

N311 Care Plan 1

Nick Alford

Lakeview College of Nursing

N311: Foundations of Professional Practice

Kristal Henry

9/26/2024

Demographics (5 points)

Date of Admission 9/16/24	Client Initials N.H.	Age 77	Gender Male
Race/Ethnicity Caucasian	Occupation CTA (is currently unemployed)	Marital Status Married	Allergies Codeine, Iodine
Code Status Full	Height 6'	Weight 251 Lb 1.6 oz	

Medical History (5 Points)

Past Medical History: Acute Myeloblastic Leukemia, Cardiomyopathy, CKD (Chronic Kidney Disease)/AKI (Acute Kidney Injury), Left-Sided Systolic & Diastolic Heart Failure, CAD (Coronary Artery Disease), DM (Diabetes Mellitus), GERD (Gastroesophageal Reflux Disease), episodes of Hypo & Hypernatremia, Iron Deficiency Anemia, NSVT (Non Sustained Ventricular Tachycardia), Protein-Calorie Malnutrition, SBO (Small Bowel Obstruction), Restless Leg Syndrome, Stroke.

Past Surgical History: Appendectomy, Tonsillectomy, Cardiac Defibrillator Placement, PERC (Percutaneous Coronary Intervention). Unspecified removal of mass from left thoracic cavity (per patient report, no associated findings in chart)

Family History: Paternal Grandmother – Diabetes; Father – Heart Attack; No known issues with other immediate family (siblings/mother/other).

Social History (tobacco/alcohol/drugs including frequency, quantity and duration of use):
Denies use of any/all.

Admission Assessment

Chief Complaint (2 points): Worsening SOB (Shortness of Breath) >1 week, with significant increase x2-3 days prior to admission.

History of Present Illness – OLD CARTS (10 points):

Patient reports issues began approximately 2 weeks ago, with progressive worsening, and that by day 7 it became severe enough he reported to the ED. Has now been hospitalized approximately 1 week; discharging today. Patient notes that the issue feels localized to his chest but endorses some swelling in distal extremities too. While the current presentation is associated with acute changes, patient notes underlying issues have been in play for some time now. His diagnosis of CHF is not new, but he denies prior hospitalizations for complications with breathing before this episode. He notes that the chief issue is significant chest pressure that feels resistive to deep breathing that leaves him progressively short of breath, with tachypnea that fails to keep up with his oxygen hunger. Further relates that he cannot tolerate supine positions as it disrupts his ability to breathe sufficiently. Without supplemental oxygen he becomes increasingly short of breath, taking more shallow breaths to try and compensate, and gets resulting anxiety. Also details a chronic wound on left lateral trunk that influences level of discomfort with positions or deep breathing, further contributing to his issues. He states the wound was a result of a reported 12.5 Lb mass removed from the area in 2021 which reopened in September of last year. He has been on antibiotics ever since, and the wound persists. Notes that “whatever meds they are giving me” and supplemental oxygen do help alleviate dyspnea, as does continuing to remain sitting upright in bed to ease breathing. He additionally reports that supplemental oxygen and the care he is receiving are assisting, endorsing significant improvement since he first arrived at the hospital. Exertion continues to be when shortness of breath is most pronounced, as anything increasing oxygen demand is a struggle for him. Patient relates that at the time he arrived at the ED he would have rated the severity of his issues at a 9/10 due to significant struggle to breathe, but that he now feels much better, 3/10 severity.

Primary Diagnosis

Primary Diagnosis on Admission (3 points): Acute on chronic left sided systolic heart failure

Secondary Diagnosis (if applicable): AFIB (Atrial Fibrillation), DM (Diabetes Mellitus), OSA (Obstructive Sleep Apnea)

Pathophysiology

Pathophysiology of the Disease, APA format (20 points):

The diagnosis of Congestive Heart Failure is a serious condition but is often misrepresented by the emphasis placed on the heart itself. In truth, the condition is significantly more systemic than simply a failing of the heart. When looking at the disorder from a whole-of-body perspective, and taking into account the patients entire chronology of preexisting complications, we often find that the involvement of the heart is actually a physiological change that is several rungs along on a ladder of deteriorating conditions in the body that was preceded by a number of long standing factors. Its name then, rather, is a signpost for how severe these changes have become.

Congestive Heart Failure, or CHF for short, is most often a result of other cardiac complications, which in turn may also owe their existence to the failings of other systems, or chronic effects of poor lifestyle choices related to diet and activity. Lack of activity (sedentary behavior), as well as high sodium and sugar diets comprised heavily of processed foods can have a systemically detrimental effect on the body leading to significant impacts on fluid balance, vessel health, and kidney performance. These dietary and activity choices can also produce additional contributing factors to CHF such as Diabetes Mellitus, Hypertension, and Obesity. The sum of which, if left chronically unmanaged will create changes to the bodies “plumbing” that leads to narrowing and hardening of vessels, increased fluid within these vessels with an

increased particle content, and deposition of these sediments throughout vessels that create additional peripheral resistance to blood flow in the form of plaque obstructions that may become complete blockages. In this way we arrive at the use of the term “congestive”, as this metaphorical plumbing becomes increasingly clogged with resistance to the necessary flow of bodily fluids throughout the blood vessels from any or all of these factors, resulting in increased pressure on the primary pumping activity of the heart itself. This is commonly referred to as “Volume Overload” and is the primary instigator of CHF. So severe does this overload become, that part of CHF’s character presentation is a swelling of the extremities from the fluid itself overwhelming the blood vessels, and like a high-pressure pipe spraying at its seams, we see what is known as “third space” edema, where the fluid begins to collect in distal extremities as it spills over interstitially. The result is the unmistakable peripheral edema common with CHF.

