Pharmacology: Mind Mapping Case Study

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The client we chose for this case study had a variety of chronic and episodic health challenges that were treated with several different medications. In this case study we discuss in depth these chronic and episodic health challenges; the prescribed medications and the pharmacology behind them; the patient’s lab values and diagnostics; and discuss how all the indicated points link together and affect the client. Finally, we included six strategies for our patient’s care and discussed how to implement these strategies into our clinical practice to benefit the patient and promote optimal health and healing.

# Chronic and Episodic Health Challenges

## Bipolar Disorder

Previously referred to as manic depression (Parker & Parker, 2003); bipolar disorder is a mental disorder characterized by reoccurring episodes of depression and the abnormal elevation of mood (2003). The elevation in mood is characterized by severe mood elevation, prolonged mood elevation, or the association with psychosis (Parker & Parker, 2003); if symptoms are less severe, short in duration, and associated with low levels of disturbance it is called *hypomania* (2003). Depression is being in a state of low mood, aversion to activity, and can result in altered perception to the negative (Parker & Parker, 2003). Symptoms of depression and mania may occur alone or together (Parker & Parker, 2003). Bipolar disorder is divided into bipolar I, characterized by episodes of depression and mania, and bipolar II being characterized by episodes of depression and hypomania (Parker & Parker, 2003). Exact causes and mechanisms behind bipolar disorder are not fully understood making risk assessment difficult (Goldberg, 2019); in addition, comorbidities with other psychiatric disorders are common and further complicate the epidemiology of bipolar disorder (2019). Bipolar disorder has an overall similar prevalence between men and women (Goldberg, 2019); with men having a higher prevalence of bipolar I, and women having a higher prevalence of bipolar II (2019). The primary causes for bipolar disorder are related largely to genetic predisposition, heritage, and environmental factors (Goldberg, 2019); those with a family history, having experienced physical or sexual abuse as a child, or having experienced severe or chronic stress all have a much high incidence of bipolar disorder (2019). Less commonly, neurological injury such as trauma, stroke, or infection may result in bipolar disorder (Goldberg, 2019). Signs and symptoms of bipolar are as previously described, including episodes of mania or hypomania, and episodes of depression (Parker & Parker, 2003).

**Hypothyroidism**

Hypothyroidism is a disorder of the endocrine system (Chaker, Bianco, Jonklaas, & Peeters, 2017). The primary cause of hypothyroidism is when the thyroid gland is unable to produce adequate levels of thyroid hormones (thyroxine, tri-iodothyronine, and calcitonin) resulting in the slowing of a person’s metabolism (Chaker et al., 2017). Other causes include thyroid stimulating hormone deficiency, thyrotropin-releasing hormone deficiency, or over expression of deiodinase-3 enzyme seen with tumor tissues (Chaker et al., 2017); together, these three are responsible for less than one percent of all hypothyroidism cases (2017). Women are more likely to develop hypothyroidism due to increased prevalence of hormonal imbalance, hormonal fluctuations, and increased autoimmune susceptibility (Hashimoto’s disease) (Chaker et al., 2017). Additionally, people over the age of sixty are at an increased risk due to the increase in prevalence of autoimmune thyroiditis as a result of aging (Chaker et al., 2017). Those with a pre-existing autoimmune disease are also at an increased risk for developing hypothyroidism as the body can mistake thyroid cells and their enzymes as foreign and destroy them (Chaker et al., 2017). The surgical removal (complete or partial) of the thyroid gland will result in hypothyroidism (Chaker et al., 2017). Those receiving radiation (radioactive iodine) treatment will lose part or all of their thyroid function (Chaker et al., 2017). Those taking specific medications such as amiodarone, lithium, interferon alpha, and interleukin-2 can all impair thyroid function (Chaker et al., 2017). Those with too much or too little iodine in their diet may develop hypothyroidism as iodine is required for the body to make thyroid hormones (Chaker et al., 2017). Fatigue, constipation, weight gain, cold intolerance, lethargy, dry skin, and changes in the voice (hoarseness) are the most common symptoms of hypothyroidism (Chaker et al., 2017). Signs and symptoms of hypothyroidism vary widely in clinical presentation based on a variety of factors including gender, age, and the time between onset of hypothyroidism and the diagnosis (Chaker et al., 2017). However, these signs and symptoms are not good predictors of hypothyroidism and alone are often not enough to identify patients with the condition (Chaker et al., 2017). Furthermore, depending on the cause of the hypothyroidism, patients may be asymptomatic (Chaker et al., 2017). The current treatment for hypothyroidism is drug therapy in the form of Levothyroxine (Brenta et al., 2013); a drug identical to the thyroid hormone your body can no longer naturally produce (2013). Assessment of the thyroid gland includes observation and palpation of the thyroid gland, assessing for swelling (goiter), firmness (Hashimoto’s), or tenderness (thyroiditis) (Brenta et al., 2013). Assessing for worsening or unresolving symptoms includes activity intolerance, cold intolerance, constipation, and weight gain (Brenta et al., 2013). Interventions should follow the nursing diagnosis of symptoms/ presenting issues which include promoting rest, protection against cold, development of an exercise regime, proper diet (referral to dietician) (Brenta et al., 2013). Lastly, patient teaching of the disease and medication compliance includes letting the patient know that the treatment is not curative and involves lifelong medication compliance in order to maintain thyroid levels (Brenta et al., 2013).

**Glaucoma**

Glaucoma is optic neuropathy that is characterized by the degeneration of retinal ganglion cells (Weinreb, Aung, & Medeiros, 2014). It is progressive and results in “cupping” of the optic disc and eventually leads to vision loss (Weinreb, Aung, & Medeiros, 2014). It is the leading cause of irreversible blindness and can remain asymptomatic until it has become severe (Weinreb, Aung, & Medeiros, 2014). There are two categories of glaucoma which include open-angle and angle-closure (2014). Primary disease, secondary glaucoma can result from trauma, corticosteroids, tumors, and inflammation (Weinreb, Aung, & Medeiros, 2014). Risk factors for glaucoma include age, race (African Americans are at a higher risk), high intraocular pressure, corticosteroid use, and a family history of glaucoma (Weinreb, Aung, & Medeiros, 2014). Those with a family history of glaucoma should be referred for examination for the possible early detection of glaucoma (Weinreb, Aung, & Medeiros, 2014). High intraocular pressure is a risk factor for the development of glaucoma, however not all people who have high pressure develop glaucoma (Weinreb, Aung, & Medeiros, 2014). Glaucoma is often asymptomatic until the disease has advanced far enough to have caused advanced neural damage (Weinreb, Aung, & Medeiros, 2014). Glaucoma results in vision loss thus reducing quality of life and inhibiting the activities of daily living of those affected (Weinreb, Aung, & Medeiros, 2014). Early intervention is crucial to prevent glaucoma from advancing far enough to cause blindness, and those who are at risk should be referred to optometrists (Weinreb, Aung, & Medeiros, 2014). The appearance of the optic nerve is affected, as well has the retinal nerve fiber layer due to ganglion cell death and nerve fiber loss (Weinreb, Aung, & Medeiros, 2014). These are considered the most important features that can be used to diagnose glaucoma therefore it is important for patients who are at risk to receive an ophthalmologic examination (Weinreb, Aung, & Medeiros, 2014). Because glaucoma often goes undetected, slowing the disease progression as well as preserving the quality of life of those affected are the main goals of treatment for glaucoma (Weinreb, Aung, & Medeiros, 2014). Early diagnosis and treatment is important to avoid severe damage, and the reduction of intraocular pressure is shown to be the only proven method for the treatment of glaucoma (Weinreb, Aung, & Medeiros, 2014). Lowering the intraocular pressure was shown to prevent the development, and slow the progression of glaucoma (Weinreb, Aung, & Medeiros, 2014). Nurses must ensure to assess the patient’s history of corticosteroid use, if the patient has experienced any vision loss, and changes in ambulation or mobility (Weinreb, Aung, & Medeiros, 2014).

**Pancreatitis**

Pancreatitis is an inflammatory disease that most often occurs when localized inflammation turns into systemic inflammation (Johnstone, 2018). This can cause tissue damage, and organ dysfunction, and long-term can be a cause of diabetes mellitus due to pancreatic damage (Johnstone, 2018). Pancreatitis is commonly associated with gallstones which can cause an obstruction in the pancreatic duct which may require surgery to remove the gallbladder (Johnstone, 2018). Pain management, fluids, and nutritional care should be implemented to treat the symptoms of pancreatitis (Johnstone, 2018). Risk factors for pancreatitis include smoking, diet, metabolic factors, and injury or surgery to the abdomen (Johnstone, 2018). Alcohol is a big risk factor for pancreatitis, and prolonged excessive alcohol intake accounts for around twenty-five percent of all pancreatitis cases (Johnstone, 2018). Gallstones are the most common cause of pancreatitis, accounting for fifty percent of all cases (Johnstone, 2018). Regardless of the cause, it is thought that trypsin is initially activated and in turn triggers the activation of other digestive enzymes (Johnstone, 2018). Pancreatitis is characterized by a severe and constant abdominal pain that is described to wrap around the body (Johnstone, 2018). It causes a bloated and distended abdomen, nausea and vomiting, fever, and unintended weight loss (Johnstone, 2018). These signs and symptoms can be associated with other conditions as well, so for pancreatitis to be diagnosed the patient needs to be exhibiting acute and sudden abdominal pain that radiates to the back accompanied with elevated blood lipase or amylase levels (Johnstone, 2018). Treatment of pancreatitis is hospitalization with intravenous fluids and a tailored diet to allow the pancreas to heal (Johnstone, 2018). Nasogastric and jejunostomy tubes may be required depending on the situation (Johnstone, 2018). Surgery for pancreatitis is not uncommon (Johnstone, 2018). It is important for the nurse to monitor the abdomen for distention, tenderness, bowel sounds, pain, fluid balance, and nutritional status (Johnstone, 2018).

