%; $\chi^2 = 4.706$; $p = .055$).

A significantly higher proportion of trauma patients required *MV* than non-trauma patients (72.2% versus 33.3%; $\chi^2 = 9711$; $p = .003$).. Trauma patients had a significantly shorter *outcome LOS* (5.3 versus 7.8; $t = 1.995$; $p = .048$). Trauma patients were also more likely to have *chest tube days >20* than non-trauma patients (75.0% versus 25.0%; $\chi^2 = 11.684$; $p = .011$).

CHAPTER V

DISCUSSION

The incidence of NI among patients in ICU has been reported to be among the highest of all hospital departments (Jarvis et al., 1991). The cost of treating NI and the subsequent health complications associated with these infections makes it necessary that serious efforts be directed to their prevention. Identification of risk factors of NI is a first step in this prevention process. One major contributing factor to the high rate of NI among ICU patients is the extensive use of invasive devices, such as central venous lines, MV and chest tubes. While substantial work has focused on examining the impact of central lines (Adal & Farr, 1996; Antonelli et al., 1996; Banerjee et al., 1991; Edgeworth et al., 1999; El-Masri, 2003; Papia et al., 1999; Pearson & Abrutyn, 1997; Pittet et al., 1999) and MV (Apostolopoulou et al., 2003; Chastre & Fagon, 2002; George, 1995; Harris, Joshi et al., 2000; Kollef, 1999) on the development of NI, little attention has been paid to chest tubes. Although some studies have indicated that chest tubes were associated with increased risk of nosocomial pneumonia (Apostolopoulou et al., 2003; Brunner et al., 1990; Helling, Gyles, & Einstein, 1989; LeBlanc & Tucker, 1985), El-Masri et al. (2004) were the first to report that the presence of a chest tube was an independent risk factor for the development of NBSI. Unfortunately, none of the aforementioned studies examined specific chest tube-related factors that contribute to making chest tubes a risk factor for the development of NI. Therefore, the focus of this study was to examine chest tube-related factors to determine their impact on the development of NI. A secondary focus of the study was to examine predictors of NI in a community ICU setting.

The findings of this study suggested that NI in the ICU has three independent predictors: *MV, outcome LOS, and number of chest tube days*. According to "Cox and Snell" and "Negerleke" tests, these three predictors explained between 34.8% and 48.3% of the total NI variance. The data further suggested that the regression model has a specificity of 92.5%, a sensitivity of 65%, a positive predictive value of 81.3%, a negative predictive value of 84.1%, and an overall precision of 83.3%. These classification indices indicate that the resulting regression model has a clinically acceptable ability to screen patients for risk of NI. While the sensitivity of the predictive model was relatively low (65%), the positive predictive value (81.3%) was large enough to be clinically useful as a predictive tool. This is because the positive predictive value indicates that if a patient was predicted to be at risk for developing NI, then it is 81.3% likely that this patient will truly develop NI. In other words, unlike sensitivity, the positive predictive value enables clinicians to calculate the risk of infection prior to its development.

Despite the fact that only three variables were found to be independent predictors of NI, many others were considered for their potential contribution to NI. The following discussion focuses on specific findings pertaining to the study variables. Recommendations, implications for nursing, and study limitations are also discussed.

Chest Tube-Related Factors

This study was the first to examine several chest tube-related factors, such as indication, size, location and number of chest tube use days. The unadjusted univariate analysis suggested that none of these factors was associated with NI. However, the adjusted regression analysis suggested that of all chest tube-related variables that were

studied, only *number of chest tube days* was an independent predictor of NI (*OR*, 5.795; 95% CI; 1.459 – 23.015). This variable alone added from 4.1% to 5.6% to the total explained variance of NI.

In this study the variable *chest tube days* was derived from summing the number of days that each chest tube was present in a patient. Univariate analysis of the data did not show a significant difference between patients with NI and those without NI in terms of *chest tube days* (*t*, -1.836; $p = .069$). However, results of the adjusted analysis suggested that patients who had chest tubes for more than 20 days were almost 6 times more likely to develop NI than those who had chest tubes for 20 days or less (*OR*, 5.795; 95% CI; 1.459 – 23.015). This finding highlights the importance of statistical adjustment in predictive studies.

This study was the first to examine the impact of chest tube days on the risk for developing NI. Although there is no evidence to support a causal relationship with regard to the impact of chest tube days on the risk for the development of NI, the finding of this study is consistent with the conceptual proposition of Fallon (1994) who suggested that the extent of injury related to gunshot wounds predisposes patients to longer periods of chest tube drainage, subsequently increasing their risk of infectious sequelae – either as a portal of entry or as a foreign body. Other studies (Brunner et al., 1990; Fallon, 1994; Gonzalez & Holevar, 1998) recommend continuing prophylactic antibiotics as long as a chest tube is in place, an implicit indication that prolonged use of chest tubes presents a potential risk for NI. .

Although *chest tube days* was the only significant variable of all chest tube-related factors, other chest tube-related factors may carry clinical significance regardless

of their lack of statistical significance. Thus, these factors need to be further considered in future research. For instance, patients who had a chest tube inserted for *Pneumohemothorax* were 6.249 times more likely to develop NI than other patients (p = .139). Given the large odds ratio for this association, a potential significant association between the indication for chest tube use and increased risk of NI in future research would not be surprising. In fact, Hix (1984) reported that blood in the pleural cavity is an excellent medium for bacterial growth, which seems to suggest that chest tubes for treatment of pneumohemothorax may carry an increased risk of NI.

Interestingly, all seven patients who had a pneumohemothorax were trauma patients. This small number of patients may explain the lack of significant association. However, it is difficult to draw definite conclusions with regard to the impact of pneumohemothorax on the risk of NI based on such a small number of patients. Nonetheless, the relatively large odds ratio associated with this relationship necessitates that this relationship be further investigated in future studies. In addition, it is prudent that nurses and other health care providers be particularly vigilant and use appropriate infection control techniques when caring of this subgroup of patients.

Similarly, patients requiring a chest tube for treatment of pleural effusion were at 3.676 times the risk of NI (p = .305). All nine of the patients who had pleural effusions were non-trauma patients. No reports were found in the literature linking pleural effusion to NI. Therefore, this finding warrants further investigation.

Mechanical Ventilation

The study findings indicate that the use of MV was an independent risk factor that increased the risk of developing NI by almost 5 times (*OR*, 4.879; 95% CI, 1.816-13.110) as compared to the risk among patients without MV. This finding was not a surprising one in light of the fact that MV has frequently been associated with the development of nosocomial pneumonia for a long time, to the point that nosocomial pneumonia among mechanically ventilated patients is known as VAP. Various studies reported that the incidence of VAP ranges from 8% to 50% (Chastre & Fagon, 2002; Dodek et al., 2004), with the risk increasing by 1% to 3% for each MV day (Apostolopoulou et al., 2003; George, 1995). In this study, 67.7% of patients who developed pneumonia were mechanically ventilated and 53.3% of the patients who were mechanically ventilated developed pneumonia. However, given the case-control nature of this study, an actual rate that represents the true incidence could not be calculated.

