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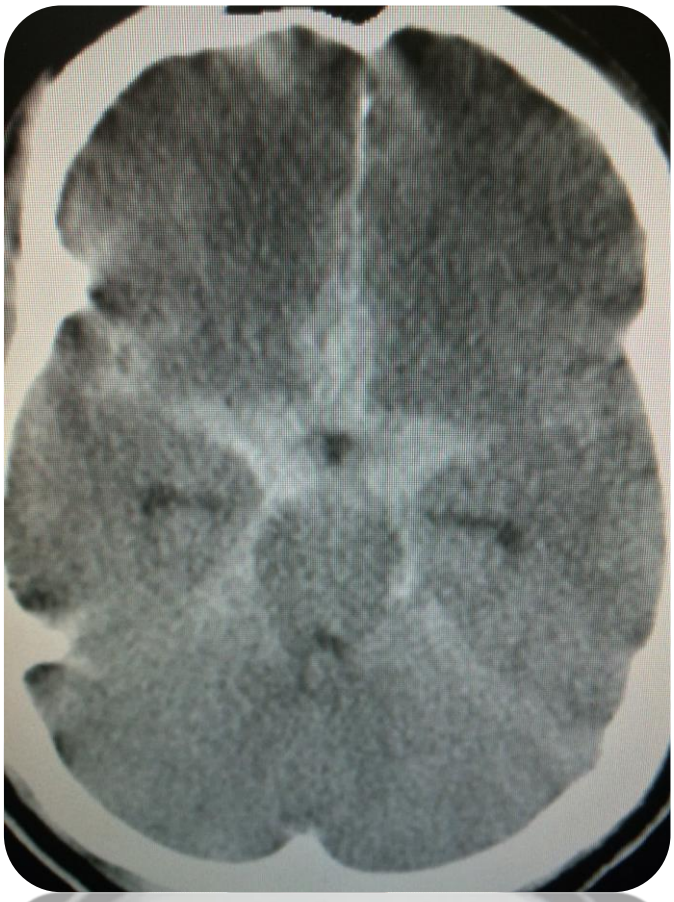

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FROM THE EDITOR'S DESK

January 18, 2016

In the past, the *Jackson Park Hospital Journal* allowed students, residents, and physicians the opportunity to publish their research and observations, and we hope to continue that tradition with the revival of this journal, but in a form more easily accessible. This journal should be used as an educational tool, which will enhance knowledge, encourage bedside learning, and escalate the field of medicine at Jackson Park Hospital.

The journal also serves as an avenue by which staff and students alike may remain aware of the original research that is being conducted at Jackson Park Hospital. Additionally, case reports will also be featured of unique cases managed by the faculty and staff. The hope is that we may work in tandem with one another in our efforts to understand and resolve any disparities that exist within our community and the medical world at large.

While the content of this journal may vary, we encourage submission of research and observations by any member of Jackson Park Hospital. We hope this is an opportunity that everyone will seize to further their career in medicine

Sincerely,

Usman Khan, MD
Dhaval Patel, MD
Afsha Rais, MSY4
Shaan Valji, MSY4
Editors-in-Chief

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ORIGINAL CONTENT

Sepsis in the African American Population

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Jackson Park Hospital and Medical Center

ABSTRACT

According to Centers for Disease Control and Prevention (CDC), there are over 1 million cases of sepsis each year, and is becoming one of the leading causes of deaths in the United States. A general conceptual criterion for diagnosis of sepsis has been established but there are some atypical presentations of the illness that may present. The incidence of sepsis varies among ethnic groups and genders in the population and it appears to be the highest among the African American males.[1, 2] The continuum of sepsis includes an increased risk for mortality. Therefore, it is vital to have a set of criteria for the diagnosis of sepsis in this population in an effort to decrease morbidity and mortality from severe sepsis and eventual multi-organ failure. Furthermore, it is imperative for clinicians to recognize the incidence of atypical presentations in possible septic patients. This study was designed to focus on the presentation of this sepsis in the African American population at Jackson Park Hospital. In doing so, precedence for criteria in the early detection and care for sepsis will be established.

INTRODUCTION

Definitions

The terms systemic inflammatory response syndrome (SIRS), sepsis, severe sepsis, and septic shock was initially defined in 1991 by The American College of Chest Physicians and Society of Critical Care Medicine. In 2001, these terms were further modified to include a broad range of clinical criteria that can assist in early bedside diagnosis of sepsis.

There are many diseases that present with similar physiologic changes in the human body. The phrase "systemic inflammatory response syndrome" was proposed to better understand the general inflammatory response to various insults versus inflammatory processes arising from infection.[1,2] The cardinal signs of SIRS (***Table 1***) include change in body temperature, heart rate, respiratory rate and alternation of white blood count from baseline in the absence of known causes for such abnormalities including chemotherapy and induced leukopenia.[1,2,3,4] When the etiology of an inflammatory insult is infectious, and two or more of the SIRS criteria is identified, the term used is sepsis.

Table 1. Criteria for SIRS	
Temperature	>38° C or <36° C
Heart Rate	>90 beats/minutes
Respiratory Rate	>20 breaths/minute or $PaCO_2$ lower than 32mmHg
White Blood Cell Count	>12,000/uL or <4,000/uL or >10% bands

An infection is classified as a microbial phenomenon. A microbe has the ability to penetrate a tissue when the epithelial barrier is interrupted causing the activation of an inflammatory response by the normally sterile host tissue. Severe sepsis becomes the diagnosis when there is involvement of an organ dysfunction, hypoperfusion abnormality or sepsis-induced hypotension.[2] Severe sepsis is now considered to be the most common cause of death in non-coronary critical care units.[4] Examples of organ dysfunction include – but are not limited to – decrease in urinary output, lactic acidosis, acute altered mental status, acute respiratory distress, and the presence of coagulapthy.[3]

Sepsis-induced hypotension is defined as blood pressure of less than 90 mmHg systolic or its reduction by 40 mmHg or more from baseline blood pressure, in the absence of other cause for hypotension.[2] Septic shock refers to late stage sepsis which occurs when the cache or pro-inflammatory molecules existing within the body are provoked by an infectious etiology, thereby inducing hypotension secondary to systemic vasodilation, or decreased vascular resistance. This physiological disturbance often persists despite adequate volume resuscitation. ***Figure 1*** illustrates a conceptual framework of the relationship between SIRS, sepsis, severe sepsis and septic shock.

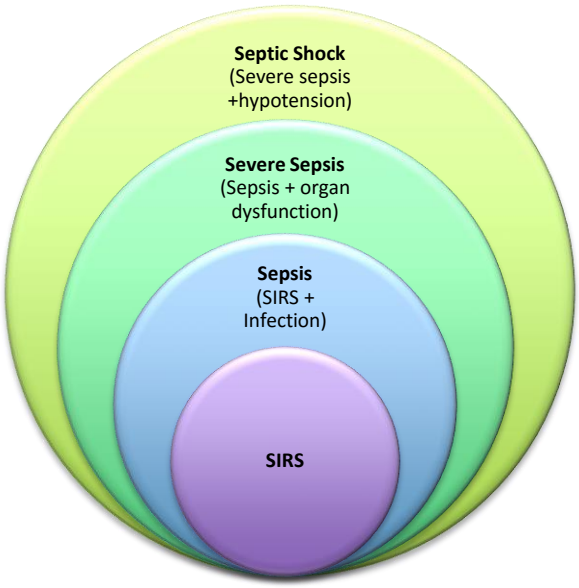

Figure 1. Relationship from SIRS to septic shock

The 2001 Consensus Conference resulted in modified criteria to increase the accuracy and clinical diagnosis of sepsis. The expanded definition of sepsis enhanced the SIRS criteria to a list of six principles: sepsis, general parameters, inflammatory parameters, hemodynamic parameters, organ dysfunction parameters and tissue perfusion parameters (***Table 2***).[3, 4]

Table 2: Modified criteria for sepsis based on the 2001 Consensus Conference [3,4]

Sepsis	Documented or suspected infection
General parameters	Fever, hypothermia, tachycardia, tachypnea, altered mental status, significant peripheral edema or (+) fluid balance, hyperglycemia in absence of diabetes
Inflammatory parameters	Leukocytosis (>12K/uL), leukopenia (<4K/uL), >10% bands with normal WBC, increased C-reactive protein, increased procalcitonin
Hemodynamic parameters	Hypotension, elevated cardiac index
Organ dysfunction parameters	Hypoxemia, acute oliguria, increased creatinine level, coagulation abnormalities, thrombocytopenia, hyperbilirubinemia, ileus
Tissue perfusion parameters	Lactic acidosis, decreased capillary refill, skin mottling

Epidemiology

Bacterial infection leading to systemic illness has become a progressively common condition among hospitalized patients. These patients are also considered severely ill when compared to patients admitted with comparable diagnoses. In the recent years, there has been an increase in the incidence of sepsis, severe sepsis and sepsis related deaths in the United States. It affects more than 750,000 people annually, with an estimated annual mortality between 25% to 30% for severe sepsis and nearly 50% for septic shock.[5, 6] A fourth of the hospitalized patients with severe sepsis will expire during the course of their treatment. A plethora of hypotheses exist in an effort to explain mortality from sepsis, some of which include: increasing life expectancy, increasing prevalence and incidence of chronic diseases, immunosuppression, invasive procedures and multidrug-resistant infections.[2] It is the lethality of sepsis that indicates the need for early recognition and treatment with appropriate antimicrobial agents.