**Anemia**

Anemia is a blood disorder characterized by a deficiency in erythrocytes, hematocrit (Hct), and the quantity of hemoglobin (Hgb) (Lewis, Heitkemper, Bucher, Dirksen & Camera, 2014). Iron deficient anemia is further characterized by inadequate iron stores, resulting in insufficient Hgb which may cause the cells to appear abnormal, microcytic, and hypochromic (Doenges, Murr, & Moorhouse, 2019). Anemia is a manifestation of a pathological process or diseased state in which there is a decreased production of erythrocytes or increased destruction of erythrocytes (Lewis et al., 2014). Anemia is the result of either a primary hematological dilemma or is a secondary consequence of other defects within the body (Lewis et al., 2014). Decreased Hgb synthesis is a common cause of iron-deficient anemia that causes decreased red blood cell (RBC) production (Lewis et al., 2014). Additionally, anemia has been associated with hypothyroidism in that hypothyroidism has been shown to contribute to the decreased production of erythropoietin and contributes to the malabsorption of iron and malnutrition that it common within anemic patients (Wopereis et al., 2018). Iron-deficient anemia is a common chronic hematological disorder that affects approximately thirty percent of the world’s population (World Health Organization, 2012). Populations that are most susceptible to iron-deficient anemia in Canada include women, children, and those with poor dietary habits (Lewis et al., 2014). The incidence of anemia is twice as prevalent in women than in men (Doenges et al., 2019). Iron-deficient anemia often develops due to inadequate dietary intake, malabsorption, or hemolysis (Lewis et al, 2014). Since iron is obtained from food and dietary supplements, those with poor dietary habits or health conditions may not obtain sufficient amounts of dietary iron on a daily basis (Lewis et al., 2014). Anemia is common in older adult patients which is typically due to insufficient dietary intake, renal insufficiency, or malabsorption (Lewis et al., 2014). The likelihood of anemia occurrence increases significantly in older adult patients with co-morbid conditions, such as hypothyroidism (Lewis et al., 2014). In addition, iron absorption occurs primarily in the duodenum therefore, certain malabsorptive syndromes or gastrointestinal surgery may result in the malabsorption of iron (Lewis et al., 2014). Pregnant or postmenopausal woman are also at increased risk for iron-deficient anemia due to postmenopausal bleeding and the diversion of iron to the fetus for erythropoiesis or blood loss during delivery (Lewis et al., 2014). Clinical manifestations of anemia reflect the body’s response to tissue hypoxia and the presentation of symptoms may vary depending on the severity of the anemia and presence of coexisting disease (Lewis et al., 2014). Generally, iron-deficient anemia does not present with clinical manifestations during the early stages of the condition, however, some reported symptoms include palpitations, dyspnea, and diaphoresis (Lewis et al., 2014). General manifestations can occur as the disease progresses into a moderate to severe condition which include: pallor, jaundice, blurred vision, retinal hemorrhage, glossitis, tachycardia, increased pulse pressure, systolic murmurs, angina, tachypnea, orthopnea, headache, irritability, anorexia, splenomegaly, hepatomegaly, bone pain, sensitivity to cold, weight loss, and lethargy (Lewis et al., 2014). The goal of treatment is to cure the cause of anemia and may include acute interventions and/or dietary and lifestyle changes (Lewis et al., 2014). Acute interventions would include blood transfusions, volume replacement, oxygen therapy, or drug therapy, whereas dietary and lifestyle changes may include collaboration with a dietician, increases in iron and protein intake, and alterations in physical exercise (Lewis et al., 2014). It is important for nurses to recognize patients with, or at risk for, iron-deficient anemia (Lewis et al., 2014). Nurses can provide nutritional education that emphasizes the importance of ingesting iron-rich foods and how to maximize iron absorption (Lewis et al., 2014). Drug therapy is another option for iron-deficient anemics however, the regime may require the patient to be compliant with their iron supplements for at least two to three months after Hg levels have returned to normal (Lewis et al., 2014). It is important to reassess the Hg and RBC levels throughout treatment to evaluate the effectiveness of therapy (Lewis et al., 2014). Additionally, patients that require long-term iron supplementation should have frequent blood and hepatic studies to monitor for potential liver dysfunction related to the storage of iron (Lewis et al., 2014).

**Atrial Fibrillation**

Atrial fibrillation is characterized as a high frequency excitation that results in both an unsynchronized atrial contraction and irregular ventricular excitation (Iwasaki, Nishida, Kato, & Nattel, 2011). The impulse and rhythm within the atria is irregular because of the multiple pacemaker sites and a rhythm conducted by the ventricles that is unpredictable (Potter, Perry, Stockert, & Hall, 2019). In an electrocardiogram, the QRS complex is normal, however the electrical conduction occurs at irregular intervals (Potter, Perry, Stockert, & Hall, 2019). Due to the irregular activity of the atria, it consequentially results in an irregular ventricular response and ultimately an irregular cardiac rate and rhythm (Potter, Perry, Stockert, & Hall, 2019). There is a loss of coordination between the squeeze of the ventricles and atrial contraction resulting in an ineffective cardiac output (Potter, Perry, Stockert, & Hall, 2019). Additionally, there is the pooling of blood in the atria due to an ineffective heart pump producing a lower cardiac output, and the development of micro-emboli due to the stasis of blood within the atria (Potter, Perry, Stockert, & Hall, 2019). Atrial fibrillation is the most common arrythmia diagnosed and affects approximately 200,000 Canadians (Heart and Stroke Foundation of Canada, 2018). There are several unmodifiable risk factors contributing to the incidence of atrial fibrillation including, but not limited to, genetics, advanced age, gender, and racial differences (Iwasaki, Nishida, Kato, & Nattel, 2011). Incidence of atrial fibrillation is higher in men in comparison to women in North American and European populations (Staerk, Sherer, Ko, Benjamin, & Helm, 2017). The modifiable risk factors that can contribute to the development of atrial fibrillation include physical inactivity and a sedentary lifestyle, smoking, obesity, diabetes, obstructive sleep apnea, and hypertension (Staerk, Sherer, Ko, Benjamin, & Helm, 2017). Symptoms of atrial fibrillation can range from asymptomatic to symptomatic (American Heart Association, 2016). Symptoms can include fatigue, rapid heartbeat, fluttering in chest, dizziness, shortness of breath, anxiety, faintness, or chest pressure (American Heart Association, 2016).

