Reflection & Analysis Critique

on a Client with Chronic Illness

Chronic condition can affect any span of a person's life. With improved medical management, decreased mortality rates, and advances in medical technology, people are living longer despite their ailing conditions (Hoffman, Rice, & Sung, 1996). Unlike acute conditions, many people with chronic diseases accrue higher medical costs due to recurrent exacerbations, miss a greater number of workdays, and increase the risk of future disabilities and limitations (Hoffman, Rice, & Sung, 1996). According to Hoffman, Rice, and Sung (1996), in 1995, it was estimated that approximately 100 million Americans had one or more chronic conditions. More specifically, 88% of all older adults lived with at least one chronic condition and 69% of had more than one chronic condition (Hoffman, Rice, & Sung, 1996).

With an increasing prevalence of chronic conditions in the society, it is important to understand and properly manage these individuals and their families. Therefore, the purpose of this paper is to critique a student's clinical encounter for an elderly client with multiple chronic co-morbid conditions seen at Waltonwood Clinic in Canton, Michigan.

Subjective:

JF is a 90 year old white male with a history of COPD, DM, CAD, BPH, atrial fibrillation, HTN, CHF, hyperlipidemia, left eye macular degeneration, and right eye cataract. JF had sharp chest pains three weeks ago while bending down. The pain was relieved by rest. Patient wears a nitropatch during the day. Currently, he does not have any chest pains or had any more chest pains since then. Patient is afraid to bend down in fears that he will experience the chest pains again. JF also complains of increasing SOB in the night and in the morning. This is relieved by getting up and sitting at the side of the bed and using an inhaler. He does not complain of HA, dizziness, dysuria. JF states he uses the bathroom every 1-2 hours. According

to the nursing staff, JF has been fully unable to carry out his ADLs and has increasingly become more incontinent.

Current Medications:

1. Digoxin 0.125mg QOD PO
2. Glipizide 10mg QD PO
3. Zocor 10mg QD PO
4. Diltiazem CD 240mg QD PO
5. Lisinopril 5mg QD PO
6. Avandia 4mg QD PO
7. Furosemide 80mg QD PO
8. Coumadin 5mg QD PO
9. Nitroglycerin patch 0.2mg/hr applied daily and removed at pm
10. Combivent Inhaler 2 puffs q4 hr PRN

Allergies: NKDA
Surgical history: prostatectomy
Social: lives in the independent facility, girlfriend is at the assisted living facility

Objective:

Overall, patient is somewhat an accurate historian, labored breathing, but of smooth speech. JF uses a wheeled walker and has one sock on, shirt not buttoned all the way, and food stains noted on his pants.

1. RR 28, P 88
2. C/V atrial fibrillation, irregularly irregular, no JVD
3. Lungs: wheezes bilaterally throughout, barrel chest
4. GI: positive BS, NT/ND
5. Peripheral: DP +/+, bilateral feet edema +2, gross sensation intact
6. Skin: W/D/I

Assessment:

1. COPD
2. Chest Pain
3. Urinary Incontinence/BPH

Plan:

1. Atrovent 0.02% solution, use one unit does vial 0.5mg/2.5ml via nebulizer TID
2. Albuterol 2.5mg/3ml unit does 0.083% solution one unit dose via nebulizer TID

3. Nitroglycerin SL 0.3mg q 5minutes w/ max of 3 doses for acute angina attacks
4. Labs: UA and C/S, PT/INR, BUN, Creatinine, lytes, Hbg A1C, digoxin level, ALT, AST, lipid panel
5. Begin to review/prepare patient and staff for transfer to assisted living
6. Refer to Urology Associates

Subjective Critique

The information I documented addressed JF's issues as well as the staff's concerns. JF was concerned about his chest pain and his worsening shortness of breath. The staff was concerned about his inability to fully maintain his ADLs at the independent facility. In addition, his incontinence was not only a health issue, but also a social issue. The other residents at the independent facility were complaining about JF's urine odor and the staff was unable to keep up with JF's incontinence hygiene. Although I had received this information, I realized that I did not dictate the information on the physician's progress notes.

In retrospect, there were many questions that I should have asked since it was my first encounter with JF. By living at the independent facility, smoking and drinking are not allowed. Although my nurse practitioner is fully aware of JF's social history, I should have inquired more about his past smoking and drinking patterns. I should have also asked about his family history or at least completed a family genogram.