The results of this study differ from those of El-Masri et al. (2004), who examined the impact of MV on NBSI and reported that it was not an independent predictor of bacteremia. It is essential to consider this lack of agreement in light of the fact that majority of patients in this study had pneumonia, while those of El-Masri had NBSI. Given that only 3 patients in the current sample had NBSI, the lack of association is not surprising. It is interesting to note that two of the three patients who had NBSI were also mechanically ventilated. However, it is difficult to make an inference based on the small number of NBSI cases in this study. Thus, it is important that the impact of MV on NBSI be further investigated in future research.

Outcome LOS

While many studies have reported that longer LOS was associated with the development of NI (Bochicchio, Joshi, Bochicchio, Tracy, & Scalea, 2004; Bochicchio et al., 2001; El-Masri et al., 2002; Hurr et al., 1999; Laupland, 2002; Laupland et al., 2004; Papia et al., 1999), it appears the LOS has been regarded as a consequence of NI, rather than a risk factor. It is not clear whether the development of NI is the result of a long LOS, or whether the long LOS is a function of the NI. Hurr et al. suggested recording the number of days from admission to the first NI as an endpoint when examining predictors of NI. El-Masri et al. were the first to use this approach in examining risk factors for nosocomial bloodstream infections in critically ill trauma patients. They reported that patients who developed NBSI had a significantly lower *outcome LOS* than patients who did not develop infection (*OR*, 0.89; 95% CI, 0.83-0.95). Findings of the current study support those reported by El-Masri et al., showing that patients who develop NI have a shorter *outcome LOS* than those who do not develop NI (*OR*, 0.72; 95% CI,.624-.839). This finding does not infer that patients who develop NI have shorter lengths of stay in either the ICU or the hospital. Rather, it suggests that patients who develop NI tend to do so during the early phases of their ICU stay.

APACHE II Score

The results of this study suggest that infected patients had a higher *APACHE II* score (15.3 versus 12.7; $p = .023$) in the univariate analysis. However, in the adjusted regression model, APACHE II was not an independent predictor of NI (*OR*, 1.040; 95% CI: .997 - 1.085). Hurr et al. (1999) also found that while the APACHE II score was

related to the development of NI among trauma patients, it was not found to be an independent predictor of NI. However, this finding did not support those of other studies that reported an independent relationship between APACHE II score and NI (Bohnen et al., 1988; Chevret et al., 1993; Fernandez-Crehuet et al., 1997; Ibrahim et al., 2001; Warren et al., 2003). The finding was also inconsistent with that of Scheckler et al. (2003) who reported an independent association between APACHE II and NBSI in a community hospital. The reason for the inconsistency between our finding and the aforementioned studies is not clear. However, it could be attributed to several potentially valid factors such as design issues, number and type of variables considered, patient populations, and type of NI investigated. For instance, unlike the aforementioned studies, this study accounted for the use chest tubes, which was reported to be a surrogate marker of injury severity (El-Masri, Hammad, & Fox-Wasylyshyn, 2005).

Age

Results of this study showed that while the infected group tended to be older, the difference in age was not statistically different (63.8 years versus 58.5; p - .083). Age was identified in the first step of the initial logistic regression model as being one of the four non-chest tube-related factors that impacts NI (OR, 1.037; 95% CI; 1.005 to 1.070). However, when the chest tube-related factors were forced into the model, age was no longer a significant factor. The clinical significance of these findings though, suggest that older patients should be identified as being at higher risk for NI not merely because of their age. Rather, the findings suggest that age is a confounding variable to other variables that should be accounted for in predictive studies. This is especially important for variables that can be targeted for intervention and manipulation while age is not.

Several authors have reported an association between age and NBSI among trauma patients (Bochicchio et al., 2001; Stawicki, Grossman, Hoey, Miller, & Reed, 2004) and non-trauma patients (Pittet et al., 1997; Rebollo et al., 1996). Similarly, age has been reported to be predictive of development of pneumonia in both the trauma (Harris, Joshi et al., 2000) and non-trauma groups (Akca et al., 2000; Kollef, 1993). Other researchers reported no significant association between age and development of NBSI (El-Masri et al., 2002) and other infections among trauma patients (Hurr et al., 1999; Offner et al., 2002; Papia et al., 1999). These contradictory findings with regard to the impact of age on the risk for the development of NI could be attributed to the fact that age may be confounded with several other risk factors. That is, findings with regard to age tend to depend largely on the number and type of variables included in the regression model. Nonetheless, it is important that extra vigilance with infection control practices be exercised when caring for older patients.

Category of Admission (Trauma versus Non-trauma)

The results of this study suggested no significant difference between trauma and non-trauma patients in terms of NI, although trauma patients tended to develop NI more than non-trauma patients (55.6% versus 29.4%; χ^2 4.706; p = .055) Given that trauma patients comprised only 15% (n = 18) of our sample, it is difficult to draw definite conclusions from such a small sub-sample. However, trauma patients were more likely to have *chest tube days >20* than non-trauma patients (75.0% versus 25.0%; χ^2 11.684; p = .011). In addition, a significantly higher proportion of trauma patients required *MV* than non-trauma patients (72.2% versus 33.3%; χ^2 9.711; p = .003). These findings suggest that trauma patients had higher use of invasive devices, which may explain the trend of

increased NI among this group of patients. This assumption appears to be consistent with the findings of several authors who reported that trauma patients are at higher risk for NI and pneumonia related to unclean penetrating injuries and suppressed immune responses (El-Masri et al., 2004; Meert et al., 1995; O'Mahony et al., 1984; Stillwell & Caplan, 1989), massive blood loss and blood transfusion (Agarwal et al., 1993; El-Masri et al.; Papia et al., 1999; Ryan et al., 1997), inadequate nutritional support (Bochicchio et al., 2001; El-Masri et al.; Papia et al.; Pittet et al., 1994) and extensive use of invasive devices (El-Masri, 2003; Ibrahim et al., 2001; Papia et al.).

The variable *category of admission* was not found to be an independent predictor of NI in the current study. This may be because of the small proportion of patients who were admitted for trauma injuries as opposed to non-trauma illnesses. This finding may also be attributed to adjustment of factors that were more common among trauma patients such as MV and prolonged use of chest tubes. There is sufficient supporting evidence from other studies however to suggest that further research would be beneficial to verify whether such a factor is significant in a community hospital setting.