Risk factors

Many factors predispose a patient in the risk for developing sepsis. For example, advanced age greater than sixty-five, obesity, male gender, black race and increased chronic diseases are important risk factors for the development and continuum of sepsis.[2] More than half of patients who have progression of sepsis to severe sepsis have at least one chronic health condition, including diabetes mellitus, cancer, chronic pulmonary, renal and liver disease. Cancer is one of the most common co-morbidities in which patients experience severe sepsis, with the source of infection being related to the type of cancer.[2] For example, individuals with lung cancer are at 10-fold risk of developing pneumonia. The proposed explanation is due to the depression of host defense system, signifying that any disease process that suppresses the host immune system is at increased risk for developing sepsis. Environmental risk factors must also be taken into account, especially with regards to the seasons. It has been found that respiratory infections peak in the winter months while genitourinary infections peak in the summer. [2]

Studies consistently report a higher incidence of severe sepsis among black patients when compared to white patients. It is somewhat unclear, however, whether these statistics are due to higher infection rate among the black population, differences in susceptibility to infections, or an increased risk of developing acute organ dysfunction.[2,7] It is imperative to understand why this population is at risk in an effort to decrease mortality from sepsis, severe sepsis and septic shock during hospitalization. Many African Americans have a condition known as “benign ethnic neutropenia,” however, it remains in question whether this condition puts the population at an increased risk of infection. On the other hand, it has been established that a higher prevalence of chronic diseases among the black population leads to an increased incidence of organ dysfunction. In this case, primary prevention should be the goal with close follow up and management of chronic diseases. Furthermore, efforts at implementing primary prevention at the community level – such as increasing availability of vaccinations – would help abolish any increase in incidence of sepsis is due to differences in susceptibility to infections. [7]

Hematopoiesis: Granulocytes/Leukocytes

Hematopoiesis is the production of new blood cells in the body. The major cell line responsible for inflammation and immune response is known as granulocytes, also known as polymorphonuclear leukocytes (PML). There are different types of granulocytes, including neutrophils, eosinophils, basophils and mast cells. The blood cell lineage is portrayed in ***Figure 2***. The most abundant of these cells are neutrophils. The main function of neutrophils is to destroy bacteria, but they also play an important role in defense against other infections, including those of viral etiologies. The normal functioning of neutrophils depends on their ability to leave the bone marrow and anchor to the endothelium. During the inflammatory process, they must then demarginate from this endothelium and migrate between neighboring cells in response to chemotactic gradients to ingest and kill the microbial invader.[14] A defect in any of these functionalities can increase the risk of developing an infection.

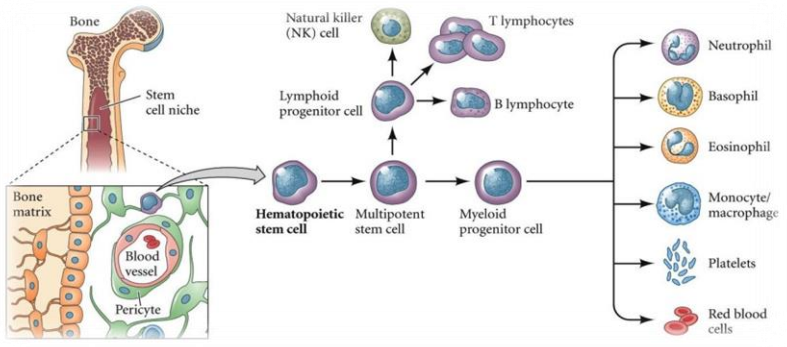

Figure 2. Hematopoiesis – the blood cell lineage

http://www.studyblue.com/notes/note/n/stemcell-/deck/11180611

Neutropenia is defined as a reduction in circulating PML and increase in band formation (immature neutrophils) with an absolute neutrophil count below 1.5 x 10^9/L in children and adults over the age of one.[13] Because neutropenia is the single most important risk factor for an infection, the ranges have been classified as mild (1.0 to 1.5 x 10^9/L), moderate (0.5 to 1.0 x 10^9/L) and severe (<0.5 x 10^9/L).[13] The relationship between neutropenia and infection has been a main focus for researchers for many years. It was originally recognized in 1902 by Brown and Ophuls who found the occurrence of severe leukopenia in a patient with fatal infectious pharyngitis. Further research in 1930 by Roberts and Kracke showed that neutropenia came prior to the infection.[13] Years later, Doan was able to differentiate between acute and chronic neutropenia, highlighting bone marrow insufficiency as a very possible and relevant etiology of chronic neutropenia. This eventually became known as "maturation arrest," which is a decrease in the production of mature cells from the cell line itself.[13]

Normal blood leukocyte counts are generally between 4 – 10 x 10^9/L, with neutrophils representing 45–74% of the cells, bands 0–4%, lymphocytes 16–45%, monocytes 4–10%, eosinophils 0–7%, and basophils 0–2%.[14] These values differ among individuals of different ethnic groups, with lower leukocyte numbers for African-American ethnic groups.[14] As previously mentioned, each of these cells have specific functions and can be informative in diagnosing a disease process. Because an alteration in the number and type of leukocytes can be associated with specific disease processes, total white blood cell (WBC) count (cells per µL) and differential counts should be obtained.[14]

Benign Ethnic Neutropenia

The African American population is at an increased risk for sepsis for reasons that remain unclear. A study conducted by Broun reported significant granulocytopenia in 25% of black patients admitted for elective surgery.[13] Another study conducted by Karayalcin found that 30% to 40% of 231 black hospital employees had low WBC values attributed to a significant decrease in granulocytes.[13] The second National Health and Nutrition Examination Survey conducted in 1978 to 1980 demonstrated that 42.6% of black females ages 3 to 74 years had leukopenia with neutropenia, whereas only 27.1% of white females in that age group had WBC levels below 5.0 $x10^9$/L. Among black males between the ages of 3 to 74 years, 48.1% had WBC levels below 5.0 $x10^9$/L compared to 25.2% of white males.[13] Benign ethnic neutropenia (BEN) is commonly noted in African American adults as a diagnosis of exclusion.[7] This condition is classified as neutrophil counts less than 1.5 x 10^9 cells/L.[12] There have been many discussions on the etiology of BEN, whether it is inherited or acquired due to environmental factors, including poor nutrition.[13]

It is important to differentiate BEN from other causes of neutropenia including congenital neutropenia, cyclic neutropenia and chronic idiopathic neutropenia. These conditions are common in the white population, associated with severe neutrophil count less than 0.5 x 10^9 cells/L, and lead to oral, cutaneous or systemic infections.[12] History and physical examinations are imperative in diagnosing BEN. It is important to ask questions about frequency and duration of infections, severity of infections, exposures to toxins, and symptoms of malabsorption.[13] Physical exam should include comprehensive evaluation for infection with attention particularly to the ears, lungs, soft tissues, oral mucosa, gingiva and perianal mucosa. These findings are important in excluding other causes for neutropenia, which should be eliminated prior to diagnosis of BEN. These include but are not limited to pharmacologically induced suppression, comorbid infections, neoplastic disease, autoimmune disease, hematologic/oncologic disorders, and metabolic disturbances.[12]

The prevalence of BEN is low in the United States when compared to the African population outside of the United States. Most recent data showed that BEN is described in up to 25% to 40% of those of African descent outside of the United States, while 4% of adult African American men and 2% to 3% of African American women in the United States.[12] Many studies have been concluded to determine the mechanism of BEN. Mason confirmed that bone marrow aspirates from normal healthy neutropenic black adults had normal cellularity and normal maturation of all cell lines.[13] This indicates that the defect in these patients was in the release of mature granulocytes from bone marrow storage to the circulating blood.[13]

A more detailed study on BEN was published in the Journal of Clinical Oncology by Hsieh, Tisdale and Rodgers to determine when to initiate or resume chemotherapy in cancer patients with BEN. Analysis of direct bone marrow examination in 12 individuals with BEN showed normal cellularity and leukocyte maturation. A retrospective analysis showed bone marrow from 240 African American donors contained lower nucleated cell and CD34+ cell number per recipient weight.[12,13] Many studies continued to show that African Americans had lower total nucleated cells. The summary concluded from the retrospective and meta-analysis was such that African Americans have normal stem-cell number, normal myeloid maturation, and a minor reduction in hematopoietic myeloid progenitors at steady-state.[12] The minor reduction may account for the

lower number of demargination of neutrophils from the vascular endothelium and bone marrow stores during stress, such as infections. However, the exact etiology still remains unclear.

Pathogenesis of Sepsis

The normal physiologic response to an infection is a complex process that recognizes the microorganism invasion and initiates repair of the injury caused by the invasion. There are many mediators that come into play to fight the infection in an immunocompetent host. However, a microorganism that does not ordinarily cause systemic disease in an immunocompetent host can still occur and lead to sepsis. The human immune system works in a very sophisticated way to ensure that infections are defeated prior to injury to tissue. Nevertheless, certain microbes often exploit acquired deficiencies in host defenses, indwelling catheters or foreign matter, or obstruct fluid drains to survive within the human body.[16] This explains the variation in the source of infection from respiratory, genitourinary, gastrointestinal, skin and soft tissue.[9] The most common site of infection in patients with sepsis is the respiratory tract, with pneumonia being the leading presentation. Furthermore, gram-positive microorganisms have increased in frequency over gram-negative infections, with a small number of patients manifesting with fungal, viral or parasitic infections.[2,9]

The process for regulating infection invasion is quite intricate. It is regulated by a mixture of pro-inflammatory and anti-inflammatory mediators. These are secreted by the body's immune host defenses, such as macrophages, that become activated and invade tissues to engulf the microbe. The main pro-inflammatory mediators include tumor necrosis factor-alpha (TNFα) and interleukin-1 (IL-1).[15] TNFα stimulates leukocytes to release other mediators to express cell-surface antigens that will allow neutrophil adhesion at the site of infection. The other mediators released are the interleukins responsible for B-cell and T-cell release to destroy microbes causing infections. It is TNFα, along with IL-1, that is in large part responsible for fever, tachycardia, hypotension and other nonspecific symptoms of illness that occur during a course of an infection. Anti-inflammatory mediators, such as IL-10, are cytokines that inhibit the production of excess TNFα and IL-1. TNFα at high levels can lead to shock and death, which explains why anti-inflammatory mediators are necessary.[15] Other important mediators include coagulation factors. Intravascular thrombosis, an inflammatory response, is important to wall off the microbe causing the infection. This helps prevent the spread of inflammation and infection to surrounding tissues.[16]

The balance between pro-inflammatory and anti-inflammatory mediators explains the normal response to infection invasion – adherence, chemotaxis, phagocytosis of microbe, killing of microbe and cleaning of debris from injured tissue.[11] Sepsis is the release of pro-inflammatory mediators beyond the limited boundaries of the infection to cause a more generalized response.[15] Pinsky and Matuschak conceptualized sepsis as malignant intravascular inflammation – uncontrolled, unregulated and self-sustaining.[17] It was thought to be intravascular because the blood spreads mediators that are confined to cell-to-cell interactions within the interstitial space and inflammation because the characteristics visualized during sepsis are augmentations of normal inflammatory response.[11,16,17] Occasionally during sepsis, the circulating bacteria causing the infection can elicit multi-organ dysfunction and hypotension directly by overstimulating

inflammatory responses within the vasculature.[16] This process explains the transition from sepsis to severe sepsis and septic shock.