On a cellular level, this increased pressure drives stress related changes to the anatomical structure and physiological function of the heart. In response to this increasingly demanding work environment, the heart’s muscle cells- known as myocytes, begin to enlarge. This leads to hypertrophy of the heart, or cardiomegaly. However, long-term this will not be enough, and is largely just a coping effort. The chronic nature of this pressure will lead to chronic inflammation and stress to the underlying tissues, which will result in an eventual cascade of exhaustive effects on the heart – myocytes will die, calcium that fuels the pumping action becomes dysregulated, pumping strength & rhythm will deteriorate, and the overloaded plumbing will be further exacerbated by the pump itself now failing to compensate for the systemic issues at play.

It is at this point in disease progression that we have seen how some systems failings lead to the heart’s failings, and now we will begin to see how the heart’s failings will in turn lead to other systems’ failure. The heart and the lungs are intrinsically entangled, and where one's

primary objective fails, so too will the others. This cascading of failures builds up cumulatively and leads to ever increasing internal pressure within the vessels of the heart and lungs, leading to an increasing static fluid pressure accumulation, oversaturation of tissues from fluid imbalance, and additional cellular and tissue level changes. These changes will lead to pulmonary impairments such as loss of tissue elasticity or bronchial accommodation, fluid accumulation (blood/other), and impaired gas exchange in the lungs- all further diminishing the body's ability to oxygenate adequately when under peaks of congestive buildup. (Cross et al., 2020)

As a result of these ongoing maladaptive attempts to address the systemic failings of the body to compensate for its deteriorating conditions, we see the onset of dyspnea with CHF. The impaired lung function, coupled with the pressure of the volume overload discussed earlier, leads to significant and life-threatening complications with patient respiratory function. This is what is generally called a “CHF exacerbation” and is what typically motivates Pt’s back to an emergency room. Dyspnea from cumulative volume overload begins to effectively suffocate a patient, which leaves them seeking emergency care.

Once in medical care, diagnostics are performed to confirm and assess the patient's CHF. Imaging procedures of all kinds can visualize the heart and its function, but the echocardiogram remains the most typical. Labs can be drawn to assess the function of underlying fluid balance systems such as analyzing electrolytes to determine kidney involvement, or even scan for natriuretic peptides suggestive of heart stress. Functional examinations like a stress test may be ordered to see how the patient's heart responds to exertion. Diagnostic measures are then utilized to determine a level on a CHF classification system to rank the patient's disease status based on severity. Class 1 mild heart failure demonstrates no real impediments to function, whereas class 2 mild heart failure demonstrates the initial onset of low-grade fatigue, dyspnea, activity

intolerance, and heart palpitations. Class 3 reaches moderate heart failure status and will evidence more pronounced versions of Class 2's identified alterations to function. Class 4 is severe heart failure and is when a Pt has reached a level at which they are unable to maintain their normal ADL's without significant discomfort and/or alterations. (Capriotti, 2024)

Treatment in the hospital for CHF centers around management of the fluid volume overload and the underlying physiological functions (or failures) contributing to the buildup. Pt's are placed on a suite of medications- starting with diuretics to try and motivate the body to excrete the accumulated fluid to relieve pressure and alleviate breathing. Cardiac medications like ACE Inhibitors are utilized to improve the impaired heart function contributing to the issue. Anti-hypertensives are utilized to assist with vessel diameter to decrease overall pressure within the "plumbing" and facilitate increased flow. The net effort being to improve the fluid management systemically to restore balance and offload the overwhelmed components involved. However, this effort is largely palliative in nature, as the patient's complexity at this point has been so long-standing and multi-faceted that reversal of the disease is not a realistically attainable goal. Patients of this nature will often rebound in and out of the hospital multiple times with these exacerbation events over the course of the remainder of their lifetime, as their condition ebbs and flows under their maintenance efforts at home. However, age allowing, with diligent adherence to specific dietary, activity, and medication guidelines established by their primary provider- patients can continue to live meaningful lives for years to come. Science continues to explore new treatment options, with new medications still coming onto the market, but CHF remains a maintenance-based disease due to its progressive nature.

Pathophysiology References (2) (APA):

References:

Cross, T. J., Kimm, C.-H., Johnson, B. D., & Lalande, S. (2020, January 1). *The Interactions Between Respiratory and Cardiovascular Systems in Systolic Heart Failure*. *Journal of Applied Physiology - American Journal of Physiology*.

<https://www.physiology.org/doi/10.1152/jappphysiol.00569.2016>

Capriotti, T. (2024). *Davis Advantage for Pathophysiology* (3rd ed.). F. A. Davis Company.

<https://bookshelf.vitalsource.com/books/9781719650533>

Vital Signs, 1 set (5 points) – HIGHLIGHT ALL ABNORMAL VITAL SIGNS

Time	Pulse	B/P	Resp Rate	Temp	Oxygen
1430	120 BPM	117/73	28 RR	97.2 *f	100% SpO2

Pain Assessment, 1 set (5 points)

Time	Scale	Location	Severity	Characteristics	Interventions
1430	0-10 scale	Left Lateral Trunk	4/10	Ache, discomfort	Pt declines Pn meds at this time.