Limitations

Data on several variables that were included in the conceptual model were not available, which may compromise the control for potentially confounding variables. The lack of data on albumin levels among a large portion of the subjects within our sample made it impossible to examine the impact of malnutrition on the risk of developing NI. Three other variables (*microbial resistance*, *pre-existing infection* and *use of immunosuppressive medications)* were not included in the adjusted data analysis because they had a split in their categories that was greater than 90-10 with regard to the outcome variable (NI). Consequently, the impact of these variables on the development of NI was not explained. Because of the case-control nature of this study, it was not possible to draw conclusions regarding NI rates. This may be a topic of interest in a future study.

Implication and Recommendations

The findings of this study add to the body of knowledge with regard to identifying the independent predictors of NI. It is the first study to examine the impact of chest tube-related factors on the risk for development of NI. The finding that the chest tube related factors account for between 7.4% and 10.2% of the NI variance carries a meaningful clinical significance with regard to prevention strategies. Although some of the variables investigated in this study were found not to be independent predictors of NI, they still possess some clinical importance, and thus should be further investigated in future research.

Recommendations for Practice

Given that MV was a major risk factor in this study, critical care nurses must make every effort to prevent VAP. Evidence-based practice guidelines, such as those developed by the Canadian Critical Care Trials Group (AACN, 2004; Dodek et al., 2004) must be implemented for every ventilated patient. Nursing educators and administrators responsible for intensive care units should make special efforts to ensure that appropriate measures are employed to inform bedside nurses of these guidelines and to ensure that they are implemented. Such measures may include ensuring that VAP prevention guidelines are incorporated into all standardized care plans, and that documentation in patients' records reflects implementation of these guidelines. It may be helpful to inform ICU staff members of the current VAP rate in their units as compared to the rates in other benchmark hospitals. Displaying posters with this information could be a constant reminder of the need to reduce VAP incidence rate and the need to adhere to prevention guidelines.

A second recommendation is that enhanced infection control practices be implemented in conjunction with the insertion of chest tubes and in the initial care of patients with chest tubes. While most chest tubes in a community hospital are placed in the operating room under ideal circumstances, many are inserted in the emergency department or in the ICU. Staff members in these departments who assist with chest tube insertion receive few guidelines with regard to the procedure and to the necessary infection control techniques to be followed. Thus, more attention and training need to be given to help these nurses better adhere to the necessary infection control strategies. Ensuring that all the required equipment is readily available is key to preventing

infections associated with chest tube placement, particularly in departments where it is an infrequent procedure. Assembling a pre-packed chest drain insertion set containing all the basic items needed for safe insertion of a chest tube (Hyde, Sykes, & Graham, 1997) and including a visual procedure guide may help prevent NI. It may also be beneficial to use visual aids such as videotapes to demonstrate ideal chest tube insertion techniques. In addition, videotaping chest tube insertions can be helpful in allowing staff to observe their own techniques to assist them to assess their own breaches of aseptic techniques. These observations could be used as the basis for ongoing and reflective teaching that would be particularly beneficial in helping nurses refine their skills with respect to assisting in chest tube insertion and caring of patients with chest tubes.

A third practice recommendation would be to ensure that critical care nurses are aware that patients with several chest tubes are at higher risk for NI than other patients, in that they are more likely to reach *chest tube days > 20* than patients with only one chest tube. This group of patients requires that even more attention be paid to nursing measures to prevent pneumonia. The usual nursing measures employed to prevent pneumonia in patients with thoracic surgery or injury must not be overlooked. Adequate pain control must be provided to enable the patient to perform hourly deep breathing and coughing exercises, to participate with physiotherapy, and to ambulate early and often.

In addition, critical care nurses caring for patients with chest tubes must be aware of factors that may contribute to development of NI. Careful hand washing before handling chest tube equipment is essential. Use of proper dressing techniques to reduce the number of times that the chest tube insertion site is exposed may help in reducing NI. Movement of the chest tube at the insertion site can irritate the wound and be a source of

bacterial entry to the site. Careful and proper taping of the chest tube to the chest wall can minimize this risk.

Recommendation for Teaching

Several teaching strategies can raise awareness of nurses and nursing students with regard to increased risk of infections associated with chest tubes. Education should include information about the factors that have been demonstrated by the study findings as being independent predictors of NI. Clinical experiences during orientation should reflect the added infection control practices that are developed by the organization to help guard against NI in patients with chest tubes. Schools of nursing may develop infection control modules that focus on the prevention of NI. Students may be taught about risk factors for NI and be expected to demonstrate adherence with appropriate infection control measures that are directed toward the prevention of NI during patient care.

Recommendations for Research

Since this study was the first to examine the impact of chest tube-related factors on the development of NI in a community ICU, the findings of this study require replication before they can be generalized. It would be particularly useful if the study were repeated while accounting for variables that could not be investigated in this study, such as nutritional status and microbial resistance.

It was interesting to note that patients who had a chest tube inserted for pneumohemothorax were more than 6 times as likely to develop NI, and those who were treated for pleural effusion were at nearly 4 times the risk. Although neither finding was statistically significant, further research concerning these relationships is warranted. Another area that warrants further investigation is the use of injury severity scores to

predict NI. While this study did not find that the APACHE II score to be predictive of NI, further research may be warranted.

Conclusion

NI continues to be a major concern among health care providers. Recognizing the risk factors is key to preventing its development. The purpose of this study was to examine chest tube-related factors that contribute to development of NI in a community hospital, and to identify other independent predictors of NI in such a setting. Of the chest tube-related factors examined in this study, only *chest tube days* was found to be associated with NI. Findings of this study showed that of all the other non-chest tube-related factors examined, only two are independently predictive of NI in a community ICU: *MV* (*OR*, 4.879, 95% CI, 1.816-13.11) and *outcome LOS* (*OR*, .724, 95% CI, .624-.839). Given the non-experimental nature of this study, one cannot conclude that the presence of a chest tube and MV play causative roles in the development of NI. However, the use of these invasive treatment devices can serve to identify patients who are at high risk for the development of hospital-acquired infection, highlighting the need for vigilance with infection control practices.

References

AACN. (2004). *Ventilator Associated Pneumonia.* Retrieved September 5, 2005, from http://www.aacn.org/AACN/practiceAlert.nsf/Files/VAPi/$file/VAP.pdf

Adal, K. A., & Farr, B. M. (1996). Central venous catheter-related infections: A review. *Nutritional Review, 12*(3), 208-213.

Agarwal, N., Murphy, J. G., Cayten, C. G., & Stahl, W. M. (1993). Blood transfusion increases the risk of infection after trauma. *Archives of Surgery, 128*, 171-177.

Akca, O., Koltka, K., Uzel, S., Cakar, N., Pembeci, K., Sayan, M. A., et al. (2000). Risk factors for early-onset, ventilator-associated pneumonia in critical care patients: Selected multiresistant versus nonresistant bacteria. *Anesthesiology, 93*(3), 638-645.