The risk of developing sepsis depends on the type of microorganism invading the host. The bacterial cell wall components, such as endotoxin, peptidoglycan, and lipoteichoic acid and the bacterial toxins contribute to the development of sepsis. In a study conducted on a large series of patients with positive blood cultures, the risk for developing severe sepsis was related to the site of the primary infection.[16] Results showed that bacteremia arising from pulmonary or abdominal sources were eightfold more likely to be associated with severe sepsis than urosepsis.[16] Bacterial proteins and toxins lead to cellular injury and tissue damage, which is the precursor to organ dysfunction. This process is accompanied by excess release of pro-inflammatory and anti-inflammatory mediators, worsening organ dysfunction and ultimately leading to organ and multi-organ failure – the hallmark of severe sepsis. The pathogenesis of severe sepsis thus may differ according to the microorganism, the ability of the host's innate defense mechanisms to sense and respond to it, the site of the primary infection, the presence or absence of immune defects, and the prior physiologic status of the host.[11,15,16,17]

METHODS

The study was a retrospective, population-based analysis of hospitalized patients from April 2013 to September 2014 with the diagnosis of sepsis and septicemia at the Jackson Park Hospital, Chicago, IL. The study was limited to African American patients because of the rise in incidence rates of sepsis seen in this population. There were set exclusion and inclusion criteria prior to data collection to ensure accurate determination of presentation of sepsis in African American population. The exclusion criterion was immunocompromised patients including those with cancer either receiving or not receiving chemotherapy. The inclusion criteria were as follows: 1) must meet some of the criteria for SIRS; 2) show evidence of bacteremia; 3) probable multi-organ failure; 4) must have normal white blood count and/or be afebrile. The evidence of bacteremia was determined by cultures obtained: blood, urine, sputum, wound.

The data was classified into SIRS criteria on arrival and on day 4 of admission. This helped recognize the initial presentation of patients with sepsis or pre-sepsis, in order to determine management and treatment plan. While eradicating the microbe is essential in treatment of infection, we analyzed when the first antibiotic was given to the patient and how that altered the SIRS criteria. Lastly, we evaluated risk of organ dysfunction on day 4 of admission and the disposition of the patient during their hospital visit. Database management and calculation of statistics were performed using Microsoft Excel.

RESULTS

A total of 139 patients with the diagnosis of sepsis or severe sepsis were studied. While more patients had the diagnosis of sepsis, they did not meet our inclusion criteria and many were excluded due to their immunosuppressed state. The average age of the patients in the study was 66.6 years old with the range between 22 years to 98 years old. The presence of infection was determined by cultures obtained on admission. Among those that were in the study, the most

common infection was site unspecified, indicating that there was bacteremia but the exact site is unknown and/or multiple. Of the multiple sites, patients had concurrent bacteremia and urinary infection. Additionally, of the patients with positive cultures, 15 were MRSA positive ***(Table 3)***. Other common areas of infections were respiratory, genitourinary, and wound. There were 4 patients who had suspected sepsis but continued to have negative cultures during their hospitalization.

Table 3: Number of patients and their sites of infection

Characteristic	**# of patients**
Site of infection	
Respiratory	9
Genitourinary	8
Wound	13
Bacteremia, exact site unspecified	50
Error in obtaining culture	32
Negative culture in suspected sepsis	4
Multiple sites of infection	23

Table 4: SIRS criteria - on admission

Febrile	20
Tachycardia	70
Tachypnea	37
Leukocytosis	75
Leukopenia	4
Elevated percentage of neutrophils	105

The data was further analyzed to define the presentation of patient on admission. It was essential to determine the SIRS criteria on admission to determine if the African American population presents with signs differ from innate SIRS criteria. ***Table 4*** depicts how many patients presented with SIRS criteria. A commonality determined during the study was the increased percentage of neutrophils and absolute neutrophil counts in patients with the diagnosis of sepsis/severe sepsis. Of the 139 patients, 54% had elevated white blood count and 50% presented with tachycardia. However, 76% presented with elevated percentage of neutrophils, indicating that this value should be considered when evaluating African Americans for risk of sepsis (10.3, 95% CI 68.03-82.83%, p <0.0001).

According to the original guidelines of diagnosis of SIRS, only 67 patients (48.2%) met two or more of the criteria. Additionally, 31 patients (22%) presented with elevated neutrophils, positive culture, and diagnosis of sepsis but had less than one of the SIRS criteria. While a significantly low percentage, it is important to acknowledge all the factors in diagnosis sepsis to decrease mortality in the African American population.

Table 5: Number of patients and time of antibiotic initiation; Disposition	
Initiation of antibiotics	
Within 1 hour of admission	16
Within 4 hours of admission	34
Within 12 hours of admission	67
Within 24 hours of admission	18
Within 48 hours of admission	0
Within 72 hours of admission	1
Within 96 hours of admission	3
Disposition	
Deceased during hospitalization	45
Discharged (home/nursing home/rehab/etc.)	94

Early initiation of antibiotics in suspected sepsis improves the outcome for the patient. Of the patients receiving antibiotics, 48.2% were given antibiotics within 12 hours of admission. Furthermore, 67.6% of the patients with sepsis and/or severe sepsis were discharged from the hospitalization, whereas 32.3% deceased during treatment (difference of 35.2%, 95% CI 23.24 to 46.07%, p<0.001). The most common cause of death was multi-organ failure from infection, despite initiation of antibiotics during hospitalization. Of the 45 patients who deceased during hospitalization, 5 patients deceased within a day of admission, 1 patient within two days of admission, 4 patients within three days of admission and the remaining 35 after four or more days of admission.

Lastly, it was determined that the percentages of PMNs were elevated in 76% of the patients in the study. Using this measurement, the SIRS criteria plus the percentage of neutrophils were evaluated on day four of admission, where applicable. Forty-seven patients continued to have elevated percentage of neutrophils after antibiotics were initiated, even without leukocytosis. This data could be used to determine the rate of infection eradication. Additional data was collected, but was not adequate for analysis or to yield accurate results. The utilization of this data will be discussed under recommendation for continuation of research.

DISCUSSION

Prompt diagnosis and treatment for sepsis is essential to decrease mortality and increase outcome. Delay in the management of sepsis can lead to severe sepsis and shock, which yields poor outcome. A meta-analysis of randomized trials in 2008 concluded that applying early quantitative resuscitation on patients with sepsis significantly reduces mortality.[6,16,18] The early resuscitation included administration of fluids within six hours of admission, in part to avoid persistent hypoperfusion.[8,15,16,18] Next, it is imperative to determine the site of infection to properly eradicate the infection using appropriate antimicrobial therapy. In a retrospective study conducted between July 1989 and June 2004 in fourteen intensive care units, it was concluded that effective antimicrobial administration within the first hour of documented hypotension increased survival and discharge from hospitalization in adult patients with septic shock.[6,18,19] Therefore, the time and duration for diagnosing sepsis and initiating appropriate antimicrobial therapy is the strongest predictor of mortality in patients.[6,8,15,18,19]

The purpose of this study was to determine if presentation of sepsis differs in the African American population, in which mortality from sepsis appears to be high. During the study, 67% of the patients met the SIRS criteria, and while tachycardia and leukocytosis were evident in approximately 50% or more of the patients, it was still not enough to diagnose the patient with

sepsis and initiate early treatment. As seen in the results, only 16 patients were treated with antibiotics within the first hour of admission, with majority receiving therapy within 12 hours of admission. However, only 20 patients spiked a fever during their admission, even with positive cultures showing evidence of sepsis. Analysis showed 76% of the patients had elevated percentage of neutrophils on admission. Therefore, it is important to understand if this finding can be useful in identifying risk of sepsis in the African American population, where leukocytosis and fever may not always elevate to suggest a sign of infection.

Neutrophilia, or an increase in the number of neutrophils circulating in the body, can be associated with many conditions, including inflammation or any active infection, pregnancy, chronic disease of the bone marrow such as leukemia, shock, trauma, family history, recent vaccination, and many more.[8,10,14,16,20] In a study conducted on 292 patients, the neutrophil left-shift parameter was evaluated as a marker for inflammatory and/or infectious disease process.[20] The focus of the study was to determine if presence of bands can be an indication for infectious process. It was concluded that while band count may have high specificity for inflammation, it is not useful as a screening test due to low sensitivity.[20] The study also concluded that neutrophilia can be embarked secondary to stress, and not necessarily as part of an infectious process. However, it could be argued that an infection and stressful occurrence in the body can be congruent.

CONCLUSION

There is an increase in the incidence of sepsis in the African American population, secondary to delay in diagnosis. The study portrayed some inconclusive data that requires further investigation. While this may be evident, there were promising results to better evaluate the presence of infection leading to sepsis in the African American population. Clinicians must evaluate the patient thoroughly and maintain a high index of suspicion whilst evaluating the laboratory results to determine the early signs of infection in this population. Applying the SIRS criteria is effective and should be considered, but sepsis must not be preemptively ruled out in African American patients who do not fit established criteria.