After a review of the literature, there were series of questions I should have asked. In regards to his chest pain, I needed to fully differentiate the origination of his pain (anginal vs. nonanginal). To do this, I should have determined if the pain he was describing was actually anginal. According to Constant (1983), multiple pain locations could exist. For instance, 50% of patients with angina could also experience musculoskeletal chest pain or costochondritis (Constant, 1983). Additionally, I should have inquired about the *duration* of the chest pain. Angina pain can last between 20-30minutes (Constant, 1983). Since JF had only one acute pain

episode within the past 3 weeks and if it did last for 20-30minutes, the pain could have been due to myocardial infarction (Constant, 1983).

Although JF's pain was relieved by rest, I should have asked *how* he rested. Anginal pain is worse when a patient lies down due to the increased in venous return and blood volume (Constant, 1983). In some circumstances, a patient may lie down because he/she is not feeling well, but is unaware that lying down is persisting the pain (Constant, 1983). This situation could have possibly been true of JF due to his mental status and his feelings of impending doom. Since some patients do lie down, it is important to ask if the pain is relieved "within a *few seconds* of lying down." (Constant, 1983). To further rule out nonanginal pain, I should have asked if he had eaten food or drank water during his rest period and if the pain was relieved from food. If this was the case, it could be ruled out as nonanginal pain associated with gastrointestinal tract (Constant, 1983).

Another criteria to distinguish anginal and nonanginal pain are respiratory movements. Pain that increase with inspiration is considered nonanginal (Constant, 1983). Constant (1983) warns that the question needs to be asked, "if *one* deep breath can either bring on the pain or make it worse." Patients often misinterpret pain with hyperventilation and pain with inspiration. Repeated inspirations (hyperventilation) increase venous return and causes vasoconstriction making anginal pain worse (Constant, 1983). With JF's COPD, this question may have or not have been appropriate since he was already hyperventilating. If the pain was anginal, it could have been aggravated more by his hyperventilation.

Although I did discover his pain started when JF was bending down, I should have clarified on what he was doing as he stooped over. Was he tying his shoelaces or was he picking up something from the floor? Constant (1983) states that a person may experience true angina

while bending forward when hands are used such as tying one's shoelaces. If no hand movement is involved and the pain occurs within a few seconds of bending over, the pain can be classified as nonanginal (Constant, 1983).

Another question I did not inquire about was the specific radiation of the pain. According to Constant (1983), if the patient's pain radiates beyond the nuchal area (occipital protuberance) of the neck, it can be classified as nonanginal. To be more precise, Constant recommends for the patient to exactly point to the location of the radiation. In order to further narrow down the list of chest pain differentials, I should have asked JF if he experienced this similar chest pain before and if he experienced any other associated symptoms.

The other set of specific questions should have been directed toward his worsening dyspnea. Questions regarding cough, recent viral infections, and sputum production would point toward a case of COPD exacerbation while questions regarding fatigue, weight gain, paroxysomal nocturnal dyspnea, and edema would point more towards CHF exacerbation (Uphold & Graham, 2003).

In the history, I needed to have specifically asked questions to rule out nonanginal pain and life threatening conditions. Since this was my first meeting with him, it would have been prudent for me to inquire more about his mental status by asking some short and long- term memory questions. I realized that I regularly do not ask the simple neurology questions because I have found that the elderly gets frustrated and majority of the patients have some form of dementia. Regardless, I should still remain objective and ask questions to determine their baseline neurological status.

Objective

Given the presenting problem of chest pain, JF's general appearance, vital signs, cardiovascular, respiratory, and gastrointestinal systems were important to assess. In order to rule out any emergent conditions at the current time, his general appearance and vital signs would provide an immediate picture of whether or not to call for an ambulance. Although I had taken his respiratory rate and pulse, I failed to take his temperature and blood pressure. I could have also asked him if he had experienced any fevers within the past 3 weeks in the history as well. Despite my lack my questioning and measuring the temperature, when I palpated his abdomen and legs, the skin was warm, dry, and intact. In addition, from his general appearance, he was not anxious or diaphoretic. JF was tachypneic, but this could have been contributed more by his COPD or CHF. His somewhat disheveled outfit was an indicator of his lack of self- care.