Antonelli, M., Moro, M. L., D'Errico, R. R., Conti, G., Bufi, M., & Gasparetto, A. (1996). Early and late onset bacteremia have different risk factors in trauma patients. *Intensive Care Medicine, 22*, 735-741.

Apostolopoulou, E., Bakakos, P., Katostaras, T., & Gregorakos, L. (2003). Incidence and risk factors for ventilator-associated pneumonia in 4 multidisciplinary intensive care units in Athens, Greece. *Respiratory Care, 48*(7), 681-688.

Applegren, P., Hellstrom, I., Weitzberg, E., Soderlund, V., Bindslev, L., & Ransjo, U. (2001). Risk factors for nosocomial intensive care infection: A long-term prospective analysis. *Acta Anaesthesiologica Scandinavica, 45*(6), 710-719.

Arruda, E. A., Marinho, I. S., Boulos, M., Sinto, S. I., Caiaffa, H. H., Mendes, C. M., et al. (1999). Nosocomial infections caused by multiresistant Pseudomonas aeruginosa. *Infection Control and Hospital Epidemiology, 10*(9), 620-623.

Banerjee, S. N., Emori, T. G., Culver, D. H., Gaynes, R. P., Jarvis, W. R., Horan, T. C., et al. (1991). Secular trends in nosocomial primary bloodstream infections in the United States, 1980-1989. National Nosocomial Infections Surveillance System. *American Journal of Medicine, 91*(3B), 86S-89S.

Bercault, N., & Boulain, T. (2001). Mortality rate attributable to ventilator-associated nosocomial pneumonia in an adult intensive care unit: A prospective case-control study. *Critical Care Medicine, 29*(12), 2303-2309.

Berg, R. D. (1995). Bacterial translocation from the gastrointestinal tract. *Trends in Microbiology, 3*(4), 149-154.

Bhaskaram, P. (2002). Micronutrient malnutrition, infection, and immunity: An overview. *Nutritional Review, 60*(5 Pt 2), S40-45.

Bochicchio, G. V., Joshi, M., Bochicchio, K., Tracy, K., & Scalea, T. M. (2004a). A time-dependent analysis of intensive care unit pneumonia in trauma patients. *Journal of Trauma: Injury, Infection and Critical Care, 56*(2), February.

Bochicchio, G. V., Joshi, M., Bochicchio, K., Tracy, K., & Scalea, T. M. (2004b). A time-dependent analysis of intensive care unit pneumonia in trauma patients. *Journal of Trauma, 56*(2), 296-303.

Bochicchio, G. V., Joshi, M., Knorr, K. M., & Scalea, T. M. (2001). Impact of nosocomial infections in trauma: Does age make a difference? *The Journal of Trauma: Injury, Infection and Critical Care, 50*(4), 612-619.

Bohnen, J. M. A., Mustard, R. A., Oxholm, S. E., & Schouten, B. D. (1988). APACHE II score and abdominal sepsis: A prospective study. *Archives of Surgery, 123*, 225-229.

Bordin, J. O., Heddle, N. M., & Blajchman, M. A. (1994). Biologic effects of leukocytes present in transfused cellular blood products. *Blood, 84*, 1703-1721.

Brashers, V. L. (2002). Structure and function of the pulmonary system. In K. L. McCance & S. E. Huether (Eds.), *Pathophysiology: The Biologic Basis for Disease in Adults and Children* (4th ed., pp. 1082-1104). St. Louis: Mosby, Inc.

Brun-Buisson, C., Doyon, F., & Carlet, J. (1996). bacteremia and severe sepsis in adults: a multicenter prospective survey in ICUS and wards of 24 hospitals. french Bacteremia-Sepsis Study Group. *American Journal of Respiratory and Critical Care Medicine, 154*(3 Pt 1), 617-624.

Brunner, R. G., Vincent, G. O., Alexander, R. H., Laneve, L., & Fallon, W. F. (1990). The role of antibiotic therapy in the prevention of empyema in patients with an isolated chest injury (ISS 9-10): A prospective study. *The Journal of Trauma: Injury, Infection and Critical Care, 30*(9), 1148-1154.

Carlisle, P. S., Gucalp, R., & Wiernik, P. H. (1993). Nosocomial infections in neutropenic cancer patients. *Infection Control and Hospital Epidemiology, 14*(6), 320-324.

Castillo, E. M., Rickman, L. S., Brodine, S. K., Ledbetter, E. K., & Kelly, C. (2000). Streptococcus pneumoniae: Bacteremia in an era of penicillin resistance. *American Journal of Infection Control, 28*(3), 239-243.

Catania, R. A., & Chaudry, I. H. (1999). Immunological consequences of trauma and shock. *Annals of the Academy of Medicine of Singapore, 28*(1), 120-132.

Charalambous, C., Swoboda, S. M., Dick, J., Perl, T., & Lipsett, P. A. (1998). Risk factors and clinical impact of central line infections in the surgical intensive care unit. *Archives of Surgery, 133*(11), 1241-1246.

Chastre, J., & Fagon, J. Y. (2002). Ventilator-associated pneumonia. *American Journal of Respiratory & Critical Care Medicine, 165*(7), 867-903.

Chevret, S., Hemmer, M., & Claret, J. (1993). Incidence and risk factors of pneumonia acquired in intensive care units: Results from a multicenter prospective study on 996 patients. *Intensive Care Medicine, 19*, 256-264.

Claridge, J. A., Sawyer, R. G., Schulman, A. M., McLemore, E. C., & Young, J. S. (2002). Blood transfusions correlate with infections in trauma patients in a dose-dependent manner. *The American Surgeon, 68*, 566-572.

Cook, D., Randolph, A., Kernerman, P., Cupido, C., King, D., Soukup, C., et al. (1997). Central venous catheter replacement strategies: A systematic review of the literature. *Critical Care Medicine, 25*(8), 1417-1424.

Craven, D. E., Kunches, L. M., Lichtenberg, D. A., Kollisch, N. R., Barry, M. A., Heeren, T. C., et al. (1988). Nosocomial infection and fatality in medical and surgical intensive care unit patients. *Archives of Internal Medicine, 148*(5), 1161-1168.

Craven, D. E., & Regan, A. M. (1989). Nosocomial pneumonia in the ICU patient. *Critical Care Nursing Quarterly, 11*(4), 28.

Craven, D. E., Steger, K. A., & Hirshhorn, L. R. (1996). Nosocomial colonization and infection in persons infected with human immunodeficiency virus. *Infection Control and Hospital Epidemiology, 17*(5), 304-318.

Daly, R. C., Mucha, P., Pairolero, P. C., & Farnell, M. B. (1985). The risk of percutaneous chest tube thoracostomy for blunt thoracic trauma. *Annals of Emergency Medicine, 14*(9), 865-870.

Deitch, E. A., & Berg, R. D. (1987). Endotoxin but not malnutrition promotes bacterqail translocation of the gut flora in burned mice. *Journal of Trauma: Injury, Infection and Critical Care, 27*(2), 161-166.