Many African American patients present with chronically low level of leukocytes, which may cause hindrance when applying SIRS criteria. Furthermore, many infectious processes are noted by leukocytosis – leading to missed diagnoses within the studied population, increasing risk for severe sepsis and possibly septic shock. Therefore, it would be advised to obtain differentials when ordering a complete blood count to see if there is an elevation in the percentage of neutrophils. Early treatment in patients with suspected sepsis can be achieved by obtaining these results proactively when evaluating for leukocytosis or leukopenia. The results that may be obtained in future studies as suggested by this analysis may be promising in the attempt to decrease morbidity and mortality in septic patients both in the African American population as well as the general population.

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ORIGINAL CONTENT

Coping Through Texting: A Way to Regulate Emotions

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INTRODUCTION

The popularity of texting in today's society can be observed simply by going through your daily routine where you will likely encounter people tapping, dragging, and sliding their fingers across their cell phone screens as other tasks such as driving, walking, listening, working, or studying are neglected, if not abandoned entirely. In these instances, it is common for people to assume that the texter's motives are social—a desire to communicate with friends instead of participating in present activities that they deem not as interesting. However, another possibility that is rarely discussed and scarcely researched is that people may text to regulate their emotions and improve their mood.

Students who text during class are typically viewed as lacking discipline to desist from an activity they enjoy. An adolescent who decides to text friends during a family gathering is viewed as inconsiderate of family elders and the importance of spending quality time with them. A party attendee who is more consumed with texting than the festivities is viewed as arrogant in their belief that they have more important matters to discuss than the entertainment. Such views of texting assume that the decision to text is primarily an attempt to socialize, albeit excessively and unnecessarily. However, these assumptions overlook the possibility that other motives may be responsible for texting besides being social.

A student's decision to text during class could be motivated by a desire to cope with the stress related to difficulty grasping what is being taught. From this viewpoint, texting would be motivated by a desire to avoid and escape rather than socialize. Similarly, dissatisfied employees may text excessively while on the clock to help cope with feelings of job dissatisfaction, depression, burnout, or value conflict between their personal values and those of their employers. Similarly, coping with social anxiety could also be a better explanation for why people may text during family gatherings or parties than social motives. Research supports this possibility and has shown that adolescents who reported more social anxiety were more likely to prefer texting over talking on the phone or talking in person (Reid & Reid, 2007).

Other research about texting has shown correlations between personality traits related to emotion dysregulation, such as impulsivity and neuroticism, with more frequent texting (Billieux, Van der Linden, d'Acremont, Ceschi, & Zermatten, 2007). Billieux and associates' (2007) discussion of texting and impulsivity supports the concept of emotion regulation motives for texting because impulsivity entails experiencing strong urges in the presence of stress or negative affect as well as an inability to maintain focus on difficult, boring, or stressful tasks. Contrary to impulsivity, emotion regulation refers to the ability to effectively manage emotional responses

in a manner that is appropriate to the situation and social expectations and allows for goal-directed behavior (Gratz & Roemer, 2004).

In 2005, mindfulness teacher and researcher Jon Kabat-Zinn suggested that individuals might excessively use communication technology to distract themselves from upsetting emotional states. Kabat-Zinn's (2005) suggestion represented the first time someone introduced the idea that people may use communication technology, which would include texting, as a means of regulating their emotions. Texting may offer individuals an opportunity to regulate their emotions by shifting their attention away from undesirable thoughts, moods, and emotions, and instead focusing them on the topic of the text conversation. This shift in attention may be subtle and occur with little awareness that the motive for texting is related to a desire to escape undesired thoughts and emotions. It is likely that these distracting text conversations would be random, lack a clear purpose, and involve sending generic questions or comments towards the recipient (e.g., "wyd," what you doing?).

OBJECTIVE

Examining if people text to regulate their emotions and cope with negative mood is an important next step to take regarding research about texting. Only one published study was found that explored the possibility of emotion-regulation motives for texting, and this study was correlational and limited to texting while driving (Feldman, Greeson, Renna, & Robbins-Monteith, 2011). Feldman and associates (2011) found a significant relationship between emotion regulation motives for texting and texting while driving. The current study sought to expand upon these findings by experimentally manipulating mood to see if a negative mood would increase the desire to text. Through experimental manipulation, this study sought to rule out alternative explanations for excessive and problematic texting that could have been responsible for Feldman and associates' (2011) results, such as social motives for texting, personality traits, or stress levels. Experimental manipulation was chosen to allow the current study to more definitely suggest that emotion regulation motives for texting is the main factor that influences the desire to text when experiencing negative emotions and mood.

The current study also expanded beyond the risky behavior of texting while driving studied by Feldman and associates (2011). This was done by examining whether emotion-regulation motives for texting are associated with more texting-related problems, such as poorer academic performance, diminished interpersonal relationships, or work-related problems. Researchers have started to look at excessive and problematic texting, and, in some cases, measures have been developed to assess the varying levels of text dependence and problematic usage (Atchley & Warden, 2012; Bianchi & Phillips, 2005; Igarashi, Motoyoshi, Takai, & Yoshida, 2008; Rutland, Sheets, & Young, 2007). However, except for Feldman and associates' (2011) study concerning texting while driving, no published studies were found that used such measures of problematic texting to see how they would be associated with an emotion regulation motive for texting. Existing research related to substance abuse literature has shown that emotion regulation motives for alcohol use are associated with more alcohol related problems (Cooper, 1994). Thus, this study similarly sought to determine if emotion regulation motives for texting were associated with more problematic outcomes from texting.

The current study explored how difficulties in emotion regulation are related to emotion regulation motives for texting as well as texting-related problems. Emotion regulation is best defined by Gratz and Roemer (2004), who conceptualized emotion regulation as involving the (a) awareness and understanding of emotions, (b) acceptance of emotions, (c) ability to control impulsive behaviors and behave in accordance with desired goals when experiencing negative emotions, and (d) ability to use situationally appropriate emotion regulation strategies flexibly to modulate emotional responses as desired in order to meet individual goals and situational demands. Based on this conceptualization, it seems likely that good emotion regulation would reduce the desire to text when experiencing negative emotions or moods. Individuals who are better at regulating their emotions are likely to quickly and effectively manage their emotions through their thought processes, outlook, and perspective, which would preempt a desire to text to achieve this effect. Subsequently, less texting-related problems would occur in individuals with fewer difficulties with emotion regulation.

The current study sought to continue the work of scholars such as Kabat-Zinn (2005), Feldman and associates (2011), and Billieux and associates (2007) by going beyond a theoretical or solely self-report, correlational approach to studying desire to text and emotion-regulation motives for texting. This approach provides stronger evidence that mood can affect the desire to text. Thus, the goal of the following study was to address the following questions: Do individuals' text to regulate their emotions when experiencing a negative mood? Are emotion regulation motives for texting associated with greater texting-related problems? Will individuals with greater difficulties in emotion regulation have greater emotion regulation motives for texting as well as experience greater texting-related problems?

Hypotheses

Hypothesis 1. Participants in the negative mood induction group will report a higher desire to text post-induction compared to the neutral mood induction group.

Hypothesis 2. Participants in the negative mood induction group with the highest emotion regulation motives for texting will also report the highest desire to text post induction.

Hypothesis 3. Higher emotion regulation motives for texting will be correlated with greater texting-related problems.

Hypothesis 4. Greater difficulties in emotion regulation will be correlated with greater emotion regulation motives for texting.

Hypothesis 5. Greater difficulties in emotion regulation will be correlated with greater texting-related problems.

Significance of Study

Testing if individuals' text to regulate their emotions is a concept worthy of investigating and has clinical significance. Existing research has shown associations between excessive texting and diminished psychological well-being such as increased stress levels, sleep disturbances, and symptoms of depression (Thomée, Härenstam, & Hagberg, 2011). Additionally, recent data have shown that texting has increased in prevalence and popularity, increasing the need and urgency to understand its influence and effects (Lenhart, 2010). A 2010 survey conducted by the Pew Institute was revealing in showing that the number of text messages sent monthly in the United

States exploded from 14 billion in the year 2000 to 188 billion in 2010 (Lenhart, 2010). Lenhart (2010) also found that Americans ages 18-29 send and receive an average of nearly 88 text messages per day, compared to only 17 phone calls and 32% of all respondents said they would rather communicate by text rather than talking, even with people they know very well. Such data demonstrates how prevalent texting has become and advocates for research like the current study which seeks to provide more understanding of the underlying processes that lead to texting, and the outcomes associated with these motives. Given the popularity, accessibility, lack of social stigma, inexpensiveness, and increased reliance that seems apparent in regards to texting, it is important to determine if other motives, such as emotion-regulation, underlie texting in addition to social motives.

METHODS

The current study involved an experiment using an autobiographical mood induction procedure to induce either a negative or neutral mood to test if individuals text to regulate their emotions. This study represented the first known study to specifically measure the direct effects of mood on the desire to text, and do so with the use of experimental manipulation. This study borrowed the concept of the experiment from substance abuse literature (Birch, Stewart, Wall, McKee, Eisnor, & Theakston, 2004) that tested if individuals who drink alcohol to cope have an increased desire to drink upon experimental induction of a negative mood. The primary goal of this study was to test if individuals text to regulate their mood in the presence of negative emotions and moods. Furthermore, this study examined whether individuals with higher self-reported emotion-regulation motives for texting have the highest desires to text in the presence of negative moods. The study also examined whether texting to regulate emotions as opposed to texting for other purposes was associated with more texting-related problems. Lastly, the study examined if greater difficulties in emotion regulation was related to greater emotion regulation motives for texting as well as less texting-related problems.

RESULTS

Hypothesis 1: Effect of Mood Induction on Desire to Text

The first hypothesis addressed whether the negative mood-induction group would report a higher desire to text post-induction than the neutral mood-induction group. Data was collected from 159 participants regarding their desire to text after the mood induction procedure with 84 of those participants placed in a negative mood-induction group and the other 75 participants in a neutral mood-induction group. Hypothesis 1 was not supported and the t-test was not computed because the mean of the neutral mood group's desire-to-text (M= 6.73) was higher than the negative mood group's desire-to-text (M= 6.31). Therefore, contrary to Hypothesis 1, participants randomly assigned in the negative mood-induction group actually reported less desire to text than participants placed in the neutral mood-induction group. The lack of support for Hypothesis 1 despite the effectiveness of the mood induction procedure being statistically significant could be due to the mood induction procedure's relatively small effect size (Cohen's d= .37, r=.18). Although Hypothesis 1 was not supported by the experimental results, it was

potentially supported by correlational results which showed that when considering participants' self-reported mood, regardless of the mood-induction group they were randomly assigned to, negative mood was correlated with a higher desire to text, r= -.159, n=159, p<.05 (see Table 2).