**Medications**

**Esomeprazole**

Esomeprazole trihydrate, also known as Nexium, is part of the functional class of Proton Pump inhibitors (Lilley, Collins, Snyder, & Swart, 2016). Proton pump inhibitors are the first-line therapy for various types of ulcers including active duodenal ulcers, active benign gastric ulcers, and NSAID-induced ulcers (Lilley, Collins, Snyder, & Swart, 2016). They are considered to be first-line therapy for erosive esophagitis, hypersecretory conditions such as Zollinger-Ellison syndrome, and gastro-esophageal reflux disease that has been unsuccessfully treated with H2 antagonists (Lilley, Collins, Snyder, & Swart, 2016). Proton pump inhibitors have also had success in treating H. pylori infections when used in combination with antibiotics (Lilley, Collins, Snyder, & Swart, 2016). This classification of medications are powerful because they directly bind to the hydrogen-potassium-ATPase pump to inhibit the enzyme, and as a result blocks the secretion of hydrogen ions out of the parietal cells (Lilley, Collins, Snyder, & Swart, 2016). Some common side effects associated with proton pump inhibitors include headaches, diarrhea, nausea, flatulence, stomach pain, constipation and dry mouth (Healthline, 2018). Although proton pump inhibitors are generally well tolerated, there is an increased risk of acute kidney injury and interstitial nephritis in the older adult population (Lilley, Collins, Snyder, & Swart, 2016). Because proton pump inhibitors make patients temporarily achlorhydric, they are also thought to increase bone mineral loss (Lilley, Collins, Snyder, & Swart, 2016). Additionally, with long term use of esomeprazole can increase the risk of osteoporosis-related fractures and atrophic gastritis (Healthline, 2018). It is important to consider the drug interactions of Esomeprazole as it can increase the serum levels of other medications, increasing the risk of toxicity (Skidmore-Roth, 2019). These drug interactions include diazepam, digoxin, penicillin, saquinavir, cilostazol, clozapine, warfarin, and drugs that are metabolized by the CYP2C19 enzyme in the liver (Skidmore-Roth, 2019). In the older adult population, excretion of esomeprazole is decreased, and bioavailability is increased therefore the risk of toxicity is increased (U.S. Food and Drug Association, 2006). Esomeprazole can also decrease the effect of certain medications such as atazanavir, clopidogrel, calcium carbonate, Vitamin B12, and iron (U.S. Food and Drug Association, 2006). It is important to perform regular gastrointestinal assessments for bowel sounds, abdominal pain, distention, anorexia, and bloody stools (Skidmore-Roth, 2019). After oral administration peak plasma levels occur at approximately 1.5 hours (U.S. Food and Drug Association, 2006); the plasma levels increases proportionally when the dose is increased (2006). Esomeprazole is 97% bound to plasma proteins (U.S. Food and Drug Association, 2006). Esomeprazole is extensively metabolized in the liver by the cytochrome P450 (CYP) enzyme system (U.S. Food and Drug Association, 2006). The plasma elimination half-life of esomeprazole is approximately 1-1.5 hours (U.S. Food and Drug Association, 2006). 80% of an oral dose of esomeprazole is excreted as inactive metabolites in the urine, and the remainder is found as inactive metabolites in the feces (U.S. Food and Drug Association, 2006). Hypoglycemia may occur in patients with diabetes therefore it is important to know the signs and symptoms of hypoglycemia and to monitor blood sugars regularly (Skidmore-Roth, 2019). Avoid taking Esomeprazole with alcohol, salicylates, and NSAIDs because it may cause GI irritation (Skidmore-Roth, 2019). Take Esomeprazole at least 1 hour meals but do not crush or chew the tablet (Skidmore-Roth, 2019).

**Latanoprost**

Latanoprost (ophthalmic), also known as Xalatan, is part of the functional class of Antiglaucoma agents, specifically the prostaglandin agonists (Skidmore-Roth, 2019). For increased intraocular pressure in patients with open-angle glaucoma and are otherwise unresponsive to other intraocular lowering medications (Skidmore-Roth, 2019). Latanoprost is a prodrug of a prostaglandin known as prostaglandin F2a· (Lilley, Collins, Snyder, & Swart, 2016). When Latanoprost is administered, it is converted via hydrolysis to prostaglandin F2a1 which decreases intraocular pressure (Lilley, Collins, Snyder, & Swart, 2016). Prostaglandins work to decrease intraocular pressure by increasing the outflow of fluid between the uvea, the sclera, and the trabecular meshwork (Lilley, Collins, Snyder, & Swart, 2016). Common side effects of Latanoprost include sensation of a foreign body in the eye, conjunctival hyperemia, iris colour change, ocular pruritis, stinging, blurred vision, and burning (Skidmore-Roth, 2019). It is important to assess therapeutic effects of Latanoprost by assessing the intraocular pressure in patients who experience ongoing increased intraocular pressure (Skidmore-Roth, 2019). If Latanoprost is used with other intraocular lowering medications, be sure to administer them at least 5 minutes apart from each other (Skidmore-Roth, 2019). After ocular route administration, the onset of action is 30-60 minutes with peak plasma concentrations occurring after 2 hours (Lilley, Collins, Snyder, & Swart, 2016). It has an elimination half-life of 17 minutes and its duration of action is 24 hours allowing for less frequent dosing (Lilley, Collins, Snyder, & Swart, 2016). Latanoprost is absorbed through the cornea and is 90% plasma protein bound (U.S. Food and Drug Association, 2011). It is hydrolyzed by esterase in the cornea to its biologically active form and is metabolized by the liver (U.S. Food and Drug Association, 2011). It undergoes rapid elimination by the kidneys (U.S. Food and Drug Association, 2011). Contact lenses should be removed prior to administration, and reinserted 15 minutes afterwards (Skidmore-Roth, 2019). Administer the drops by tilting the head back and pulling the lower eyelid down (Skidmore-Roth, 2019); once you have administered the drops, do not blink and keep eyes closed for 1-2 minutes (2019). Make sure to keep the tip the dropper clean (Skidmore-Roth, 2019).

**Zopiclone**

Zopiclone, also known as Imovane or Rhovane, is a short acting benzodiazepine-like drug and belongs to the class of sedative-hypnotics (Lilley, Collins, Snyder, & Swart, 2016). Used for short term treatment of insomnia (Lilley, Collins, Snyder, & Swart, 2016). Zopiclone interacts with the GABA-A receptor to enhance the inhibitory actions of GABA. This ultimately produces the hypnotic and anxiolytic therapeutic effects of Zopiclone (DrugBank, 2020). Common side effects can be seen especially in the senior population and includes agitation, behavioural changes, fast or irregular heartbeat, bitter taste, anorexia, diaphoresis, trembling or shaking in extremities, vomiting, confusion, and difficulty with coordination (Rexall Pharmacy Group, 2020). Avoid this medication in patients who have severe dyspnea, liver disease, or sleep apnea (Rexall Pharmacy Group, 2020). The use of Zopiclone with alcohol increases the risk of severe dyspnea (Rexall Pharmacy Group, 2020). Zopiclone when used with other CNS depressants (antidepressants, opioids, sedatives/hypnotics) can increase the risk of CNS depression (F. A. Davis Plus, 2015). Zopiclone may also increase the risk of CNS depression when administered with medications that inhibit the CYP3A4 enzyme system (F. A. Davis Plus, 2015). It is important for the nurse to assess patient mental status, and sleep patterns (F. A. Davis Plus, 2015). Zopiclone should not exceed usage more than 7-10 days as it may lead to physical and psychological dependence (F. A. Davis Plus, 2015). Additionally, it is important to assess patient’s pain as increased pain levels may decrease sedative effects (F. A. Davis Plus, 2015). Zopiclone after being administered orally has an onset of 30 minutes, peaks its plasma concentration at 90 minutes, has an elimination half-life of 5 hours, and has a duration of action of 6-8 hours (Lilley, Collins, Snyder, & Swart, 2016). After oral administration, Zopiclone is rapidly absorbed (approximately 75%) and is rapidly distributed from the extracellular compartments (F. A. Davis Plus, 2015). Zopiclone is metabolized by the CYP3A4 enzyme system in the liver and 4-5% of its metabolites are excreted in the urine (F. A. Davis Plus, 2015). The use of Zopiclone with alcohol increases the risk of severe dyspnea (F. A. Davis Plus, 2015). Because of Zopiclone’s rapid onset, it is best to go to bed as soon as possible (F. A. Davis Plus, 2015). Zopiclone may cause daytime drowsiness or dizziness so it is best to avoid activities that require alertness until the response to the medication is known (F. A. Davis Plus, 2015).

**Acetaminophen**

Acetaminophen, also known as Tylenol, is a non-opioid analgesic medication used for mild to moderate pain (Hazard-Vallerand & Sanoski, 2018). Acetaminophen peripherally blocks pain impulses (Hazard-Vallerand & Sanoski, 2018). Specifically, it inhibits prostaglandin synthesis in the central nervous system thus reducing inflammation and increasing the pain threshold (Hazard-Vallerand & Sanoski, 2018). Common side effects of Acetaminophen include hypersensitivity reactions and kidney damage with prolonged use (Hazard-Vallerand & Sanoski, 2018). With intravenous use, nausea, headache, abdominal pain/discomfort are more often seen (Hazard-Vallerand & Sanoski, 2018). Prior to administration, assessment of patient for malnourishment or chronic alcohol use is advised (Hazard-Vallerand & Sanoski, 2018); these individuals are at greater risk developing hepatotoxicity with chronic use of this drug (2018). It is also very important for the nurse to evaluate the patient’s pain and/or fever, and the therapeutic effects of the medication (Hazard-Vallerand & Sanoski, 2018). Depending on the dosage and schedule of medication administration, periodic evaluation of lab test results for hepatic and renal damage may be necessary (Hazard-Vallerand & Sanoski, 2018). It is important for the nurse to notify patients to discontinue the use of the medication if a rash occurs and to inform the primary health care provider immediately (Hazard-Vallerand & Sanoski, 2018). Additionally, it is important to advise patients to not exceed dosage in excess of four grams in a 24 hour period as it can result in hepatotoxicity, renal or cardiac damage (Hazard-Vallerand & Sanoski, 2018); these effects are further exacerbated by alcohol consumption (2018). Acetaminophen is well absorbed following oral administration while rectal absorption is variable (Hazard-Vallerand & Sanoski, 2018). Intravenous administration results in complete bioavailability (Hazard-Vallerand & Sanoski, 2018). Acetaminophen is widely distributed, it can cross the placenta, and it can enter breast milk in low concentrations (Hazard-Vallerand & Sanoski, 2018). This medication is 85– 95% metabolized by the liver via the CYP2E1 enzyme system and the metabolites are excreted by the kidneys (Hazard-Vallerand & Sanoski, 2018). In adults, acetaminophen has a half-life of one to three hours (Hazard-Vallerand & Sanoski, 2018). It has an onset of 30 minutes to one hour, a peak of 30 minutes to three hours, and a duration of three to eight hours (Hazard-Vallerand & Sanoski, 2018). The onset, peak, and duration of acetaminophen are dependent on the route of administration (Hazard-Vallerand & Sanoski, 2018).