A general observation of his appearance is important because a person manifesting MI or pulmonary embolism may appear diaphoretic, pale, anxious, and tachypnea (Dains, Ciofu-Baumann, & Scheibel, 2003). I did attempt to take a blood pressure. However, due to his atrial fibrillation, it was difficult for me to hear the Karatkoff's sounds and felt it was an unreliable measurement. Regardless, I realize this should have been noted in the documentation.

Chest pain can arise from various systems of the body such as the cardiovascular, respiratory, and gastrointestinal. For the cardiovascular assessment, I heard his distinct irregularly irregular rhythm characteristic that of atrial fibrillation or atrial flutter with varying blocks (Bickley, 2003). Other abnormal sounds such as transient S_3 or a S_4 could be a clinical sign associated with myocardial ischemia or infarction, HTN, or congestive heart failure (Dains, Ciofu-Baumann, & Scheibel, 2003). If I was present when he first experienced his chest pain, I

would have ordered for an EKG. The absence of jugular venous distention was another indicator that JF was not in *severe* heart failure (Bickley, 2003).

Due to his COPD, his anteroposterior was greater than his transverse diameter. This barrel chest figure is typical of a COPD patient (Bickely, 2003). However, I heard wheezes throughout his lungs bilaterally. Wheezes are indicative of fluid in the large airways or bronchospasms (Dains, Ciofu-Baumann, & Scheibel, 2003). I should have asked him to cough to see if the adventitious sounds cleared up. If it did, secretions may have caused the wheezes as in bronchitis or atelectasis (Bickley, 2003). With the presence of adventitious sounds, Dains et.al (2003) recommends additional auscultation for bronchophony, egophony, and whispered pectoriloquy. In either case, I felt his tachypnea, wheezes, and his past COPD history were enough to justify for a respiratory treatment.

Since chest pain can originate from the GI tract, abdominal assessment is needed. JF had positive bowel sounds and did not complain of any tenderness upon light and deep palpation. With his heart conditions, I examined his lower extremities. Bilaterally, his feet had pitting edema that extended up to his calves. This is typical sign of heart failure or venous stasis (Dains, Ciofu-Baumann, & Scheibel, 2003). Due to his edematous feet, his shoes were difficult to put back on and the client was unable to do it by himself. Despite the edema, JF's dorsalis pedis pulses were equal and palpated bilaterally. Due to his diabetes, I checked his feet for sores and lesions as well as for his gross sensations.

After a review of the literature, I should have measured his blood pressure and temperature, palpated entire chest wall for tenderness, percussed the chest, performed additional auscultation of the lungs, examined clubbing of the fingers, and observed the presence of peripheral cyanosis (Dains, Ciofu-Baumann, & Scheibel, 2003). Additionally, in addressing his

incontinence, I should have examined the genitalia, performed a digital rectal examination (DRE), conducted a neurological and musculoskeletal examination (Dains, Ciofu-Baumann, & Scheibel, 2003).

In terms of the textbook guidelines, I believe that they are reasonable to do. However, I deferred the genitalia examination and the DRE. Upon reflection, I believe it was mainly due to my discomfort in performing a DRE and the fact JF was already followed up by urology. Regardless, as a future primary care provider, I should not defer this or refer to an urologist until I have performed the examination.

For this encounter, I had requested the following labs: urinalysis (UA), urine culture and sensitivity(C&S), prothrombin (PT)/international normalized ratio (INR), blood urea nitrogen (BUN), creatinine, electrolytes, hemoglobin A_1C, digoxin level, alanine transaminase (ALT), aspartate transaminase (AST), and a lipid panel. The UA and C&S were done to rule out any infection or systemic disease that may have contributed to incontinence (Dains, Ciofu-Baumann, & Scheibel, 2003).

Majority of the labs were performed due to his medications. The PT/INR was to check the therapeutic levels for coumadin. BUN and creatinine was to test his renal functions for glipizide, diltiazem, lisinopril, furosemide, and for urinary obstruction or retention. The electrolytes were monitored due to digoxin, dilitiazem, furosemide, and lisinopril. Hbg A_1 C was monitored for glucose and digoxin level acquired to ensure a therapeutic level. ALT and AST were drawn for gipizide, zocor, diltiazem, and avandia (Hopper-Deglin & Vallerand, 2003).