Hypothesis 2: Emotion-Regulation Motives as Moderator of Mood Effect on Desire-to-Text

The second hypothesis predicted that high emotion regulation motives for texting would have a moderating effect on the relationship between mood and desire to text such that it would result in a stronger desire to text. Desire to text scores were subjected to a two-way analysis of variance with two levels of mood-induction group (negative, neutral) and two levels of emotion-regulation motives (high, low). Participants who scored ½ of 1 standard deviation above the sample mean were placed in the high group, while participants who scored ½ of 1 standard deviation below the sample mean were placed in the low group. The second hypothesis was not supported. As seen in Table 1, the interaction effect between mood group and emotion-regulation motives was not significant, $F(1,109)= .339$, $p>.05$. Therefore, contrary to Hypothesis 2, participants with high emotion-regulation motives in the negative mood-induction group did not have a greater desire to text than participants with low emotion-regulation motives in the negative mood induction group. As noted above however, due to the small effect size of the mood-induction procedure, mood differences between the mood-induction groups may have been too small to result in sufficient statistical power to detect an interaction effect between mood and another variable.

Table 1: ANOVA for Mood-Induction Groups, Emotion Regulation Motives and Desire to Text

Dependent Variable: Desire To Text

Source	Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	407.849[a]	3	135.950	17.521	.000
Intercept	5124.931	1	5124.931	660.49	.000
Mood Groups	.590	1	.590	.076	.783
Emotion Regulation Group	407.614	1	407.614	52.532	.000
Mood Groups * Emotion Regulation Group	2.630	1	2.630	.339	.562
Error	845.762	109	7.759		
Total	6139.000	113			
Corrected Total	1253.611	112			

Note. R Squared = .325 (Adjusted R Squared = .307)

Because the mood-induction procedure's small effect size may have contributed to the non-significant ANOVA interaction, additional analyses using linear regressions were conducted, with self-reported mood as a predictor in place of mood-induction groups. In order to use participants' self-reported mood ratings as a predictor variable, and not mood-induction groups, a regression was required instead of ANOVA because participants' mood is a continuous rather

than categorical variable. As in the ANOVA, the interaction of mood and emotion regulation motives for texting did not predict individuals' desire to text, β = -.005, $t(158)$ = .228, p = .820 (one-tailed). Thus, even when considering participants' self-reported mood as a predictor instead of their randomly assigned mood-induction groups, the interaction between mood and emotion regulation motives did not predict desire to text.

Table 2: Regression for Self-Reported Mood, Emotion Regulation Motives and Desire to Text.

Step		B	Std. Error	Beta	t	Sig
1	(Constant)	9.012	1.266		7.116	.000
	Mood	-.518	.256	-.159	-2.020	.045
2	(Constant)	2.644	1.412		1.872	.063
	Mood	-.021	.237	-.007	-.090	.929
	Texting Motives	.199	0.03	.527	7.162	.000
	Mood x Texting Motives	.005	.020	.017	.228	.820

Note. Dependent Variable: Desire To Text. Mood = Self-Reported Post-Induction Mood. Texting Motives = Emotion Regulation Motives for Texting. Mood x Texting Motives = Interaction Between Mood and Emotion Regulation Motives for Texting.

As seen Table 2 however, regression analyses did indicate that emotion regulation motives for texting possibly mediated the relationship between participants' mood and their desire to text. Step 1 of the regression analysis showed that mood significantly predicted desire to text, β = -.518, $t(158)$ = -2.02, p < .05 (one-tailed). Step 2 of the regression analysis indicated that when emotion regulation motives were taken into account, mood was no longer a significant predictor, whereas emotion-regulation motives for texting was a significant predictor, β = 3.72, $t(110)$ = 6.94, p<.01 (one-tailed). This is a pattern of regression results that is consistent with a mediating effect of the emotion regulation motive variable. Thus, the regression analyses may not have supported Hypothesis 2 concerning a moderating effect of emotion regulation motives, but these results did suggest that emotion regulation motives for texting possibly mediated the relationship between mood and desire to text (see Table 2).

Correlations between Demographics and Study Variables

Correlational analyses were conducted to explore if relationships existed between the study variables (emotion-regulation motives, emotion regulation skills, post-induction desire to text, and texting-related problems), demographic factors (gender and age) and texting capabilities (frequency of texting and having a cell phone plan with unlimited text-messaging). The purpose of these analyses was to assess whether such relationships between study variables and other factors could be responsible for hypothesized relationships among study variables.

With respect to demographics and texting capabilities, several significant relationships were found with other study variables. Gender was correlated with emotion-regulation motives for texting. Specifically, being female was correlated with greater emotion regulation motives for texting r = .158, n= 159, p< .05 (two-tailed). Age was negatively correlated with frequency of

text-use *r* = -.385, n= 110, p< .001 (two-tailed), emotion regulation motives for texting *r* = -.448, n= 122, p< .001 (two-tailed), texting-related problems *r* = -.366, n= 122, p< .001 (two-tailed), difficulties in emotion regulation *r* = -.200, n= 122, p< .05 (two-tailed) and desire to text post-induction *r* = -.349, n= 122, p< .001 (two-tailed). Age was also positively correlated with mood post mood-induction *r* = .314, n= 122, p< .001 (two-tailed). More specifically, older participants reported less frequent texting, less emotion regulation motives for texting, less texting-related problems, had a more positive mood after the mood induction procedure, less difficulties in emotion regulation, and less desire to text after the mood induction procedure.

Hypothesis 3: Correlation between Emotion Regulation Motives for Texting and Texting-Related Problems

Table 3: Correlations among study variables.

Variables	PTI	RETS	Mood Rating	Desire to Text
RETS	.529**			
Mood Rating	-.266**	-.298**		
Desire to Text	.591**	.524**	.159*	
DERS	.300**	.367**	-.640**	.282**

Note. *p < .05. **p < .01. PTI- Texting-Related Problems. RETS- Emotion Regulation Motives for Texting. DERS- Difficulties in Emotion Regulation.

To address whether emotion regulation motives for texting (RETS) would be positively correlated with texting-related problems (PTI) as predicted by Hypothesis 3, a Pearson product-moment correlation coefficient was computed. There was a positive correlation between the two variables, *r* = .53, n= 159, p< .001 (two-tailed). See Table 3 for the correlation table. Additionally, based on the significant correlations between age and both emotion regulation motives for texting as well as texting-related problems, a partial correlation was conducted by controlling for age to rule out the possibility that age could be affecting the relationship between emotion regulation motives for texting and texting-related problems. There remained a positive correlation between emotion regulation motives for texting and texting related problems even after controlling for age, *r* = .49, n= 122, p< .001 (two-tailed). Overall, the third hypothesis was supported and higher emotion regulation motives for texting were correlated with more texting-related problems.

Hypothesis 4: Correlation between Difficulties in Emotion Regulation and Emotion Regulation Motives for Texting

To address the question regarding if greater difficulties in emotion regulation (DERS) would be positively correlated with emotion regulation motives for texting (RETS) as predicted by Hypothesis 4, a Pearson product-moment correlation coefficient was computed. There was a positive correlation between the two variables, r = -.37, n= 159, p< .001 (two-tailed). Additionally, based on the significant correlations between age and both difficulties in emotion regulation as well as emotion regulation motives, a partial correlation was conducted by controlling for age to rule out the possibility that age could be affecting the relationship between difficulties in emotion regulation and emotion regulation motives for texting. There remained a positive correlation between difficulties in emotion regulation and emotion regulation motives

for texting even after controlling for age, *r* = .35, n= 122, p< .001 (two-tailed). Overall the fourth hypothesis was supported and greater difficulties in emotion regulation were significantly correlated with greater emotion-regulation motives for texting. See Table 3 for the correlation table.

Hypothesis 5: Correlation between Difficulties in Emotion Regulation and Texting-Related Problems

To address whether difficulties in emotion regulation (DERS) would be correlated with texting-related problems (PTI) as predicted by Hypothesis 5, a Pearson product-moment correlation coefficient was computed. There was a positive correlation between the two variables, r = .301, n= 160, p< .001 (two-tailed). Additionally, based on the significant correlations between age and both difficulties in emotion regulation as well as texting-related problems, a partial correlation was conducted by controlling for age to rule out the possibility that age could be affecting the relationship between difficulties in emotion regulation and texting-related problems. There remained a positive correlation between emotion regulation motives for texting and texting related problems even after controlling for age, *r* = .28, n= 122, p< .01 (two-tailed). Overall, the fifth hypothesis was supported and greater difficulties in emotion regulation were significantly correlated with greater texting-related problems as predicted by Hypothesis 5.

DISCUSSION

The primary research question of the current study concerned whether individuals text when they experience a negative mood in an effort to regulate and improve their emotional experience. Additional research questions concerned whether individuals with greater emotion regulation motives for texting would a) experience a greater desire to text when in a negative mood, b) have greater difficulties with emotion regulation overall (not just limited to texting); and c) report greater texting-related problems. An additional question concerned whether overall difficulties in emotion regulation are related to texting-related problems.

Regarding the primary research question, the current study provided some evidence that individuals do text to regulate their emotions when they experience a negative mood. Although the experimental manipulation of mood did not show an effect of mood on desire to text, correlational findings were consistent with such an effect. There were significant correlations between negative mood and a desire to text as well as emotion regulation motives for texting and desire to text. Regarding the secondary research questions, emotion regulation motives for texting were not associated with greater desire to text when in a negative mood, but were associated with greater texting-related problems, and greater difficulties in emotion regulation. Lastly, greater difficulties with emotion regulation were found to be associated with greater texting-related problems.