Dezfulian, C., Lavelle, J., Nallamothu, B. K., Kaufman, S. R., & Saint, S. (2003). Rates of infection for single-lumen versus multilumen central venous catheters: a meta-analysis. *Critical Care Medicine, 31*(9), 2385-2390.

Digiovine, B., Chenoweth, C., Watts, C., & Higgins, M. (1999). The attributable mortality and costs of primary nosocomial bloodstream infections in the intensive care unit. *American Journal of Respirology and Critical Care Medicine, 160*, 976-981.

Dodek, P., Keenan, S., Cook, D., Heyland, D., Jacka, M., Hand, L., et al. (2004). Evidence-based clinical practice guideline for the prevention of Ventilator-Associated Pneumonia. *Annals of Internal Medicine, 141*(4), 305-313.

Eddy, A. C., Luna, G. K., & Copass, M. (1989). Empyema thoracis in patients undergoing emergent closed tube thoracostomy for thoracic trauma. *American Journal of Surgery, 157*, 494-497.

Edgeworth, J. D., Treacher, D. F., & Eykyn, S. J. (1999). A 25-year study of nosocomial bacteremia in an adult intensive care unit. *Critical Care Medicine, 27*, 1421-1428.

Edmond, M. B., Wallace, S. E., McClish, D. K., Pfaller, M. A., Jones, R. N., & Wenzel, R. P. (1999). Nosocomial bloodstream infections in United States hospitals: a three-year analysis. *Clinical Infectious Diseases, 29*, 239-244.

El-Masri, M. M. (2003). *Predictors of nosocomial blood stream infections in the adult critically ill trauma population: A model testing approach.* University of Maryland, Baltimore.

El-Masri, M. M., Hammad, T. A., & Fox-Wasylyshyn, S. M. (2005). Predicting nosocomial bloodstream infections using surrogate markers of injury severity: Clinical and methodological perspectives. *Nursing Research, 54*(4), 273-279.

El-Masri, M. M., Hammad, T. A., McLeskey, S. W., Joshi, M., & Korniewicz, D. M. (2004). Predictors of nosocomial bloodstream infections among the critically ill adult trauma population. *Infection Control and Hospital Epidemiology, 25*(8).

El-Masri, M. M., Joshi, M., Hebden, J., & Korniewicz, D. M. (2002). Use of the injury severity score to predict nosocomial bloodstream infections among critically ill trauma patients. *AACN Clinical Issues, 13*, 367-372.

Emori, T. G., Banerjee, S. N., Culver, D. H., Gaynes, R. P., Horan, T. C., Edwards, J. R., et al. (1991). Nosocomial infections in elderly patients in the United States, 1986-1990. *American Journal of Medicine, 91*(3B), 289S-293S.

Engelhard, D., Elishoov, H., Or, R., Naparstek, E., Nagler, A., Strauss, N., et al. (1995). Cytosine arabinoside as a major risk factor for Streptococcus viridans septiciemia following bone marrow transplantation: A 5-year prospective study. *Bone Marrow Transplant, 16*(4), 565-570.

Etoch, S. W., Bar-Natan, M. F., Miller, F. B., & Richardson, J. D. (1995). Tube thoracostomy: Factors related to complications. *Archives of Surgery, 130*(5), 521-526.

Fallon, W. F. (1994). Post-traumatic empyema. *Journal of the American College of Surgeons, 179*, 483-492.

Farhat, F., Ginon, I., Lefebre, M., Lu, Z., Andre-Fouet, X., Mikaeloff, P., et al. (2003). Prospective randomized comparison between redon catheters and chest tubes in drainage after cardiac surgery. *Journal of Cardiovascular Surgery, 44*(2), 179-186.

Fernandez-Crehuet, R., Diaz-Molina, C., DeIrala, J., Martinez-Concha, D., Salcedo-Leal, I., & Masa-Calles, J. (1997). Nosocomial infection in an intensive care unit: Identification of risk factors. *Infection Control and Hospital Epidemiology, 18*, 825-830.

Field, C. J., Johnson, I. R., & Schley, P. D. (2002). Nutrients and their role in host resistance to infection. *Journal of Leukocyte Biology, 71*(1), 16-32.

Fierobe, L., Lucet, J., Decre, D., Muller-Serieys, C., Deleuze, A., Joly-Guillou, M., et al. (2001). An outbreak of imipenem-resistant Acinetobacter baumannii in critically ill surgical patients. *Infection Control and Hospital Epidemiology, 22*(1), 35-40.

Gales, A. C., Jones, R. N., Forward, K. R., Linares, J., Sader, H. S., & Verhoef, J. (2001). Emerging importance of multidrug-resistant Acinetobacter species and Stenotrophomonas maltophilia as pathogens in seriously ill patients: Geographic patterns, epidemiological features, and trends in the SENTRY antimicrobial

surveillance program (1997-1999). *Clinical Infectious Diseases, 32 Supplement 2*, S104-113.

Garibaldi, R. A. (1983). Infections in organ transplant recipients. *Infection Control and Hospital Epidemiology, 4*(6), 460-464.

Garner, J. S., Jarvis, W. R., Emori, T. G., Horan, T. C., & Hughes, J. M. (1988). CDC definitions for nosocomial infections. *American Journal of Infection Control, 16*, 128-140.

George, D. L. (1995). Epidemiology of nosocomial pneumonia in intensive care unit patients. *Clinics in Chest Medicine, 16*(1), 29-44.

Gil, R. T., Kruse, J. A., Thill-Baharozian, M. C., & Carlson, R. W. (1989). Risks of human immunodeficiency virus infection to patients and healthcare personnel. *Critical Care Medicine, 18*(12), 1440-1448.

Gilbert, T. B., Mcgrath, B. J., & Soberman, M. (1993). Chest tubes: Indications, placement, management and complications. *Journal of Intensive Care Medicine, 8*(2), 73-86.

Girou, E., Stephan, F., Novara, A., Safar, M., & Fagon, J. Y. (1998). Risk factors and outcome of nosocomial infections: Results of a matched case-control study of ICU patients. *American Journal of Respiratory and Critical Care Medicine, 157*(4), 1151-1158.

Gonzalez, R. P., & Holevar, M. R. (1998). Role of prophylactic antibiotics for tube thoracostomy in chest trauma. *American Surgeon, 64*(6), 1343-1348.

Gordon, S. M., Serkey, J. M., Keys, R. F., Ryan, T., Fatica, C. A., Schmidt, S. K., et al. (1998). Secualr trends in nosocomial bloodsteam infections in a 55-bed cardiothoracic intensive care unite. *Annals of Thoracic Surgery, 65*(1), 95-100.

Grap, M. J., & Munro, C. L. (1997). Ventilator-associated pneumonia: Clinical significance and implications for nursing. *Heart & Lung, 26*(6), 419 - 429.