I did not obtain a level for cardiac enzymes since JF stated his chest pain occurred three weeks ago. Troponin-I is a cardiac specific marker for MI and remains elevated for 7-10 days after the cardiac event (Dains, Ciofu-Baumann, & Scheibel, 2003). Lactic dehydrogenase (LDH)

is another confirmatory test for MI. LDH rises 12-28 hours after MI and will remain elevated for up to 14 days (Dains, Ciofu-Baumann, & Scheibel, 2003). Since the chest pain occurred 3 weeks ago, both of these enzymes would not have provided a sufficient evidence of a MI. A lab value I should have ordered was a complete blood count (CBC). The CBC would have been appropriate for avandia as well as to rule out anemia as the precipitating factor for the unstable angina (Lowry, 2003).

I should have ordered another EKG to identify any changes in JF's rhythm after the chest pain incident. However, on an EKG, an ischemic event is not always evident on a patient who is experiencing an angina attack (Dains, Ciofu-Baumann, & Scheibel, 2003). Additionally, if changes did occur in an EKG during this possible ischemic event, these changes normally return to baseline after the ischemic event (Lowry, 2003). JF had a stress 2-D echocardiogram that was negative for induced ischemic response in November 2000 and has already been diagnosed with CAD. I could have ordered another stress echocardiogram to confirm an acute ischemic event, but my management of the patient would have been the same with or without the echocardiogram. JF is 90 years old, the less tests I order, the better it will be for his mental status and a wiser use of available resources.

Assessment:

I had originally indicated diagnosis of COPD, chest pain, urinary incontinence, and BPH. After a review of the literature, I should have listed a list of differential diagnosis to rule out other possibilities of chest pain. If I had asked more questions in the history such as ones I stated in my subjective critique, it would have been easier to narrow down my top 3 differentials. In any chest pain, it is prudent to rule out the most life threatening diseases: acute MI, aortic

dissection, acute coronary insufficiency, pulmonary embolism, dysrhythmias, and pnuemothorax (Dains, Ciofu-Baumann, & Scheibel, 2003). With the information I did acquire, my top three differential diagnoses for JF's chest pain could have been unstable angina, acute MI, and pulmonary hypertension.

JF has a history of angina and CAD. Although JF has several positive risk factors (e.g. over 45years old, hyperlipidemia, hypertension, diabetes, history of CAD) for MI, the pain was relieved by rest (Dains, Ciofu-Baumann, & Scheibel, 2003). During a MI, pain is usually not relieved by rest or nitroglycerin (Dains, Ciofu-Baumann, & Scheibel, 2003). Pulmonary hypertension can also mimic symptoms of chest pain and cause dyspnea (Lowry, 2003).

Since EKG was not done or cardiac enzymes drawn, I do not know with certainty to rule out MI. Pulmonary hypertension is evaluated through a cardiac catherization (Dambro, 2002). This test is an invasive test and could possibly pose a greater risk for JF than not receiving cardiac catherization. Therefore, from the evidence I collected, the most likely cause of JF's chest pain was unstable angina. JF already wears a nitroglycerin patch, but this may not have prevented a new attack of angina. Chronic anginal symptoms can lead to unstable angina. This results if angina occurs at rest or with little stimulation, a longer duration of anginal symptoms is experienced, an increase in severity of the symptoms, or other new findings associated with angina are evident such as pulmonary edema, mitral regurgitation murmur, S_3, hypotension, bradycardia, and/or tachycardia (Lowry, 2003). Rest also relieved JF's chest pain, which is a characteristic of anginal pain (Dains, Ciofu-Baumann, & Scheibel, 2003).

COPD exacerbation may have been a more precise description of JF's recent complaints of increasing SOB. However, the SOB could have also been due to a CHF exacerbation or possibly a combination of both COPD and CHF exacerbations. JF had evidence of peripheral

edema, paroxysmal nocturnal dyspnea, morning dyspnea, history of CHF, and wheezes. These are some signs and symptoms indicative of heart failure (Uphold & Graham, 2003). The most common symptom of COPD is exertional dyspnea that can be accompanied by chronic cough, wheezing, recurrent respiratory infections, fatigue, weight loss, and decreased libido (Uphold & Graham, 2003). Signs and symptoms for COPD exacerbation include worsening dyspnea, increase in sputum purulence, and increase in sputum volume (Uphold & Graham, 2003). Since I did not inquire about sputum, cough, or recent respiratory infections, it was difficult to distinguish exactly what may have caused JF's worsening dyspnea.