**Dimenhydrinate**

Dimenhydrinate, also known as Gravol, is indicated for nausea, dizziness, and motion sickness related to vertigo (Hazard-Vallerand & Sanoski, 2018). Dimenhydrinate produces therapeutic effects due to its anticholinergic, depressant effects on the central nervous system and the inhibition of vestibular stimulation (Hazard-Vallerand & Sanoski, 2018). Common side effects of this medication include drowsiness, dry mouth, and decreased appetite (anorexia) (Hazard-Vallerand & Sanoski, 2018). It is important for nurses to monitor the intake and output of the patient, including emesis (Hazard-Vallerand & Sanoski, 2018). It is also important to consider the risk for fluid deficiency, imbalanced nutrition, and risk for falls as a result of the side effects exerted from the use of this medication (Hazard-Vallerand & Sanoski, 2018). Nurses must educate their patients to take this medication as soon as symptoms arise, or before conditions that may precipitate an episode of nausea/motion sickness (Hazard-Vallerand & Sanoski, 2018). Additionally, advise patients to use sunscreen in case of photosensitivity (Hazard-Vallerand & Sanoski, 2018). Dimenhydrinate is well absorbed after oral or intramuscular administration (Hazard-Vallerand & Sanoski, 2018). It has a wide distribution of three to four litres per kilogram (Hazard-Vallerand & Sanoski, 2018). It is metabolized by the liver and primarily in urine as metabolites (Hazard-Vallerand & Sanoski, 2018). It has an onset of 15 minutes to one hour, a peak of one to two hours, and a duration of three to twelve hours (Hazard-Vallerand & Sanoski, 2018); the half-life of Dimenhydrinate is unknown (2018). The onset, peak, and duration of acetaminophen are dependent on the route of administration (Hazard-Vallerand & Sanoski, 2018).

**Hydromorphone**

Hydromorphone, also known as Dilaudid, is an opioid analgesic used to treat moderate to severe pain (Hazard-Vallerand & Sanoski, 2018). It may be used in conjunction with nonopioid analgesics (Hazard-Vallerand & Sanoski, 2018). Hydromorphone attaches to opiate receptors within the central nervous system creating analgesic action (Hazard-Vallerand & Sanoski, 2018). It also acts on the medulla creating a depressant effect on the respiratory function and suppresses any cough (Hazard-Vallerand & Sanoski, 2018). Opioids also have the effect of inhibition of gastric emptying and peristalsis of the gastrointestinal tract (Hazard-Vallerand & Sanoski, 2018). Common side effects of this medication include a cough, sedation, constipation, and hypotension/orthostatic hypotension (this side effect is more rare) (Hazard-Vallerand & Sanoski, 2018).It is important for the nurse to assess a patient’s blood pressure, pulses, and respirations prior to administration and periodically after administration (Hazard-Vallerand & Sanoski, 2018); keep a consideration that the sedative side effects are common (2018). Assessment of bowel function and the implementation of a bowel care plan are also important considerations (Hazard-Vallerand & Sanoski, 2018). Prior to administration, ensure that the antidote Naloxone is available in case of an overdose (Hazard-Vallerand & Sanoski, 2018). Instruct the patient on how and when to ask for pain medication before the pain becomes unbearable (Hazard-Vallerand & Sanoski, 2018). Inform the patient of the sedative effects hydromorphone and encourage patients to turn, cough, and breathe deeply at least every two hours to help prevent atelectasis (Hazard-Vallerand & Sanoski, 2018). Hydromorphone is well absorbed following oral, rectal, subcutaneous, and intramuscular administration (Hazard-Vallerand & Sanoski, 2018). The extended-release product results in an initial release of the drug, followed by a second sustained phase of absorption (Hazard-Vallerand & Sanoski, 2018). Hydromorphone is widely distributed, crosses the placenta, and enters breast milk (Hazard-Vallerand & Sanoski, 2018). It is mostly metabolized by the liver and excreted primarily through the urine (Hazard-Vallerand & Sanoski, 2018).If Hydromorphone is administered as an immediate-release oral medication, or as an injection, it will have a half-life of two to four hours (Hazard-Vallerand & Sanoski, 2018). The extended release tablets will have a half-life of eight to fifteen hours (Hazard-Vallerand & Sanoski, 2018). It has an onset of ten to thirty minutes, a peak of fifteen to ninety minutes, and a duration of two to five hours (Hazard-Vallerand & Sanoski, 2018). The onset, peak, and duration of Hydromorphone are dependent on the route of administration (Hazard-Vallerand & Sanoski, 2018).

**Metoprolol**

Metoprolol is a beta1-selective adrenergic receptor blocker (beta-blocker) (Hazard-Vallerand & Sanoski, 2018). At high doses, it can also inhibit beta2-adrenoreceptors (Hazard-Vallerand & Sanoski, 2018). This beta-blocking activity leads to a reduction in heart rate, cardiac output, and blood pressure (Hazard-Vallerand & Sanoski, 2018). Metoprolol is used to treat mild to moderate hypertension, acute myocardial infarction – reduce cardiovascular mortality, angina, New York Heart Association class II, III, heart failure, and cardiomyopathy (Hazard-Vallerand & Sanoski, 2018). Common side effects of Metoprolol include insomnia, dizziness, hypotension, palpitations, nausea, vomiting, diarrhea, cramping, hiccups (Hazard-Vallerand & Sanoski, 2018). Serious side effects include bradycardia, cardiac arrest, chest pain, pulmonary edema, and bronchospasm (Hazard-Vallerand & Sanoski, 2018). It is important for nurses to monitor blood pressure during the initial treatment and periodically after, and to notify the prescriber if there are any significant changes in the patient’s pulse rate (Hazard-Vallerand & Sanoski, 2018). It is also very important to do a baseline of renal and hepatic lab values/function and evaluate the therapeutic response (Hazard-Vallerand & Sanoski, 2018). The goal of treatment is to have a decreased blood pressure after one to two weeks, and decreased anginal pain (Hazard-Vallerand & Sanoski, 2018). Administered orally, Metoprolol has a peak of two to four hours, and a duration of 13-19 hours (Hazard-Vallerand & Sanoski, 2018). The oral bioavailability has been estimated at around 50%, around 10% of metoprolol is found in the plasma and is bound to albumin (Hazard-Vallerand & Sanoski, 2018). Metoprolol is metabolized in the liver by the CYP2D6 enzyme system and is excreted in the urine (Hazard-Vallerand & Sanoski, 2018). It is important to educate the patient to take the medication at the same time each day, do not double dose or skip doses, and to take any missed doses as soon as possible (up to 8 hours before the next dose) (Hazard-Vallerand & Sanoski, 2018).

**Ondansetron**

Ondansetron is used for the prevention of nausea and vomiting (Hazard-Vallerand & Sanoski, 2018).It prevents nausea and vomiting by blocking serotonin peripherally, centrally, and in the small intestine. A proper assessment of the patient is required during use of this medication including the absence of nausea and vomiting, signs of hypersensitive reaction – rash and bronchospasm – shuffling, tremors, grimacing, and rigidity (Hazard-Vallerand & Sanoski, 2018). It is important for the nurse to evaluate the therapeutic response of the medication to ensure the absence of nausea and vomiting (Hazard-Vallerand & Sanoski, 2018). Common side effects of Ondansetron include headaches, gastrointestinal upset (diarrhea, constipation, abdominal pain), rash, wound healing difficulties, shivering, hypoxia, and fever (Hazard-Vallerand & Sanoski, 2018). A serious side effect is bronchospasm, although it is rare (Hazard-Vallerand & Sanoski, 2018). Ondansetron has a mean elimination half-life of 3.5 to 4.7 hours with an onset of 30 minutes (Hazard-Vallerand & Sanoski, 2018). It is absorbed from the gastrointestinal tract, with some first-pass metabolism (Hazard-Vallerand & Sanoski, 2018). The bioavailability in healthy patients taking 8 milligram tablets is approximately 56% (Hazard-Vallerand & Sanoski, 2018). The bioavailability may be slightly enhanced when taken with food (Hazard-Vallerand & Sanoski, 2018). Ondansetron binds to plasma proteins and distributes into erythrocytes (Hazard-Vallerand & Sanoski, 2018). It is extensively metabolized in the liver and 45-60% of the drug is excreted in the urine (Hazard-Vallerand & Sanoski, 2018). It is important for the nurse to educate the patient to take the medication with a whole glass of water and report any negative side effects – such as gastrointestinal disturbances (diarrhea, constipation, discomfort, nausea, vomiting), and headaches requiring analgesic use (Hazard-Vallerand & Sanoski, 2018).