Thus, in summary three of the five study hypotheses were fully supported, and the remaining two were potentially supported.

Limitations

This study attempted to go beyond being correlational with the use of an experimental design that included a mood induction procedure. This was done to help infer a potential causal

relationship between individuals' mood and their desire to text. The success of the current study's experimental design, and more specifically Hypotheses 1 and 2, was contingent upon the mood-induction procedure effectively creating a more negative mood amongst participants randomly assigned to a negative mood-induction group compared to participants randomly assigned to a neutral mood-induction group. Although the mood induction procedure produced a statistically significant effect, the effect size was small, which could have caused Hypotheses 1 and 2 to not be supported. Nevertheless, the study hypotheses were supported only by correlational, not experimental, results. Thus, a primary limitation of this study is the correlational nature of the results, which leaves open the possibility of third variables being responsible for the correlations.

A look at participants' responses to an open-ended question designed to produce either a negative or neutral mood may further illuminate the insufficient mood-altering effect of the mood induction procedure. Participants were asked to recall either a previous stressful situation (negative mood induction) or the details of a typical day (neutral mood induction). A review of participants open-ended responses provided some clues as to why the mood-induction procedure may not have had a strong enough effect to influence mood. Several participants' responses showed that despite recalling events that may have caused significant distress such a betrayals, trauma, loss, or heartbreak, they still reported being in a positive mood currently.

Future Research

The current study is one of the first known studies to examine if individuals use texting as a means to regulate upsetting emotions, as well as the outcome for such behavior. It represented an exploratory look into a field of research with limited existing literature. Most research involving texting is limited to texting while driving or excessive text use. Only one study by Feldman and associates (2011) was found that mentioned the possibility of individuals texting to regulate emotions, but its primary focus was still texting while driving. The fact that all five of the current study's hypotheses were either fully supported or potentially supported created several areas for future research in a field that is still relatively new. Findings from the current study were promising enough, and consistent with findings in other fields of research such as substance abuse and emotion regulation, that it seems likely that a program of study could be developed related to motives for texting, the costs and benefits of each motive, and the outcomes associated with particular texting-styles.

As previously stated, texting to regulate emotions could take place due to two occurrences—distraction and seeking emotional support. It could be possible that some forms of texting to regulate emotions, such as seeking emotional support, could be more beneficial than other forms of texting to regulate emotions. Thus, future research should explore the possibility that adaptive coping could result from texting to regulate emotions, and determine which forms of texting to regulate emotions are the most beneficial. Implications of such research would include the ability to discourage some forms of texting which were shown to result in negative consequences while encouraging forms of texting for emotion regulation that proved adaptive and therapeutic.

Future research may also benefit from exploring more recent forms of instant messaging communication made available to individuals through mobile versions of social networking

services such as Facebook, Twitter, and Instagram. Despite texting being a relatively new in its on regard, mobile applications of social networking sites are even more current, with many being developed and becoming popular over the course of the last couple of years. Mobile versions of social networking services allow the same access and capabilities available through the web-based computer versions while on the go with the use of a smart phone or tablet computer. Thus, the all access capabilities of these mobile social networking sites would allow them the same opportunity to provide emotion regulation as the current study suggested of texting.

Additionally, due to the interactive and open-access nature of mobile social networking sites, they provide a more elaborate and group-oriented communication experience than texting. Examples include the ability to share videos, pictures, stories, quotes, link to other information sources, group interaction, and availability of a third party audiences. These additional elements unique to mobile social networking sites would likely uniquely contribute to their effect on mood and emotion regulation. More specifically, these capabilities could mean even more opportunities at emotion regulation.

Further determining the relationship between texting, emotion regulation, and mood could represent the initial establishment of a theoretical framework for an untapped area of research related to people's use of instant messaging technology for not only social purposes, but as means to achieve some sort of psychological and emotional end as well. Once established, then other associated areas such as mobile social networking sites could be explored and may provide even more information about how people may rely on instant messaging technology to psychologically cope as we as the outcomes of such behavior.

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CASE REPORT

Bronchoalveolar Carcinoma: The Radiologic Masquerader

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A CASE REPORT

A 39 year old African American woman with no significant past medical or surgical history presented to Jackson Park Medical Center complaining of shortness of breath and productive cough of 4 months duration. Until 2013, she had smoked one cigarette monthly (0.04 pack years). Over-the-counter medications did not provide any relief of her symptoms. Vital signs were within normal limits and physical examination revealed decreased breath sounds in the right lung. Lab findings were insignificant except for notable eosinophilia (22.6%). A diagnosis of community acquired pneumonia (CAP) was established and the patient was discharged with an Albuterol inhaler q6hr and Clarithromycin.

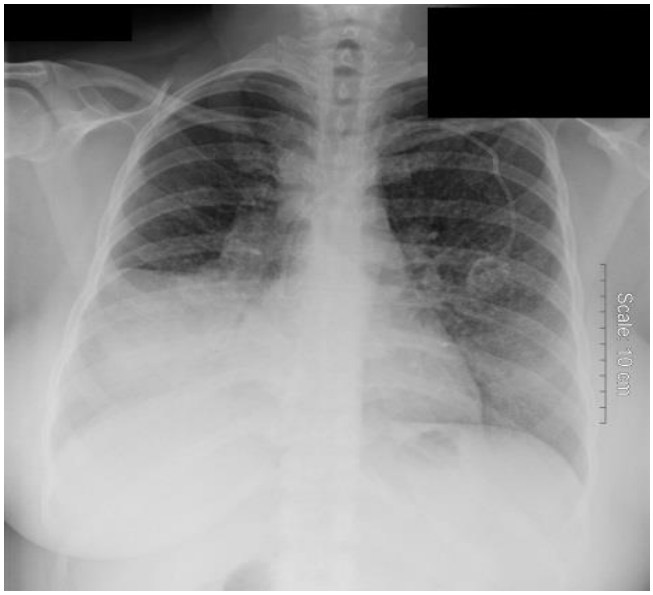

Figure 1. Initial X-ray showing consolidation

The patient presented two weeks after completion of her antibiotics without improvement. A chest x-ray was ordered and findings included a complete consolidation of the right middle lobe and diffuse bilateral patchy opacities and micro-nodules **(Figure 1)**. She was prescribed a course of Augmentin due to the high index of suspicion for resistant CAP.

When the patient returned two weeks later, there was still no improvement in her symptoms and no changes in repeat chest x-ray were appreciated. The patient was admitted to Jackson Park Hospital and a CT scan of the chest was conducted **(Figure 2)**. Bulky mediastinal and right hilar lymphadenopathy were noted as per the radiology report but it could not be clearly differentiated from a mass. While the findings of consolidation, diffuse bilateral patches of opacities and micro-nodules strongly reflected an inflammatory process, bronchoalveolar carcinoma and lymphoma could not be ruled out. Sputum cultures were negative and a lung biopsy was conducted to ascertain the existence of underlying malignancy. Findings were confirmatory for bronchoalveolar adenocarcinoma.

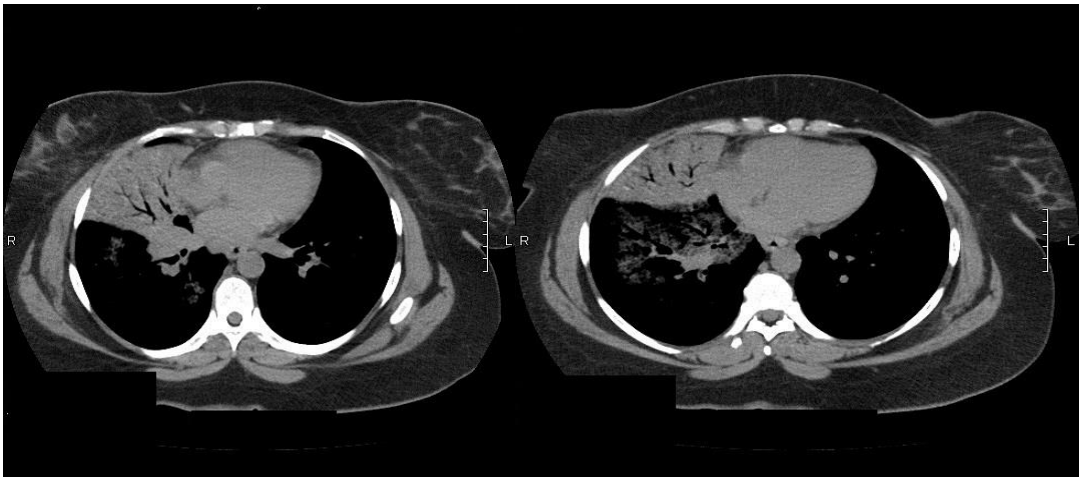

Figure 2. CT scan demonstrating mediastinal and right hilar masses and persistent complete consolidation of the right middle lobe. Note diffuse bilateral patchy opacities and micronodules.

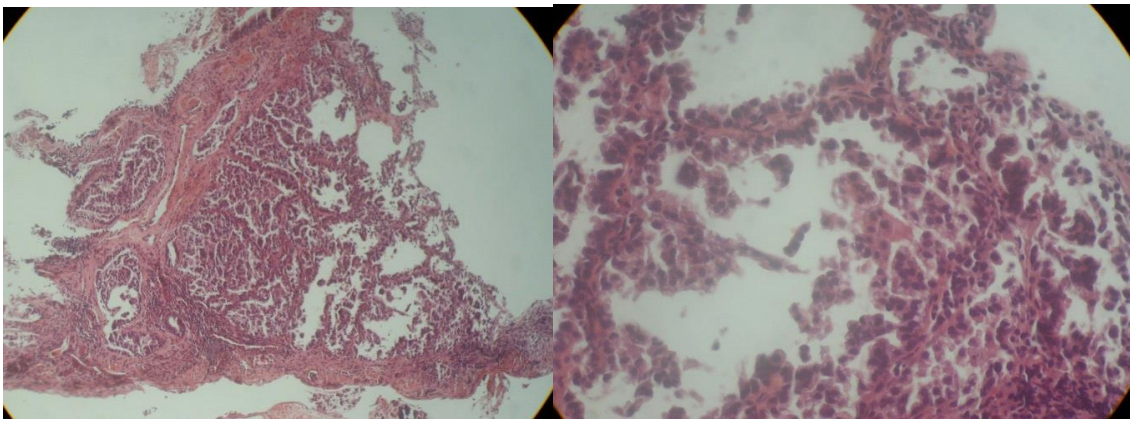

Figure 3. Biopsy confirming bronchoalveolar adenocarcinoma.