In my assessment, I did address urinary incontinence for a nursing diagnosis, but I could have specified the type of incontinence (urge, stress, total, etc.). Since JF was already diagnosed with BPH, it would be a reasonable probability the increase in incontinence could be due to worsening BPH or an infection. Other nursing diagnoses that I should have addressed are fluid volume excess, self care deficits in hygiene, grooming, and toileting, and impaired gas exchange (Carpenito, 2000). I did not address any family or psychosocial diagnoses. Since JF currently fears the pain will re-occur, psychosocial diagnoses of fear and anxiety may be considered (Carpenito, 2000).

Plan:

Many more items could have been addressed in the plan. I should have ordered an EKG and a chest x-ray. An EKG may show a change in the existing rhythm or reveal a MI. A chest x-ray would re-evaluate JF's COPD condition as well as the extent of his pulmonary edema. Both the chest x-ray and the EKG will also assist in ruling out the life threatening conditions of chest pain (MI, aortic dissection, pulmonary embolism, pnuemothorax, etc.) (Dains, Ciofu-Baumann, & Scheibel, 2003).

Depending on the results from the chest x-ray, an increase in lasix or adding spironolactone may improve JF's respiratory symptoms especially if the dyspnea was from CHF exacerbation. In the RALES trial (Randomized Aldactone Evaluation Study), over 1600 patients with NYHA Class III/IV heart failure currently treated with ACE inhibitor, +/- digoxin, loop diurectic, and beta-adrenergic receptor antagonist, were randomly assigned to receive 25mg spironolactone or placebo. The group receiving spirnolactone resulted in 30% decreased mortality rate and 35% decreased hospitalization rates (Pitt, Zennad, & Remme, 1999).

Another option may be to add metolazone. It is common for patients who have been on long- term diuretic therapy or in advanced heart failure to experience a state of relative diuretic resistance (Regan, 2003). In this situation, adding metolazone in combination with a loop diurectic may promote more efficient diuresis (Regan, 2003).

Since JF has concomitant diseases, drugs that could treat multiple diseases are preferred. Although beta blockers are one of the first line management for heart failure, they are contraindicated in patients with COPD (Uphold & Graham, 2003). JF is already receiving an ACE inhibitor, digoxin, diurectic, nitroglycerin patch, and CCB for the management of his heart failure, hypertension, and angina. Although CCB may be good for management of chronic stable angina, they have a negative inotropic effect and have been associated with increased risk of cardiovascular events (Eisenberg, Brox, & Bestawros, 2004). Eisenberg et.al. recommend that long acting CCB can be safely used to manage hypertension and angina, but may not be protective in heart failure (Eisenberg, Brox, & Bestawros, 2004). Thus, a change to amlodipine, a dihydropyridine, may be a better option to manage his angina and heart failure (Uphold & Graham, 2003).

JF had only combivent inhaler available to control his COPD symptoms. With his worsening dyspnea, the nebulizer treatments may be beneficial to manage the acute dyspnea (Uphold & Graham, 2003). However, in review of literature, there has been an association of inhaled beta agonists with increased risk of unstable angina and MI in patients with COPD (Au, Randall Curtiss, Every, et.al., 2002). Therefore, once JF's symptoms improve after the nebulizer treatments, a change of combivent to a PRN order for an anticholinergic agent such as atrovent may be considered. According to current acute exacerbation of COPD guidelines, anticholinergic bronchodilators have fewer side effects and should be considered before a beta $_2$ agonist (Snow, Lascher, Mottur-Pilson, 2001). The current evidence based guidelines also recommend an addition of systemic oral corticosteroids for 2 weeks (Snow, Lascher, Mottur-Pilson, 2001). Since JF has diabetes, I would defer corticosteroids due to hyperglycemia side effect unless the dyspnea is not controlled by the inhaler.

To address his chest pain, short-acting SL nitrates were ordered. Due to his cognitive status and his deteriorating self- care, I am not sure if JF would be capable to administer the nitrates when he needed it. If this was the case, I should have increased the nitroglycerin patch dose to 0.4mg/hour. Because of his polypharmacy, the labs were needed to make appropriate medication changes as well as to rule out infection.