**Levothyroxine**

Levothyroxine is used for hypothyroidism, thyroid hormone replacement, congenital hypothyroidism, some types of thyroid cancer, and pituitary thyroid-stimulating hormone (TSH) suppression (Hazard-Vallerand & Sanoski, 2018). The action of this medication originates from the thyroid hormone receptors (Hazard-Vallerand & Sanoski, 2018). Levothyroxine increases the metabolic rate and controls protein synthesis which causes an increase in cardiac output, renal blood flow, oxygen consumption, temperature, blood volume, and development on a cellular level (Hazard-Vallerand & Sanoski, 2018). Common side effects of this medication include anxiety, insomnia, headaches, nausea, gastrointestinal upset, changes in appetite (either an increase or a decrease), weight loss, and sweating (Hazard-Vallerand & Sanoski, 2018). Serious side effects are thyroid storm and cardiac arrest (Hazard-Vallerand & Sanoski, 2018). Nurses need to perform proper assessment of the patient including blood pressure and pulse periodically during treatment, and assess thyroid hormones – t3, t4, and free thyroxine index (decreased) (Hazard-Vallerand & Sanoski, 2018). Additionally, check for bleeding or bruising as this may suggest that the patient needs a decreased dose of anticoagulants (Hazard-Vallerand & Sanoski, 2018). There may be cardiac changes in those who are receiving high or rapid doses (Hazard-Vallerand & Sanoski, 2018). Bone loss may occur during long-term use, so it is important to obtain a baseline of bone density and periodically thereafter (Hazard-Vallerand & Sanoski, 2018). Taken orally, Levothyroxine has an onset of 24 hours, and a half-life of 3 to 4 days (Hazard-Vallerand & Sanoski, 2018). The main site of absorption is in the small intestine – the jejunum and ileum (Hazard-Vallerand & Sanoski, 2018). Levothyroxine’s bioavailability is 70 to 80% and may be higher in patients with hyperthyroidism (Hazard-Vallerand & Sanoski, 2018). This drug has a limited volume of distribution – around 14.7 litres in patients with hyperthyroidism (Hazard-Vallerand & Sanoski, 2018). This amount is approximately equivalent to the volume of extracellular fluid volume found in the body (Hazard-Vallerand & Sanoski, 2018). Levothyroxine is bound to plasma protein (Hazard-Vallerand & Sanoski, 2018). For the metabolism of Levothyroxine, the main route for T4 involves the removal of iodine by deiodinase enzymes – once deionized T4 is metabolized to T3, then T2, and eventually T1 (Hazard-Vallerand & Sanoski, 2018). It is important to educate the patient to report any irritability, anxiety, and excitability as this may be indicative of an overdose. Avoid over the counter medications with iodine, and administer all antacids, calcium, and iron products from 4 hours before or after administration of Levothyroxine (Hazard-Vallerand & Sanoski, 2018). It is also important to educate patients that this medication is used to control symptoms, it does not cure hypothyroidism (Hazard-Vallerand & Sanoski, 2018).

**Valproic Acid**

Valproic acid is an anticonvulsant and a vascular headache suppressant (Skidmore-Roth, 2019). It belongs to the chemical class of Carboxylic acid derivatives (Skidmore-Roth, 2019). Valproic acid increases levels of GABA – a neurotransmitter in the central nervous system – to decrease seizure activity and incidence of manic episodes (Skidmore-Roth, 2019; Hazard-Vallerand & Sanoski, 2018). Valproic acid is used as adjunct therapy to treat the patients bi-polar disorder and associated complications such as mania (Skidmore-Roth, 2019). Valproic acid is readily absorbed via oral administration and is rapidly distributed into the plasma and extracellular fluid (Hazard-Vallerand & Sanoski, 2018). The rate of valproic acid distribution may vary depending on the formulation of administration, such as a liquid or capsule; the conditions of use, such as fasting or postprandial; and the method of administration, such as a crushed or whole capsule (FDA, 2009). However, these conditions should not be of significant clinical importance if the patient maintains prolonged use (FDA, 2009). Valproic acid has a 90% bioavailability however, the protein binding is reduced in elderly patients, patients with chronic hepatic disease or renal impairment, or in the presence of other drugs (FDA, 2009). Furthermore, valproic acid is highly protein bound and can displace other medications such as warfarin, phenytoin, and tolbutamide (FDA, 2009). Valproic acid has a 6-16-hour half-life and is metabolized by the liver (Skidmore-Roth, 2019). This medication is excreted by the kidneys (Skidmore-Roth, 2019), which can be reduced in older adult populations due to decreased renal efficiency (FDA, 2009). The common side effects include sedation, drowsiness, agitation, dizziness, insomnia, peripheral edema, visual disturbance, pancreatitis, diarrhea, nausea, constipation, anorexia, and rash (Skidmore-Roth, 2019; Hazard-Vallerand & Sanoski, 2018). Assessment of the therapeutic effect on bipolar disorder includes assessment of mood, ideation, and behaviour; obtain a CBC during long-term therapy; black box warning for hepatotoxicity, monitor hepatic function including AST, ALT, and bilirubin; black box warning for pancreatitis which can occur anytime during treatment or up to seven years following discontinuation of medication use therefore it is important to monitor for abdominal pain, vomiting, and anorexia (Skidmore-Roth, 2019; FDA, 2009; Hazard-Vallerand & Sanoski, 2018). Instruct the patient to take the medication as directed, do not discontinue abruptly after long-term use, and to take the medication as soon as possible if a dose is missed (Skidmore-Roth, 2019). It is important to report all side effects to a medical professional immediately (Skidmore-Roth, 2019). In addition, it is important for the patient and family to notify a health care professional immediately of thoughts of suicide or dying, suicidal attempts or ideations, worsened depression or anxiety, and panic attacks (Hazard-Vallerand & Sanoski, 2018).

**Dalteparin**

Dalteparin is an anticoagulant that belongs to the chemical class of low-molecular-weight heparin (Skidmore-Roth, 2019). Dalteparin potentiates an inhibitory effect of antithrombin on Factor Xa and thrombin (Skidmore-Roth, 2019). It is indicted for the prevention of deep vein thrombosis (DVT) or pulmonary embolism (PE) post-surgery (Skidmore-Roth, 2019). Dalteparin is rapidly absorbed after subcutaneous injection administration with an 87% bioavailability (Skidmore-Roth, 2019). The distribution of dalteparin is unknown however, the duration of onset lasts 1-2 hours and the peak concentrations are reached within 2-4 hours (Skidmore-Roth, 2019; Dunn & Jarvis, 2000). Dalteparin is excreted by the kidneys which can be reduced in older adult populations due to decreased renal efficiency (FDA, n.d.), with the elimination half-life being 3-5 hours (Dunn & Jarvis, 2000). Common side effects of Dalteparin include hypersensitivity, dizziness, bleeding, and temporary increase of liver enzymes (Skidmore-Roth, 2019). It is important to assess blood studies during treatment including assessing for bleeding or hemorrhage in stool and around insertion sites; and assess for hypersensitivity and notify prescriber immediately if rash, urticaria, or fever occurs (Skidmore-Roth, 2019; Hazard-Vallerand & Sanoski, 2018). Advise the patient to avoid over the counter (OTC) medications that contain aspirin or other anticoagulants (Skidmore-Roth, 2019). It is imperative that the patient reports any signs of bleeding, unusual bruising, or fever (Skidmore-Roth, 2019).

**Laboratory Results**

**White Blood Cell (WBC)**

The normal range for white blood cells (WBC) is 4.0-10.0x10^9 per litre (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s WBC level on January 15, 2020 was high—16.3. A possible reason for this increased level is infection. WBC’s are important for the initiation of the body’s immune system/defense against infection and it also maintains the body’s defense system (Pagana, Pagana, & Pike-MacDonald, 2019). Dehydration can also cause increased WBC levels due to stress coupled with hemoconcentration (Pagana, Pagana, & Pike-MacDonald, 2019). Levels of WBC can also be influenced by thyroid hormones and can be a sign of thyroid storm (Pagana, Pagana, & Pike-MacDonald, 2019). WBCs help fight infections by attacking bacteria, viruses, and germs that invade the body (Pagana, Pagana, & Pike-MacDonald, 2019). Elevated WBCs indicate infection – she has acute pancreatitis so these elevated levels should be expected.

**Blood Urea Nitrogen (BUN)**

The normal range for blood urea nitrogen (BUN) is 2.5 to 6.1mmol per litre (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s BUN level on January 15, 2020 was high—6.2mmol/L. Blood urea nitrogen is an estimate/measurement of renal function and the glomerular filtration rate and can also be used as a measurement of liver function (Pagana, Pagana, & Pike-MacDonald, 2019). Urea is made in the liver and is the final product of digestion and protein metabolism (Pagana, Pagana, & Pike-MacDonald, 2019). Those with elevated BUN levels are azotemic. Interfering factors that could potentially contribute to the elevation of BUN levels include high-protein diets or tube feeds, gastrointestinal bleeding, and dehydration (Pagana, Pagana, & Pike-MacDonald, 2019). The result could indicate decreased kidney function or possible heart failure (Pagana, Pagana, & Pike-MacDonald, 2019. It is also possible that her current tube feed diet is too high in protein. The patient is not on any medications that could cause increased levels of BUN.