DISCUSSION

Bronchoalveolar carcinoma (BAC) is a very rare form of adenocarcinoma. Although mixed forms make classification difficult and comprise up to 20% of all lung cancers[1], it represents less than 4% of all non-small cell carcinomas (NSCC) in its "pure" form.[2] BAC often takes an indolent course and is commonly becoming referred to as adenocarcinoma in situ (AIS)[3] because of its characteristic lack of invasiveness. It is also the least associated with smoking and most favorable in terms of prognosis relative to the other NSCC's.[4] BAC arises in the peripheries of the lung and grows alongside the alveolar walls ("lepidic" growth pattern) without destroying the lung parenchyma. BAC may arise simultaneously in multiple lobes and typically presents bilaterally. Chest radiograph and symptoms of cough, chest pain, and sputum production make the diagnosis indistinguishable from pneumonia or other noninfectious inflammatory processes (e.g., hypersensitivity pneumonitis or bronchiolitis obliterans).[5]

There should be suspicion of BAC in diagnoses of pneumonia refractory to antibiotics and the lack of fever or leukocytosis. The cure for BAC is a complete resection via pneumonectomy or lobectomy.[1] A trial of antibiotics and reassessment of clinical findings is a reasonable approach, but biopsy or cytology is the only means of diagnosing BAC. Thus a biopsy should always be considered when pneumonia does not respond to antibiotics.[1]

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CASE REPORT

Rare Dermatologic Abscess Presenting as Malignant Tumor of Scrotum

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A CASE REPORT

A 64 year old African American male with a history of hypertension and above the knee amputation of the left lower limb (secondary to gangrene) presented to the emergency department complaining of a non-healing ulcer on his right scrotum for the past three weeks. Four months ago, patient reported undergoing surgical removal of a hard nodule at this site. He was admitted to Jackson Park Hospital shortly thereafter for a cyst that had emerged at the surgical site. The cyst ruptured during the hospital stay with purulent and foul smelling discharge.

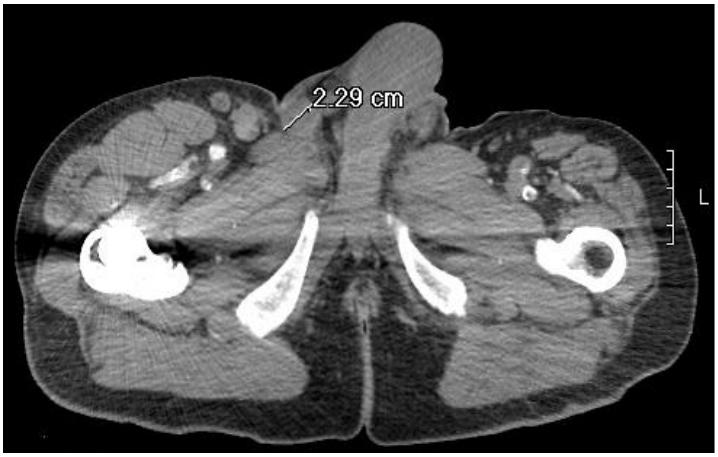

Figure 1. CT Pelvis with prominent lymph node in the right scrotum measuring 2.29 cm

Laboratory findings during this visit were insignificant and the patient was placed on a three week course of antibiotics. Despite therapy, the patient did not improve. CT scans of the abdomen and pelvis were obtained and findings included bulky retroperitoneal lymphadenopathy and right inguinal and scrotal lymphadeno-pathy. As per the radiology report, this was presumably due to nodal metastatic disease **(Figure 1, 2)**. It also allowed for visualization of a complete occlusion of the left common femoral and superficial femoral arteries with high grade stenosis of the left external iliac artery. Ultrasound of the scrotum (**Figure 3)** visualized bilateral hydroceles (greater on the left) without evidence of torsion or mass lesion. A biopsy of the scrotal ulcer was then conducted and the pathology report indicated invasive moderately differentiated squamous cell carcinoma.

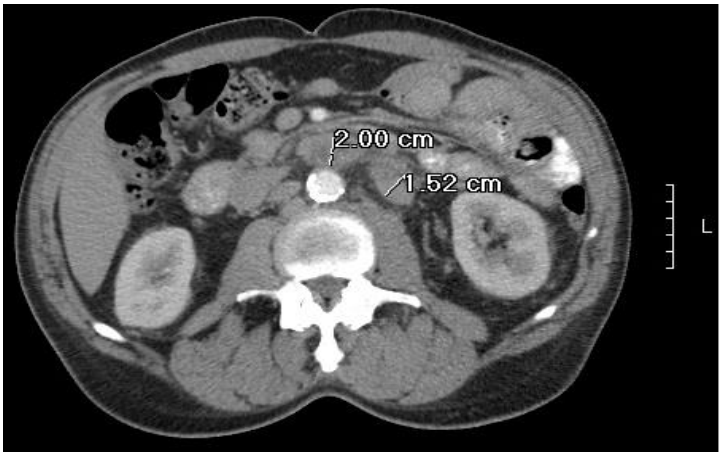

Figure 2. CT pelvis with prominent retroperitoneal lymphadenopathy; the largest nodal masses in the peri-aortic region measuring 2 cm and 1.5 cm

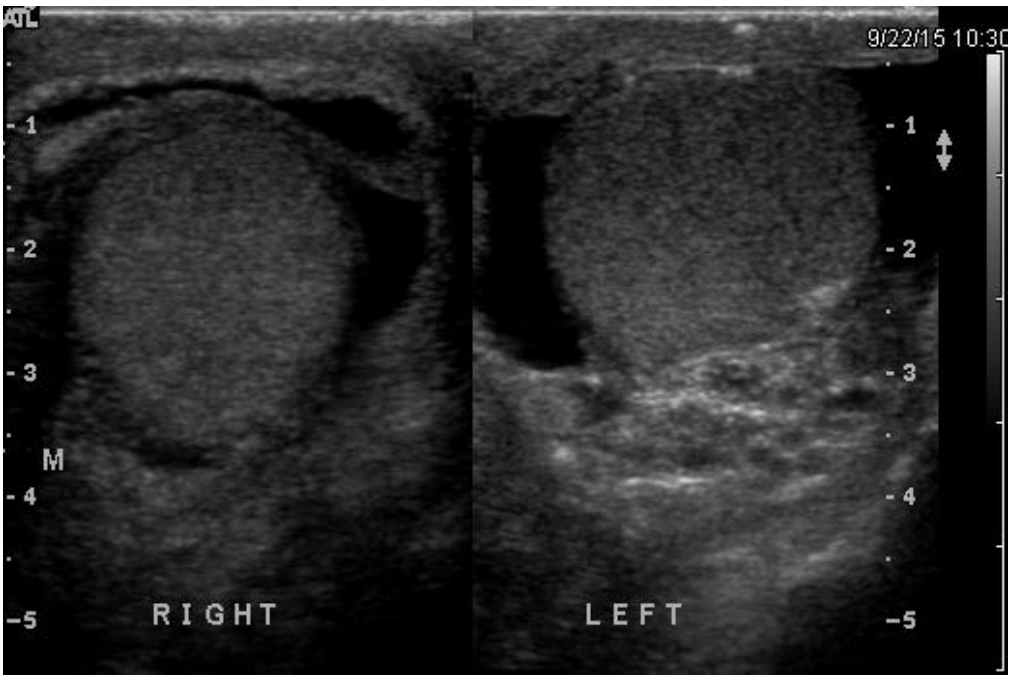

Figure 3: Ultrasound of scrotum indicating bilateral hydrocele, left greater than right.

DISCUSSION

Squamous cell carcinoma (SCC) of the scrotum was the first ever reported malignancy related to occupational exposure[1] when a high incidence was noted in the 1970's among chimney sweepers. Since then, other occupations have been associated with an increased incidence of scrotal carcinoma including paraffin or shale oil workers, mule (cotton) spinners, machine operators in engineering, petroleum wax pressman, workers in the screw-making industry, and automatic lathe operators.[1]

Cases have been reported most often among men in their fifth and sixth decades as well as individuals with poor hygiene. Patients typically present with a solitary, painless, and slow-growing skin lesion.[1] Ulceration may follow with an increase in lesion size, and the area can become infected. Scrotal SCC is usually limited to one side of the scrotal sac, with predilection to the anterior inferior surface of the scrotum.[2] Approximately 50% to 75% of patients have enlarged inguinal lymph nodes at time of initial presentation.[2]

A decline in culprit industries and increased awareness of the necessity for good hygiene and preventive work attire have caused this malignant neoplasm to become quite rare today (less than 10 cases per year in the US),[1] yet new potential etiologies continue to emerge. P and long-wave ultraviolet radiation (PUVA), a treatment for psoriasis, has recently been shown to increase the incidence of both penile and scrotal malignancies.[3] Human papilloma viruses, particularly types 16 and 18, have also been associated with the development of genital SCC.[4]

Patients often delay in seeking treatment due to the gradual progression of the growth and/or general embarrassment relating to the privacy of the affected organ. A good prognosis relies heavily upon local confinement and thus it becomes critical to include scrotal SCC as a

differential diagnosis in patients that present with non-healing, recurrent scrotal abscesses that are refractory to antibiotic regimens. [5] Inguinal node involvement decreases 5 year survival to 25% and iliac node involvement decreases it completely to 0%.[6] Treatments in patients without lymph node involvement consists of wide excision of the primary tumor. Additionally, there is evidence that indicates patients with metastasis to the lymphatics may be treated and cured with combination radiation and chemotherapy.[1]