JF's yearly health maintenance exam will be done in April, but I did notice he already received his pneumococcal vaccine and his flu vaccine. With the elderly, primary prevention is focused on maintaining physical activity, nutrition, social support, self-care, current immunizations and vaccinations, and preventing falls and injuries (Worcester, 2000). In JF's situation, the most pressing concern was his high risk for self-care deficit. With JF's incontinence issue, keeping up with his hygiene was too much for the staff and for him. Plus,

with his dyspnea and swollen ankles, it was difficult for JF to get his shoes on. This leads me to wonder how much activity he can tolerate with his CHF and COPD. Therefore, I should have discussed how much his symptoms interfered with his lifestyle and self-care. How is his outlook on his remaining years? How can I manage his physical activity so that he will not lose his current capabilities? This is because patients with CHF are still encouraged some form of physical activity and bedrest is discouraged, unless in times of acute exacerbations, due to the possibility of further decline in physical strength and abilities (Packer, 2000).

I should have documented his return visit in two or three days to see if his symptoms of dyspnea improved or if he experienced any more chest pains. Regardless, in the clinic, the nursing staff reports any concerns or worsening of symptoms of any patients to the NP the following week or the NP paged. Teaching should have been done and documented about the administration of SL nitroglycerin to patient and the nursing staff. An explanation of JF's decline in self-care and worsening of his symptoms were given as reasons for the possibility of JF's move to the assisted living facility in the future.

For billing, JF has Medicare/Medicaid and Blue Cross Blue Shield. Finance was not a concern for his management of care. For this visit, the NP used 99213. JF's appointment involved a detailed history as well as a detailed examination according to the Medicare guidelines for new physicians (AMA, 2002). The ultimate consideration to JF's management was how to improve the *quality* of life in these remaining years by improving the management of his acute and chronic symptoms. A great deal and attempt is made to ensure maximum amount of autonomy and self- maintenance. The management recommendation from texts was more appropriate for patients that are younger than JF. I felt that the changes I had recommended from

above should be done slowly and done one at a time. With his age, any sudden discontinuation or change in medication may worsen his health status.

When I was faced with managing JF's concern, I was overwhelmed by his current and past conditions. Needless to say, I did not have much knowledge or experience in handling chest pain management. Besides the labs and the SL nitroglycerin tablets, the NP recommended the other treatments and we did not have any "issues." We also felt it was not necessary to pursue invasive tests or surgeries due to his age and medical conditions.

Overall Reflection:

I particularly chose JF because he was my first experience with chest pain. In addition, JF had many other concomitant chronic diseases that challenged my critical thinking skills to the next level. Although it was difficult and time staking, I wanted to take on this case to review the current management and guidelines for the common chronic conditions.

Once again, I realized how important the subjective history is in relation to everything else in the plan. My objective, assessment, and plan all derive in my skills as an accurate history recorder. In the history, I should not automatically assume that JF experienced another angina attack. This narrow mindedness led me to forget asking other important questions and qualities regarding his chest pain. I noticed without a good history, it was difficult to narrow down the possible list of differentials. If I can't diagnose or rule out certain conditions because of missed information in the history, unnecessary tests or steps may occur. This leads to more expenses by the patient and the government and unwise use of time and resources.

Due to my lack of history taking skills, I learned much about interviewing when it came to differentiating anginal pain from nonanginal pain. JF's multiple diseases allowed me to consider management of other conditions such as his CHF, COPD, diabetes, and atrial

fibrillation and how to treat patients with co-morbid conditions. I also learned the importance of improving patient's quality of life rather than aggressively managing his/her conditions especially toward their later years.

By answering questions from each critique section, I realized how it was not natural for me to consider family, psychosocial, and financial considerations. I was so concerned with his medical management that I neglected his potential issues with his family. If JF was to move into the assisted living facility where more care is required, would the family have enough financial resources to provide for this transition? How does JF feel about the transition? Is he willing to do this? I realized that I needed to look at the patient more holistically and not just medically.

The NP uses both a medical and nursing model in her approach in managing the patients. She has been the primary care provider for many of the patients in the course of five years. Therefore, she is keen to observe the changes in mental status in individuals as well as what changes in his/her medical management may not be suitable. The NP works closely with the nursing staff and the families. The staff comments on how the NP is more independent and helpful than the physicians due to her nursing background. Many times I hear the comment of how the NP "knows" her patients. By developing and maintaining these long and trusting relationships with the patients and families, she is able to provide better care and treatment.

I am continuing to learn the "art" of medicine and I feel it begins with knowing your patient. By engaging in effective communication, providing continuity and comprehensive care, and client-centered treatments, the advanced practice nurse can truly impact the client, his/her family, and the staff in managing chronic illnesses.

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