**Creatinine**

The normal range for creatinine is 58 to 110mmol per litre (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s creatinine level on January 15, 2020 was low—37mmol/L. Creatinine is excreted by the kidneys and is directly related to renal execratory functioning (Pagana, Pagana, & Pike-MacDonald, 2019). Creatinine levels are also a reflection of the glomerular filtration rate (Pagana, Pagana, & Pike-MacDonald, 2019). Decreased levels could be due to debilitation and a decrease in muscle mass (Pagana, Pagana, & Pike-MacDonald, 2019). Low levels of creatinine can also be related to low muscle mass or a decrease in muscle mass, malnutrition, or liver disease (Pagana, Pagana, & Pike-MacDonald, 2019). This client has a rapid decline in her mobility status and has regressed to a state where she is not getting out of bed – this, coupled with the fact that she does not exhibit other classic signs of liver failure is why it would be expected that her muscle atrophy is the likely cause of her low levels.

**Red Blood Cell (RBC)**

The normal range for red blood cells (RBC) in females is 4.2-5.4x1012/L (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s RBC level on January 15, 2020 was low—3.2x1012/L.The RBC count measure the total number of circulating RBC’s (Pagana, Pagana, & Pike-MacDonald, 2019). RBC’s, which contain hemoglobin, are the primary cellular component of blood involved in the transport of oxygen (O2) to the body cells and carbon dioxide (CO2) from the tissues (Pagana et al., 2019). RBC values are commonly reduced in cases of anemia, particularly iron-deficient anemia (Lewis et al., 2014).

**Hematocrit (Hct)**

The normal range for hematocrit (Hct) in females is 0.37-0.47 volume fraction (37-47%) (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s Hct level on January 15, 2020 was low—0.32%. The hematocrit (Hct) is a measurement that represents the packed cell volume of RBC’s, which is the RBC% in comparison with total blood volume, therefore indicating the percentage of blood volume that is composed of RBC’s (Pagana et al., 2019). Similar to RBC’s, decreased levels of Hct would be suggestive of anemia (Pagana et al., 2019).

**Hemoglobin (Hgb)**

The normal range for hemoglobin (Hgb) in females is 120-160g/L (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s Hgb level on January 15, 2020 was low—103g/L. Hemoglobin levels indicate the total amount of peripheral hemoglobin (Pagana et al., 2019). Hemoglobin is a complex protein-iron compound that binds with oxygen and carbon dioxide therefore, the diagnostics reflect the oxygen-carrying capacity of the blood (Pagan et al., 2019). Decreased hemoglobin concentrations can significantly strain the cardiopulmonary system as the body is unable to maintain sufficient oxygen-carrying capacity to oxygenate the body’s cells (Pagana et al., 2019). This can increase the risk of angina, heart attack, heart failure, and stroke when hemoglobin concentrations become critically low (Pagana et al., 2019).

**Platelets**

The normal range for platelets in older adults is 150-400x109/L (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s platelet level on January 15, 2020 was high—438x109/L. Platelet count is the measurement of circulating platelets available to maintain platelet clotting function (Pagana et al., 2019). Thrombocytosis may be present when platelet counts exceed 400x109/L which is commonly associated with iron-deficiency anemia (Pagana et al., 2019). Platelet aggregation may be abnormal in patients with elevated platelets counts which can cause increased bleeding tendencies (Pagana et al., 2019). Platelet counts that exceed 1000x109/L indicate thrombocythemia which can lead to spontaneous hemorrhage (Pagana et al., 2019).

**Glucose**

The normal range for glucose in older adults is 3.9-6.1mmol/L (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s glucose level on January 15, 2020 was high—12.1mmol/L. A blood glucose test provides a direct measurement of the body’s glucose level and is most commonly used when evaluating the status of a diabetic patient (Pagana, Pagana, & Pike-MacDonald, 2019). Glucose levels are controlled by insulin and glucagon and are secreted by the pancreas (Pagana, Pagana, & Pike-MacDonald, 2019). Many forms of stress such as trauma, surgery, infections, and anesthesia can increase serum levels of glucose in the body (Pagana, Pagana, & Pike-MacDonald, 2019). This patient recently had an esophagectomy to remove the cancer on her esophagus, which can put the body in a state of stress. When the body is in a stressed state, it stimulates the release of catecholamine, which stimulates the release of glucagon causing hyperglycemia (Pagana, Pagana, & Pike-MacDonald, 2019). Certain medications such as tricyclics, beta-adrenergic blocking agents, estrogens, glucagon, lithium, and salicylates can increase serum glucose levels (Pagana, Pagana, & Pike-MacDonald, 2019). She has been prescribed to receive Metoprolol, a beta-adrenergic blocking agent, which can also be a contributing factor to the increase in her serum glucose levels (Pagana, Pagana, & Pike-MacDonald, 2019). The patient was admitted to the hospital for acute pancreatitis; when the pancreatic cells are injured during the inflammation process, the cell’s contents including glucagon, are secreted into the bloodstream, ultimately contributing to the increased glucose levels (Pagana, Pagana, & Pike-MacDonald, 2019).

**Sodium**

The normal range for sodium in older adults is 136-145mmol/L (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s sodium level on January 15, 2020 was low—130mmol/L. Sodium is the major cation in the extracellular space and is a major determinant of extracellular osmolality (Pagana, Pagana, & Pike-MacDonald, 2019). Some factors that could potentially be contributing to this patient’s low levels of sodium include a diet that is deficient in sodium, their high levels of serum glucose, or if they are receiving intravenous fluids that are deficient in sodium (Pagana, Pagana, & Pike-MacDonald, 2019. If the intravenous therapy that the patient is receiving is providing sodium at a level that is less than the body’s natural losses, then the residual sodium in the body becomes diluted, ultimately lowering serum levels of sodium (Pagana, Pagana, & Pike-MacDonald, 2019). The patient’s high glucose levels can contribute to a loss of sodium because hyperglycemia creates an osmotic effect (Pagana, Pagana, & Pike-MacDonald, 2019). The glucose pulls water from the extracellular space and as a result, dilutes the sodium levels (Pagana, Pagana, & Pike-MacDonald, 2019).

**Chloride**

The normal range for chloride in older adults is 98-106mmol/L (Pagana, Pagana, & Pike-MacDonald, 2019). This patient’s chloride level on January 15, 2020 was low—95mmol/L. Chloride is a major extracellular anion and works to maintain electrical neutrality with sodium (Pagana, Pagana, & Pike-MacDonald, 2019). Hypochloremia rarely occurs alone, it occurs with a parallel shift of sodium (Pagana, Pagana, & Pike-MacDonald, 2019).  Aldosterone, which is responsible for increased reabsorption of sodium from the kidney, is also responsible for the reabsorption of chloride (Pagana, Pagana, & Pike-MacDonald, 2019). Chloride levels normally change in direct relationship to sodium (Pagana, Pagana, & Pike-MacDonald, 2019). Hence if serum sodium is decreased, a decreased chloride level can be expected. Due to the unknown cause of the patient’s sodium levels, it is assumed that their low chloride level is a result of their low sodium level.

**Connections**

**Acetaminophen – Pain**

Acetaminophen is used as needed (PRN) for mild to moderate pain. This PRN can be used alone or together with hydromorphone if pain persists or breaks through. Acetaminophen has a limit of 4 gram in a 24-hour period (Hazard-Vallerand & Sanoski, 2018). This client did not require this PRN during her time in within our care.

**Dimenhydrinate – Gastrointestinal**

Dimenhydrinate can have the side effect of anorexia – loss of appetite (Hazard-Vallerand, & Sanoski, 2018). Our client was to receive nothing by mouth (NPO) and receiving enteral nutrition, ISO source 1.5 at 60ml/hr., so anorexia as a side effect was not of great concern. In addition, being a PRN, the risk of anorexia is further reduced as the dose is not on a continuous dosing schedule. However, it is our belief that prolonged use of dimenhydrinate throughout her recovery will contribute negatively when transitioning her off of the tube feed and back to oral nutrition.

**Dimenhydrinate – Neurological**

Another side effect of dimenhydrinate is dizziness (Hazard-Vallerand, & Sanoski, 2018). Again, as a PRN, the concern for dizziness is reduced. The risk presented by dizziness includes falls which is further counteracted by the fact that this client has been bed ridden for the past few days. Looking forward, it has been acknowledged that as this client begins to recover, she may be adversely affected when beginning to ambulate again as the dimenhydrinate *may* cause an issue.

**Hydromorphone – Pain**

The indicated use Hydromorphone is for moderate to severe pain (Hazard-Vallerand, & Sanoski, 2018). Just as with acetaminophen, the client did not require this PRN medication. The client denied pain during her time under our care and thus did not require this medication.

**Hydromorphone – Gastrointestinal**

Opioid analgesic medications have the common side effect of constipation due to their effect on gastrointestinal motility (Hazard-Vallerand, & Sanoski, 2018). This client has not been using this PRN during our tenure with her and thus this side effect was not seen. Hydromorphone does pose a serious risk for constipation should she require it in the future; the effect of her being bedridden would further exacerbate this effect and thus this connection is important to make.