Harris, J. R., Joshi, M., Morton, P. G., & Soeken, K. L. (2000). Risk factors for nosocomial pneumonia in critically ill trauma patients. *AACN Clinical Issues, 11*(2), 198-231.

Harris, J. R., Majjari, J., Morton, P. G., & Soeken, K. L. (2000). Risk factors for nosocomial pneumonia in critically ill trauma patients. *AACN Clinical Issues, 11*(2), 198-231.

Helling, T. S., Gyles, N. R., & Einstein, C. L. (1989). Complications following blunt and penetrating injuries in 216 victims of chest trauma requiring tube thoracostomy. *The Journal of Trauma: Injury, Infection and Critical Care, 29*(10), 1367-1370.

Henry, M., Arnold, T., & Harvey, J. (2003). BTS guidelines for the management of spontaneous pneumothorax. *Thorax, 58 Supplement II*, ii39-ii52.

Hilton, E., Haslett, T. M., Borenstein, M. T., Tucci, V., Isenberg, H. D., & Singer, C. (1988). Central catheter infections: Single- versus triple-lumen catheters, Influence of guide wires on infection rates when used for replacement of catheters. *American Journal of Medicine, 84*(4), 667-672.

Hix, W. R. (1984). Residue of thoracic trauma. *Surgery, Gynecology & Obstetrics, 158*, 295-301.

Hixson, S., Sole, M. L., & King, T. (1998). Nursing strategies to prevent ventilator-associated pneumonia. *AACN Clinical Issues, 9*(1), 76-90; quiz 145-146.

Horl, M. P., Schmitz, M., Ivens, K., & Grabensee, B. (2002). Opportunistic infections after renal transplantation. *Current Opinion in Urology, 12*(2), 155-123.

Horowitz, H. W., Dworkin, B. M., Savino, J. A., Byrne, D. W., & Pecora, N. A. (1990). Central catheter-related infections: Comparison of pulmonary artery catheters and triple lumen catheters for the delivery of hyperalimentation in a critical care setting. *Journal of Parenteral and Enteral Nutrition, 14*(6), 588-592.

Hosmer, D., & Lemeshow, S. (Eds.). (2000). *Applied Logistic Regression.* New York: John Wiley & Sons.

Hsueh, P. R., Chen, M. L., Sun, C. C., Chen, W. H., Pan, H. J., Yang, L. S., et al. (2002). Antimicrobial drug resistance in pathogens causing nosocomial infections at a university hospital in Taiwan: 1981-1999. *Emerging Infectious Diseases, 8*(1), 63-69.

Hurr, H., Hawley, H. B., Czachor, J. S., Markert, R. J., & McCarthy, M. C. (1999). APACHE II and ISS scores as predictors of nosocomial infections in trauma patients. *American Journal of Infection Control, 27*, 79-83.

Hyde, J., Sykes, T., & Graham, T. (1997). Reducing morbidity from chest drains. *British Medical Journal, 314*(7085), 914-915.

Ibrahim, E. H., Tracy, L., Hill, C., Fraser, V. J., & Kollef, M. H. (2001). The occurrence of ventilator-associated pneumonia in a community hospital: Risk factors and clinical outcomes. *Chest, 120*(2), 555-561.

Ibrahim, E. H., Ward, S., Sherman, G., & Kollef, M. H. (2000). A comparative analysis of patients with early-onset vs late-onset nosocomial pneumonia in the ICU setting. *Chest, 117*(5), 1434 - 1442.

Jarvis, W. R. (1996). Selected aspects of the socioeconomic impact of nosocomial infections: Morbidity, mortality, cost, and prevention. *Infection Control and Hospital Epidemiology, 17*(8), 552-557.

Jarvis, W. R., Edwards, J. R., & Culver, D. H. (1991). Nosocomial infection rates in adult and pediatric intensive care units in the united States. National Nosocomial Infections Surveillance System. *American Journal of Medicine, 91*, 185S-191S.

Jerome, R., Scott, K., Blumberg, H., Handerson, P., White, N., Ray, S., et al. (2000). The influence of the composition of the nursing staff on primary bloodstream infection rates in a surgical intensive care unit. *Infection Control and Hospital Epidemiology, 21*(1), 12-17.

Keough, V. (2001). Blunt chest trauma: Review of selected pulmonary injuries focusing on pulmonary contusion. *AACN Clinical Issues, 12*(2), 270-281.

Kline, R. (Ed.). (1998). *Principles and Practice of Structural Equation Modeling*. New York: The Guilford Press.

Knaus, W. A., Draper, E. A., Wagner, D. P., & Zimmerman, J. E. (1985). APACHE II: A severity of disease classification system. *Critical Care Medicine, 10*, 818-829.

Kollef, M. H. (1993). Ventilator-associated pneumonia: A multivariate analysis. *Journal of the American Medical Association, 270*, 1965-1970.

Kollef, M. H. (1999). The prevention of ventilator-associated pneumonia. *New England Journal of Medicine, 340*(8), 627-634.

Laupland, K. B. (2002). Population-based assessment of intensive care unit-acquired bloodstream infections in adults: Incidence, risk factors, and associated mortality rate. *Critical Care Medicine, 30*(11), 2462-2467.

Laupland, K. B., Gregson, D. B., Zygun, D. A., Doig, C. J., Mortis, G., & Church, D. L. (2004). Severe bloodstream infections: a population-based assessment. *Critical Care Medicine, 32*(4), 992-997.

Laws, D., Nevill, E., & Duffy, J. (2003). BTS guidelines for insertion of a chest drain. *Thorax, 58 Supplement II*, ii53-ii59.

LeBlanc, K. A., & Tucker, W. Y. (1985). Prophylactic antibiotics and closed tube thoracostomy. *Surgery, Gynecology & Obstetrics, 160*, 259-263.

Leibovici, L., Greenshtain, S., Cohen, O., Mor, F., & Wysenbeek, A. J. (1991). Bacteremia in febrile patients: A clinical model for diagnosis. *Archives of Internal Medicine, 151*, 1801-1806.

Maki, D. G., McCormick, R. D., Uman, S. J., & Wirtanen, G. W. (1979). Septic endarteritis due to intra-arterial catheters for cancer chemotherapy.I. Evaluation of outbreak. II. Risk factors, clinical features and management. III. Guidelines for prevention. *Cancer, 44*(4), 1228-1240.

Manglano, R., & Martin, M. (1991). Safety of triple lumen catheters in the critically ill. *American Surgeon, 57*(6), 370-372.

Maschmeyer, G., Hoskin, G. A., Ribaud, P., & Sepkowitz, K. A. (2001). Changing patterns of infections and antimicrobial susceptibilities. *Oncology, 14 (Supplement 6)*, 9-16.