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CASE MANAGEMENT STRATEGIES

Guidelines for the immediate and acute management of aneurysmal subarachnoid hemorrhage prior to relocation to a specialized neurosurgery facility

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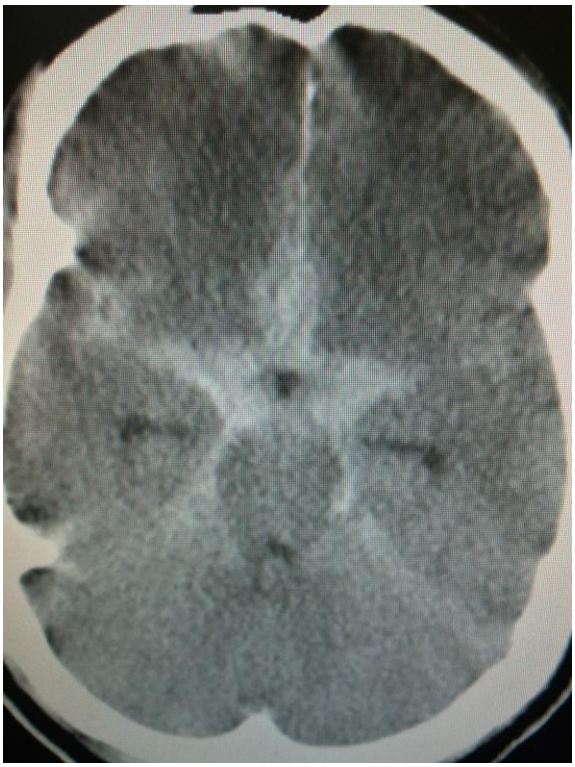
A CASE REPORT

A 53 year old African American male with a past medical history of hypertension presents to the emergency department (ED) complaining of dizziness. He is anxious to find out what is going on and how you can "make this dizziness go away." After further investigation, you come to the conclusion that this patient suffers from labyrinthitis and prescribe Meclizine, fully confident that it will take care of the problem. Two days later, the patient presents again to the ED with the "worst headache of my life". CT scan without contrast reveals the following. What is the most likely diagnosis?



A) Renal failure
B) Subarachnoid hemorrhage
C) Epidural hemorrhages
D) Normal pressure hydrocephalus
E) None of the above

INTRODUCTION

Background

Aneurysmal subarachnoid hemorrhage (aSAH) is predominantly caused by the rupture of an intracranial saccular aneurysm resulting in a spontaneous bleed into the subarachnoid space—the area between the pia and the arachnoid membranes of the brain. It is important to examine management options for aSAH as approximately 1 in 4 patients will expire and half of survivors will suffer from a neurological deficit.[1] In the United States, the incidence of aSAH is estimated

to be 9.7 per 100,000 persons.[2] Although, these values may be an underestimation of incidence because many patients die before hospital admission.[1]

Presentation

The characteristic presentation of an alert and oriented patient complaining of "the worst headache of my life" is among the most easily identified clinical presentations in medicine.[3] This headache, known as a thunderclap headache, has an extremely rapid onset and quickly reaches maximal intensity.[4] The meningeal irritation from the bleeding can explain the symptoms associated with aSAH (neck stiffness, photophobia, low back pain, nausea, vomiting and double vision).[5]

Diagnosis

A prompt and accurate diagnosis is critical in improving prognosis and decreasing potential permanent neurological damage or death. Non-contrast head CT is currently the gold standard for diagnosing aSAH.[1] If non-contrast CT is done within the first 12 hours of onset, the sensitivity is 98-100% although this decreases with time.[6] Additionally, lumbar puncture is widely used as a diagnostic tool and often reveals xanthochromia, a yellowish tinge to the cerebrospinal fluid that reflects bilirubin accumulation secondary to blood breakdown in the subarachnoid space. Technological advancements in magnetic resonance imaging of the brain provide an effective alternative if suspicion is high yet a negative CT scan may eliminate the need for lumbar puncture and its associated complications. The high cost and limited availability precludes institutions with limited neurological specialization from offering this approach to patients. The Hunt and Hess scale can be used to determine the severity of aSAH **(Table 1)**.

Table 1. Hunt and Hess Scale[11]

Grade	Description
1	Asymptomatic, or mild headache, slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, cranial nerve palsy may appear
3	Drowsiness and/or confusion, appearance of other focal neurological deficits
4	Stupor, moderate to severe hemiparesis
5	Coma, decerebrate posturing

MANAGEMENT

This following management regime is indicated for a patient that is diagnosed with aSAH in a facility with limited neurosurgical resources where aneurysmal securement is not possible. These procedures highlight steps that are intended to increase patient prognosis before transfer to an expert care facility. The transition period between non-specialized and specialized facilities should be kept to an absolute minimum.

Upon arrival, patient stabilization is the first priority. Securing the airway via intubation and mechanical ventilation is indicated for patients with a compromised airway such as those with altered mental status, lung injury, aspiration, or a Glasgow Coma Scale of less than 8.[5] Oxygenation and ventilation levels must be closely monitored to ensure adequate equilibrium with intracranial and cerebral perfusion pressures.[5]

Blood pressure control is necessary to prevent severe complications such as re-bleeding and vasospasm. Focus should be directed at maintaining a mean arterial pressure (MAP) of 70-130 mm Hg and a euvolemic state.[8] Patients with MAP >130 mm Hg should be treated with an initial bolus followed by a titrated IV drip to achieve optimum blood pressure. Sublingual hypertensive agents are contraindicated due to their sudden decrease in blood pressure and consequent cerebral ischemia.[5] A febrile state may hinder recovery. Patients with a body temperature of >38.3 degrees Celsius should be given Acetaminophen every 4-6 hours until fever resolves. If pharmacological methods fail, surface or intravascular temperature management techniques may be therapeutic.[5]

A basic metabolic panel, chemistry panel, cardiac enzymes, complete blood count and urine analysis should be performed to obtain baseline data as there is a risk for electrolyte, cardiac, pulmonary and fluid imbalances.[5] An arterial blood gas is necessary for all patients, especially if intubated, and a 12-lead EKG and chest X-ray are also to be done upon admission.[5] Because patients will most likely be sent out to a specialization clinic for surgery or coiling, oral medications, fluids and food should be avoided. To reduce morbidity and mortality associated with hyperglycemia, serum glucose should be kept within 80-120 mg/dl with insulin infusion. [5] Strict bedrest is necessary as changes in position can increase intracranial pressure (ICP). Due to lack of activity, DVT prophylaxis must be started using only stockings and sequential compression devices. Patient should be maintained in a peaceful and low volume environment[9] and it is important to keep patient informed by explaining procedures, answering any questions, and providing the highest level of comfort possible.

Medication

Antiplatelet medication and anti-thrombolytics are contraindicated. Nimodipine or Nicardipine can be used at the physician's discretion. Nimodipine is currently the only FDA approved medication to prevent vasospasm,[5] however, this evidence is controversial as other studies suggest it has a functional outcome and direct neuroprotective effects but no clear correlation with angiographic vasospasm.[10] Conversely, Nicardipine did not appear to improve functional outcome but was effective in controlling vasospasm.[10] To avoid increases in intracranial pressure (ICP), patients should be advised against straining during bowel movements and, if necessary, prescribed stool softeners. The characteristic headache prompts pain management with short acting and reversible analgesics to prevent pain-induced increases in heart rate, blood pressure, and anxiety. Short acting sedatives may be used as needed in agitated patients; however, neurological exams should be conducted when patients are not under the influence of sedatives. Vomiting can increase ICP and risk of re-bleed, therefore nauseous patients should receive anti-emetics. Elevated ICP can cause gastric ulcers or gastrointestinal hemorrhage so anti-histamines or proton pump inhibitors should be administered prophylactically.

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RESEARCH RECOMMENDATIONS

- **Sepsis research:**
  - Studies show that appropriate treatment within one hour has many benefits in sepsis. Determine exactly when antibiotics were initiated. Next, compare the progression of the disease and analyze how delay in appropriate treatment can increase mortality from sepsis.
  - Determine if IV fluids were initiated within 6 hours of arrival and analyze if this intervention decreases risk of developing shock in the African American population.
  - Source control and antimicrobial therapy: Observe routine practices for sepsis control, review policies and procedures and develop collaborative project (with nursing staff, ID specialists, other bedside clinicians and other associated departments) on best management practices for septic shock prevention or management
  - Develop potential molecular tools for determining therapeutic response to intervention therapy against multidrug resistant organisms. This approach may be useful in selecting accurate and adequate gram positive, gram negative or polymicrobial coverage in the management of septic shock

- **Pathophysiology of histamine release with cocaine and heroin use and its association with asthma exacerbation**

- **Disparities existing in proper diagnosis and treatment of metabolic encephalopathy**

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The editorial board of the *Jackson Park Hospital Journal* welcomes the submission of manuscripts for publication in the journal. When preparing your article, please follow the guidelines below:

The text should be single-spaced, in a letter quality font. The manuscript must be submitted by one or more of the authors of the original article. Online submission is preferred, and may be emailed to journalJPH@gmail.com. The following file formats are required for the main manuscript document: Microsoft word (DOC, DOCX).

Sources must be properly cited and referenced in the American Medical Association (AMA) format. Please see articles in this journal for examples on formatting.

Research Articles

- The order of presentation should include but not limited to title, abstract, introduction, methods, results and discussion, illustrations and figures, conclusions, and references.
- Tables and figures should have a title and be numbered. If the table or figure has been published previously, include the complete reference as well as a letter granting permission from the previous publisher.

Case reports

- Authors are encouraged to submit case reports on cases from Jackson Park Hospital, in any specialty. Along with the case presentation, it is advisable to include a discussion on the disease process.

Authors may submit any other forms of articles, pictures, miscellaneous pieces that they believe will contribute to the *Jackson Park Hospital Journal*.

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