**Hydromorphone – Neurological**

Hydromorphone as an opioid analgesic has a depressive effect on the central nervous system and this depression can cause sedative side effects (Hazard-Vallerand, & Sanoski, 2018). Again, since this client did not require any pain intervention while under our care, we did not see this side effect occur. It is during her future recovery that we could anticipate sedation being a negative side effect; sedation would negatively impact her ability to ambulate, as well as slowing/impeding her recovery.

**Hydromorphone – Cardiovascular**

Hydromorphone and other opioid analgesic medications can cause hypotension/ orthostatic hypotension through action of systemic vasodilation (Hazard-Vallerand, & Sanoski, 2018). While we did not have to use this medication during our time with the client, the use of this medication as she recovers poses an issue. Hypotension mixed with her atrial fibrillation and decreasing mobilization is a concoction for prolonged recovery time and the possibility for increased need for medication intervention.

**Metoprolol – Atrial Fibrillation**

Metoprolol is a selective beta-blocker that is used to treat hypertension, myocardial infarction, and reduce angina symptoms (Hazard-Vallerand & Sanoski, 2018). Blocking beta-1 receptors leads to a reduction in heart rate, cardiac output, and the reduction of blood pressure (Hazard-Vallerand, 2018). This patient is currently on metoprolol for her atrial fibrillation – a condition that is characterized as a high frequency excitation that results in both unsynchronized atrial contraction and irregular ventricular excitation (Iwasaki, Nishida, Kato, & Nattel, 2011). Metoprolol could be used prophylactically in this patient to prevent a myocardial infarction and hypertension.

**Metoprolol – Gastrointestinal**

Metoprolol can negatively affect the gastrointestinal system because nausea and vomiting are common side effects of this drug (Hazard-Vallerand & Sanoski, 2018). It can also cause gastrointestinal disturbances such as cramping and diarrhea (Hazard-Vallerand & Sanoski, 2018). This patient may experience nausea or vomiting, so she has PRN medications (Ondansetron and Dimenhydrinate) for these potential side effects caused by Metoprolol.

**Ondansetron – Gastrointestinal**

Ondansetron is an antiemetic that works in the small intestine by blocking serotonin and is used for the prevention of nausea and vomiting (Hazard-Vallerand & Sanoski, 2018). This medication was prescribed as a PRN medication to prevent nausea as the patient has undergone surgery and other procedures and may need an antiemetic. This client may feel nauseous due to the pain from her acute pancreatitis. Furthermore, ondansetron may cause gastrointestinal disturbances as a side effect.

**Ondansetron – Neurological**

A common side effect of Ondansetron is a headache that requires analgesia (Hazard-Vallerand & Sanoski, 2018). Our client had PRN’s for pain, but she did not require them while she was under our care. However, the client should know that this side effect is common, and she may require an analgesia if this side effect occurs in the future.

**Levothyroxine – Endocrine and Hypothyroidism**

Levothyroxine acts as a thyroid hormone replacement and acts on her endocrine system (Hazard-Vallerand & Sanoski, 2018). This client was on levothyroxine because of her hypothyroidism, an endocrine disorder that causes there to be an inadequate level of thyroid hormones (Bianco, Jonklaas, & Peeters, 2017).

**Blood Urea Nitrogen – Jejunostomy tube**

Urea is made in the liver and is the final product of digestion and protein metabolism (Skidmore-Roth, 2019). This could indicate decreased kidney function although the patient is not displaying any further signs of this. The patient was on a tube feed diet and it is possible that the increased BUN levels are due to her current tube feed diet being too high in protein. However, we do not have any further lab values for this patient so we cannot see if her diet was changed or if that affected her BUN levels.

**Creatinine – Musculoskeletal**

Decreased creatinine levels could be due to debilitation and a decrease in muscle mass (Pagana, Pagana, & Pike-MacDonald, 2019). The patient was bed bound and not moving so it is assumed that these increased levels could be due to muscle deterioration. We do not have any further lab values or information due to her transfer, but if she remains bed bound there could be further muscle deterioration and her creatinine levels could continue to decrease unless she began to mobilize more often and build up her muscle mass again.

**White Blood Cell – Endocrine**

The patient has elevated WBC levels – this could signify infection (Pagana, Pagana, & Pike-MacDonald, 2019). WBCs help fight infections by attacking bacteria, viruses, and germs that invade the body. The increase in WBC could be connected to her endocrine system, and due to the fact that the patient currently has acute pancreatitis, so these elevated levels are to be expected. Unfortunately, the lab values from January 15th are the only values obtained as she was transferred out of the medical-surgical unit; we would expect these levels to remain high until this issue is dealt with.

**White Blood Cell – Esophagectomy**

The patient had esophageal cancer which was removed in December 2019. Her elevated WBCs may be due to her surgery to remove the esophageal cancer a month prior. As she heals from her surgery, it could be expected that her WBCs would return back to normal levels, however she currently has pancreatitis which would also affect these levels.

**Esophagectomy — Glucose**

Many forms of stress such as trauma, surgery, infections, and anesthesia can increase serum levels of glucose in the body (Pagana, Pagana, & Pike-MacDonald, 2019). This patient recently had an esophagectomy to remove the cancer on her esophagus, which can put the body in a state of stress. When the body is in a stressed state, it stimulates the release of catecholamine, which stimulates the release of glucagon causing hyperglycemia (Pagana, Pagana, & Pike-MacDonald, 2019).

**Metoprolol — Glucose**

Certain medications such as tricyclics, beta-adrenergic blocking agents, estrogens, glucagon, lithium, and salicylates can increase serum glucose levels (Pagana, Pagana, & Pike-MacDonald, 2019). This is due to the fact that beta-blockers can reduce the sensitivity of receptors to insulin, therefore raising circulating levels of glucose (Diabetes.co.uk., 2019). She has been prescribed to receive Metoprolol, a beta-adrenergic blocking agent, which can be a contributing factor to the increase in her serum glucose levels (Pagana, Pagana, & Pike-MacDonald, 2019).

**Pancreatitis — Glucose**

The patient was admitted to the hospital for acute pancreatitis; when the pancreatic cells are injured during the inflammation process, the cell’s contents including glucagon, are secreted into the bloodstream, ultimately contributing to the increased glucose levels (Pagana, Pagana, & Pike-MacDonald, 2019).

**Latanoprost — Glaucoma**

Latanoprost is part of the functional class of Antiglaucoma agents, specifically the prostaglandin agonists (Skidmore-Roth, 2019). It is used as treatment for intraocular pressure in patients with open-angle glaucoma and are otherwise unresponsive to other intraocular lowering medications (Skidmore-Roth, 2019). Latanoprost is a prodrug of a prostaglandin known as prostaglandin F2a· (Lilley, Collins, Snyder, & Swart, 2016). When Latanoprost is administered, it is converted via hydrolysis to prostaglandin F2a1 which decreases intraocular pressure (Lilley, Collins, Snyder, & Swart, 2016). Prostaglandins work to decrease intraocular pressure by increasing the outflow of fluid between the uvea, the sclera, and the trabecular meshwork (Lilley, Collins, Snyder, & Swart, 2016).

**Glucose — Sodium**

It was stated in the notes under diagnostics that according to the physician, the reason for the abnormality of sodium is unknown. Given the data that was gathered about the patient’s status, a factor that could potentially be contributing to this patient’s low level of sodium could be their high levels of serum glucose. The patient’s high glucose levels can contribute to a loss of sodium because hyperglycemia creates an osmotic effect (Pagana, Pagana, & Pike-MacDonald, 2019). The glucose pulls water from the extracellular space and as a result, dilutes the sodium levels (Pagana, Pagana, & Pike-MacDonald, 2019).

**Sodium — Chloride**

Chloride is a major extracellular anion and works to maintain electrical neutrality with sodium (Pagana, Pagana, & Pike-MacDonald, 2019). Hypochloremia rarely occurs alone, it occurs with a parallel shift of sodium (Pagana, Pagana, & Pike-MacDonald, 2019).  Aldosterone is responsible for increased reabsorption of sodium from the kidney, as well as the reabsorption of chloride (Lewis, 2018). Because chloride levels change in a direct relationship with sodium, if sodium levels are decreased, a decrease in chloride levels will be evident (Lewis, 2018). Due to the unknown cause of the patient’s sodium levels, it is assumed that this patient’s low chloride level is a result of their low sodium level.

**Hypothyroidism — Glucose**

Thyroid hormones are necessary for the body to metabolize carbohydrates, as well as for the pancreas to synthesize and secrete insulin (Wang, 2013). If the pancreas is unable to synthesize and secrete insulin, there will be an increased amount of circulating glucose unable to access target tissues (Wang, 2013). Hypothyroidism may cause a drop in insulin levels which causes an increase in circulating glucose levels (Wang, 2013).