McGowan, J. E., Jr., Hall, E. C., & Parrott, P. L. (1989). Antimicrobial susceptibility in gram-negative bacteremia: Are nosocomial isolates really more resistant? *Antimicrobial Agents and Chemotherapy, 33*(11), 1855-1859.

McKinley, S., MacKenzie, A., Finfer, S., Ward, R., & Penfold, J. (1999). Incidence and predictors of central venous catheter related infection in intensive care patients. *Anaesthesia and Intensive Care, 27*(2), 164-169.

Meert, K. L., Long, M., Kaplan, J., & Sarnaik, A. P. (1995). Alterations in immune function following head injury in children. *Critical Care Medicine, 23*, 822-828.

Moro, M. L., Vigano, E. F., & Cozzi Lepri, A. (1994). Risk factors for central venous catheter-related infections in surgical and intensive care units. *Infection Control and Hospital Epidemiology, 15*(4 Pt 1), 253-264.

Nunnally, J., & Bernstein, I. (Eds.). (1994). *Psychometric Theory* (3rd ed.). New York: McGraw-Hill Inc.

Offner, P. J., Moore, E. E., Biffl, W. L., Johnson, J. L., & Silliman, C. C. (2002). Increased rate of infection associated with transfusion of old blood after severe injury. *Archives of Surgery, 137*(6), 711-717.

O'Mahony, J. B., Palcer, S. B., & Wood, J. J. (1984). Depression of cellular immunity after multiple trauma in the absence of sepsis. *The Journal of Trauma: Injury, Infection and Critical Care, 24*, 869-875.

Papia, G., McLellan, B. A., & El-Helou, P. (1999). Infection in hospitalized trauma patients: Incidence, risk factors, and complications. *The Journal of Trauma: Injury, Infection and Critical Care, 47*, 923-927.

Pearson, M. L., & Abrutyn, E. (1997). Reducing the risk for catheter-related infections: A new strategy. *Annals of Internal Medicine, 127*(4), 304-306.

Pepe, J. L., & Barba, C. A. (1999). The metabolic response to acute traumatic brain injury and implications for nutritional support. *Journal of Head Trauma Rehabilitation, 14*(5), 462-474.

Petrosillo, N., Pugliese, G., Girardi, E., Pallavicini, F., Carosi, G., Morro, M. L., et al. (1999). Nosocomial infections in HIV infected patients. Gruppo HIV e Infezioni Ospedaliere. *Aids, 13*(5), 599-605.

Pfaller, M. A., Jones, R. N., Doern, G. V., & Kugler, K. (1998). Bacterial pathogens isolated from patients with bloodstream infection: Frequencies of occurrence and antimicrobial susceptibility patterns from the SENTRY antimicrobial surveillance program (United States and Canada, 1997). *Antimicrobial Agents and Chemotherapy, 42*(7), 1762-1770.

Pittet, D., Harbarth, S., & Ruef, C. (1999). Prevalence and risk factors for nosocomial infections in four university hospitals in Switzerland. *Infection Control and Hospital Epidemiology, 20*, 37-42.

Pittet, D., Li, N., Woolson, R. F., & Wenzel, R. P. (1997). Microbiological factors influencing the outcome of nosocomial bloodstream infections: A 6-year validated, population based model. *Clinical Infectious Diseases, 24*(6), 1068-1078.

Pittet, D., Tarara, D., & Wenzel, R. P. (1994). Nosocomial bloodstream infection in critically ill patients: Excess length of stay, extra costs, and attributable mortality. *Journal of the American Medical Association, 271*, 1598-1601.

Pories, S. E., Gamelli, R. L., Mead, P. B., Goodwin, G., Harris, F., & Vacek, P. (1991). The epidemiologic features of nosocomial infections in patients with trauma. *Archives of Surgery, 126*(1), 97-99.

Potgieter, P. D., Linton, D. M., Oliver, S., & Forder, A. A. (1987). Nosocomial infections in a respiratory intensive care unit. *Critical Care Medicine, 15*, 495-498.

Quirk, J. (2000). Malnutrition in critically ill patients in intensive care units. *British Journal of Nursing, 9*(9), 537-541.

Raad, I., Darouiche, R., Dupuis, J., Abi-Said, D., Gabrielli, A., Hachem, R., et al. (1997). Central venous catheters coated with minocycline and rifampin for the prevention of catheter-related colonization and bloodstream ifnections. A radomized, double-blind trial. *Annals of Internal Medicine, 127*(4), 267-274.

Raymond, D. P., Pelletier, S. J., Crabtree, T. D., Evans, H. L., Pruett, T. L., & Sawyer, R. G. (2003). Impact of antibiotic-resistant gram-negative bacilli infections on outcome in hospitalized patients. *Critical Care Medicine, 31*(4), 1035-1041.

Rebollo, M. H., Bernal, J. M., Llorca, J., Rabasca, J. M., & Revuelta, J. M. (1996). Nosocomial infections in patients having cardiovascular operations: A multivariate analysis of risk factors. *Journal of Thoracic & Cardiovascular Surgery, 112*, 908-913.

Richet, H., Hubert, B., & Nitemberg, G. (1990). Prospective multicenter study of vascular catheter-related complications and risk factors for positive central-catheter culture in intensive care unit patients. *Journal of Clinical Microbiology, 202*, 2520-2525.

Rojo, D., Pinedo, A., Clavijo, E., Garcia-Rodrigues, A., & Garcia, V. (1999). Analysis of risk factors associated with nosocomial bacteraemias. *Journal of Hospital Infection, 42*(2), 135-141.

Ryan, T., Carthy, J., & Rady, M. Y. (1997). Early bloodstream infection after cardiopulmonary bypasss: Frequency rate, risk factors, and implications. *Critical Care Medicine, 25*, 2009-2014.

Safdar, N., & Maki, D. G. (2004). The pathogenesis of catheter-related bloodstream infection with noncuffed short-term central venous catheters. *Intensive Care Medicine, 30*(1), 62-67.

Saint, S., Veenstra, D. L., & Lipsky, B. A. (2000). The clinical and economic consequences of nosocomial central venous catheter-related infection: Are antimicrobial catheters useful? *Infection Control and Hospital Epidemiology, 21*(6), 375-380.

Scheckler, W. E., Bobula, J. A., Beamsley, M. B., & Hadden, S. T. (2003). Bloodstream infections in a community hospital: A 25-year follow-up. *Infection Control and Hospital Epidemiology, 24*(12), 936-941.

Scheckler, W. E., Scheibel, W., & Kresge, D. (1991). Temporal trends in septicemia in a community hospital. *American Journal of Medicine, 91*(3B), 90S-94S.

Schneider, E. (1983). Infectious disease in the elderly. *Annals of Internal Medicine, 98*, 395-400.

Sharma, S., Mullins, R. J., & Trunkey, D. D. (1996). Ventilatory management of pulmonary contusion patients. *American Journal of Surgery, 171*, 529-532.

Stawicki, S. P., Grossman, M. D., Hoey, B. A., Miller, D. L., & Reed, J. F. (2004). Rib fractures in the elderly: A marker of injury severity. *Journal of the American Geriatrics Society, 52*(5), 805-808.

Stillwell, M., & Caplan, S. (1989). The septic multiple-trauma patient. *Infectious Disease Clinics of North America, 3*, 155-183.

Stokes, G. V. (2002). Microbial resistance to antibiotics. In J. Durham (Ed.), *Emerging Infectious Diseases: Trends and Issues* (pp. 23-40). New York: Springer Publishing Company.

Stone, P. W., Larson, E., & Kawar, L. N. (2002). A systematic audit of economic evidence linking nosomial infections and infection control interventions: 1990-2000. *American Jouranl of Infection Control, 30*(3), 145-152.

Tabachnick, B. G., & Fidell, L. (1996). *Using Multivariate Statistics* (3rd ed.). New York: Harper Collins College Publishers.

Tablan, O. C., Anderson, L. J., Arden, N. H., Breiman, R. F., Butler, J. C., & McNeil, M. M. (1994, March 26, 1996). *Part 1. Issues on prevention of nosocomial pneumonia, 1994*. Retrieved March 22, 2003, from http://www.cdc.gov/ncidod/hip/pneumonia/1_bactpn.htm#top

Taylor, G. D., Buchanan-Chell, M., Kirkland, T., McKenzie, M., & Wiens, R. (1994). Trends and sources of nosocomial fungaemia. *Mycoses, 37*(5-6), 187-190.

Taylor, G. D., Buchanan-Chell, M., Kirkland, T., McKenzie, M., & Wiens, R. (1995). Bacteremic nosocomial pneumonia. A 7-year experience in one institution. *Chest, 108*(3), 786-788.

Taylor, R. W., Manganaro, L., O'Brien, J., Trottier, S. J., Parkar, N., & Veremakis, C. (2002). Impact of allogenic packed red blood cell transfusion on nosocomial infection rates in the critically ill patient. *Critical Care Medicine, 30*(10), 2249-2254.

Tennenberg, S., Lieser, M., McCurdy, B., Boomer, G., Howington, E., Newman, C., et al. (1997). A prospective randomized trial of an antibiotic- and antiseptic-coated central veous catheter in the prevention of catheter-related infections. *Archives of Surgery, 132*(12), 1328-1351.

Thelan, L. A., Davie, J. K., Urden, L. D., & Lough, M. D. (1994). *Critical Care Nursing: Diagnosis and Management* (Second ed.). Toronto: Mosby-Year Book, Inc.

Tokras, J., Cookson, S., McArthur, M., Boyer, C., McGreer, C., & Jarvis, W. R. (1999). Prospective evaluation of risk factors for bloodstream infection for patients receiving home infusion therapy. *Annals of Internal Medicine, 131*(5), 340-347.

Uldall, P. R., Merchant, N., Woods, F., Yarworski, U., & Vas, S. (1981). Changing subclavian haemodialysis cannulas to reduce infection. *Lancet, 1*(8234), 1373.

Ullman, R. F., Gurevich, I., Schoch, P. E., & Cunha, B. A. (1990). Colonization and bacteremia related to duration of triple-lumen intravascular catheter placement. *American Journal of Infection Control, 18*(3), 201-207.

van Burik, J. A., & Weisdorf, D. J. (1999). Infections in recipients of blood and marrow transplantation. *Hematology/Oncology Clinics of North America, 13*(5), 1065-1089, viii.

van de Watering, L. M., Hermans, J., & Houbiers, J. G. (1998). Beneficial effects of leukocyte depletion of transfused blood on postoperative complications in patients

undergoing cardiac surgery: A randomized clinical trial. *Circulation 1998, 97*, 562-568.

Vincent, J. L., Bihari, D. J., & Suter, P. M. (1995). The prevalence of nosocomial infection in intensive care units in Europe. Results of the European Prevalence of Infection in Intensive Care (EPIC) Study. *Journal of the American Medical Association, 274*(8), 639-644.

Walker, W. E., Kapelanski, D. P., & Weiland, A. P. (1985). Patterns of infection and mortality in thoracic trauma. *Annals of Surgery, 201*, 752-757.

Wallace, W. C., Cinat, M., Gornick, W. B., Lekawa, M. E., & Wilson, S. E. (1999). Nosocomial infections in the surgical intensive care unit: A difference between trauma and surgical patients. *The American Surgeon, 65*, 987-990.

Warren, D. K., Shukla, S. J., Olsen, M. A., Kollef, M. H., Hollenbeak, C. S., Cox, M. J., et al. (2003). Outcome and attributable cost of ventilator-associated pneumonia among intensive care unit patients in a suburban medical center. *Critical Care Medicine, 31*(5), 1312-1317.

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Research Ethics Board

Waiver of Consent

REB Study #05-MA-006
Examining the impact of chest tube related factors on the risk of nosocomial infections in a community based hospital

Under the terms of the *Tri-Council policy statement: Ethical conduct for research involving humans* article 2.1.C, the requirement to obtain informed consent from the patients whose charts will be reviewed in association with this study is waived. Strategies for ensuring confidentiality and anonymity of the patient information have been satisfactorily outlined in the Ethics Submission Form. The study may proceed under the terms of the Research Agreement.

APPROVED

APPENDIX B

Data Collection Sheet

Patient Identifier_____Age _____ Sex: Male _____Female_____

Patient category: Trauma _____Non-trauma_____

APACHE II score: _____

Indications for chest tube

Pneumothorax _____ Hemothorax _____ Post-operatively _____

For each chest tube used, indicate

Location	Size (small bore or large bore)	Date inserted	Date removed

Blood transfusions given? Yes _____No _____Total # of units_____

Were CVC catheters used? Yes _____No _____Total #_____

Was patient treated with immunosuppressive agents? Yes _____No _____

Number of days of MV during chest tube use _____

Is there evidence of microbial resistance	Yes	_____	No	_____
If yes, does the patient have MRSA?	Yes	_____	No	_____
VRE?	Yes	_____	No	_____
Presence of pre-existing infection	Yes	_____	No	_____

Albumin level on admission _____ when chest tube removed _____

Outcomes:

Did patient develop NI? Yes _____ No _____

If Yes, identify type of infection as:

		Date diagnosed	Number of days since chest tube insertion
Bloodstream			
Pneumonia			

VITA AUCTORIS

Margaret Oldfield was born in 1954 in Windsor, Ontario. She graduated from Essex District High School in 1973. From there she went on to the University of Windsor where she obtained a B.Sc. in Nursing and a B.A. in Psychology in 1977. She is currently a candidate for the Master's of Science degree in Nursing at the University of Windsor, and hopes to graduate in Fall 2005.