**Esomeprazole — Musculoskeletal**

Long term therapy of Esomeprazole is associated with a decrease in bone mineral density which is a characteristic of osteoporosis (Thong, Ima-Nirwana, & Chin, 2019). Osteoporosis leads to decreased bone strength and an individual’s susceptibility to fractures (Thong, Ima-Nirwana, & Chin, 2019). This medication therapy in combination with the fact that this patient is bed ridden further increases the risk of fractures (Takata & Yasui, 2001). Being bed ridden inhibits osteoblast-mediated bone formation and speeds up osteoclast-mediated bone resorption leading to bone loss, or disuses osteoporosis. This further increases the risk of fractures (Takata & Yasui, 2001).

**Esomeprazole — Jejunostomy tube**

Patients requiring enteral nutrition may also be in need of acid-suppressing therapy with proton pump inhibitors (Wensel, 2009). This patient is receiving isosource via her J-tube insertion. Esomeprazole may be used prophylactically in combination with the J-tube for stress ulcers by inhibiting gastric acid secretion, neutralizing gastric acid or protecting the mucosal lining of the stomach (Surgical Critical Care, 2017). Additionally, due to her recent esophagectomy, the use of Esomeprazole inhibits the gastric acid secretion to ultimately reduce the risk of GERD to prevent complications of the healing process post-surgery (Skidmore-Roth, 2019).

**Dalteparin – Atrial Fibrillation – Deep Vein Thrombosis (DVT) Prophylaxis**

Dalteparin is a low-molecular weight heparin that is indicated for the prevention of thromboembolic complications such as deep vein thrombosis (DVT) or pulmonary embolism (PE) in surgical or medical patients (Skidmore-Roth, 2019). It’s anticoagulatory effects serve to reduce the incidence of DVT or PE in post-surgical patients and/or in patients with restricted mobility during acute illness (Skidmore-Roth, 2019). The goal of anticoagulation therapy for venous thromboembolism prophylaxis is to prevent the propagation of a blood clot, reduce the development of new thrombi, and to reduce the risk of embolization (Lewis et al., 2014). Atrial fibrillation, a dysrhythmia, may cause a thrombi to form in the atria as a result of blood stasis (Lewis et al., 2014). This thrombi may develop to become an embolized clot which could travel to other parts of the body, including the brain, which would result in a stroke (Lewis et al., 2014). Patients with atrial fibrillation may be prescribed a medicine regime for acute or long-term anticoagulation therapy to reduce the risk of producing an embolized clot (Lewis et al., 2014). It is plausible to suggest that during the patients acute hospital care for treatment of other conditions, that the prescribed dalteparin for DVT prophylaxis may also aid in reducing the incidence of thromboembolic occurrences due to atrial fibrillation.

**Musculoskeletal – Cardiovascular**

Deep venous thrombosis (DVT) is a disorder in which there is a thrombus formation in one of the deep veins such as the iliac or femoral vein (Lewis et al., 2014). Venous thrombosis is caused by any of the three following factors: venous stasis, damage of the endothelium, or hypercoagulability (Lewis et al., 2014). Venous stasis commonly occurs due to muscular inactivity because normal blood flow within the venous system is dependent on recurrent actions of the muscles in the extremities (Lewis et al., 2014). Patients that have had surgical procedures and are bed-bound or, are immobile for prolonged periods of time due to other health conditions, are at increased risk for DVT resulting from venous stasis (Lewis et al., 2014). Due to the patients recent surgical procedures, increased age, and current health conditions that have resulted in hospitalization, the patient is currently immobile. Because of this, it is plausible to suggest that our patient is at increased risk for DVT due to valve stenosis (Lewis et al., 2014).

**Dalteparin – Cardiovascular**

Interactions with aspirin, oral anticoagulants, platelet inhibitors, nonsteroidal anti-inflammatory analgesics (NSAIDs), salicylates, thrombolytics, and some cephalosporins can increase the risk of bleeding when taking Dalteparin (Skidmore-Roth, 2019). Additionally, interactions with angelica, chamomile, dandelion, garlic, ginger, gingko, and horse chestnut also increase the risk of bleeding if taken with Dalteparin (Skidmore-Roth, 2019). With this in mind, it is important to assess blood studies frequently throughout treatment, particularly focusing on Hct, Hgb, platelets, and anti-Xa (Skidmore-Roth, 2019). It is also important for nurses to frequently assess for bleeding and hemorrhage (Skidmore-Roth, 2019). Bleeding such as bleeding gums, black tarry stool, epistaxis, petechia, and hematuria may indicate that there is an adverse reaction and the prescriber should be notified immediately (Skidmore-Roth, 2019).

**Valproic Acid – Pancreatitis**

Valproic acid has a black box warning for pancreatitis which may be fatal in some cases (Skidmore-Roth, 2019). It is important to inform the patient to report immediately if they experience any symptoms of nausea, vomiting, anorexia, or abdominal pain which may occur at any point during the treatment (Skidmore-Roth, 2019). These symptoms may also occur at any point within seven months to seven years following the discontinuation of drug therapy (Skidmore-Roth, 2019).

**Valproic Acid – Neurological**

Valproic acid increases levels of GABA, a neurotransmitter in the central nervous system, to decrease seizure activity and incidence of manic episodes of bipolar disorder (Skidmore-Roth, 2019; Hazard-Vallerand & Sanoski, 2018). Possible neurological side effects of valproic acid include agitation, dizziness, insomnia, headache, sedation, confusion, ataxia, tremors, and depression (Skidmore-Roth, 2019; Hazard-Vallerand & Sanoski, 2018). Considering the patient’s mental health history, it is important for the nursing staff to asses for suicidal tendencies particularly during early therapy (Hazard-Vallerand & Sanoski, 2018). Additionally, it is recommended to assess the patient for excessive somnolence frequently considering the patient’s age (Hazard-Vallerand & Sanoski, 2018).

**Valproic Acid – Blood Urea Nitrogen**

Valproic acid has a black box warning for hepatoxicity (Skidmore-Roth, 2019) and therefore, a patient on this medication should be monitored closely especially during the initial six-month period of drug therapy (Hazard-Vallerand & Sanoski, 2018). It is recommended that hepatic functions are frequently monitored, specifically AST, ALT, and bilirubin, prior to initiation of medication therapy as well as periodically throughout (Skidmore-Roth, 2019; Hazard-Vallerand & Sanoski, 2018). It is further recommended to monitor for fever, anorexia, vomiting, lethargy, and jaundice of the skin and eyes throughout drug therapy as these may be indications of hepatotoxicity (Skidmore-Roth, 2019).

**Valproic Acid – Gastrointestinal**

Common gastrointestinal (GI) side effects of Valproic Acid include abdominal pain, anorexia, diarrhea, indigestion, nausea, and vomiting (Hazard-Vallerand & Sanoski, 2018). Other possible GI side effects include constipation, increased appetite, dyspepsia, stomatitis, weight gain, and dry mouth (Skidmore-Roth, 2019; Hazard-Vallerand & Sanoski, 2018).

**Hypothyroidism –Red Blood Cell – Anemia**

The red blood cell (RBC) count measures the total number of circulating RBC’s (Pagana, Pagana, & Pike-MacDonald, 2019). RBC’s, which contain hemoglobin, are the primary cellular component of blood involved in the transport of oxygen (O2) to the body cells and carbon dioxide (CO2) from the tissues (Pagana et al., 2019). RBC values tend to decrease with age however, when the value is less than 10% of the normal range then the patient is diagnosed anemic (Pagana et al., 2019). The patients diagnostic results indicate an iron deficiency otherwise known as anemia (Pagana et al., 2019). Additionally, low RBC counts are common in clients with hypothyroidism, a condition in which the thyroid hormones levels are below the normal value (Pagana et al., 2019; Lewis et al., 2014). The thyroid hormone, in addition to various endocrine hormones, aids in stimulating RBC production (Pagana et al., 2019). These diagnostic results may also indicate that the patients medication for hypothyroidism may not be performing to optimal therapeutic level (Pagana et al., 2019).

**Red Blood Cell – Hematocrit – Anemia**

The hematocrit (Hct) is a measurement that represents the packed cell volume of RBC’s, which is the RBC percentage in comparison with total blood volume, therefore indicating the percentage of blood volume that is composed of RBC’s (Pagana et al., 2019). Hct values are typically lower in women and levels continue to decrease slightly with age (Pagana et al., 2019). Abnormal values would correlate with the same pathological state as the abnormal RBC count, further indicating anemia and reflecting the diagnosis of hypothyroidism (Pagana et al., 2019). Moreover, both oxygen demand and bone marrow cell production are decreased during hypothyroidism, which ultimately results in decreased hematocrit levels (Lewis et al., 2014). It is plausible to infer that the patient’s body is not producing enough RBC’s to meet the body’s needs which may be owing to the extensive health history including hypothyroidism and anemia (Pagana et al., 2019). In older adults, a blood transfusion may be recommended if the Hct levels are below 0.30 volume fractions (Pagana et al., 2019).

**Hemoglobin – Anemia – Red Blood Cell – Hematocrit – Hypothyroidism**

Hemoglobin levels indicate the total amount of peripheral hemoglobin (Pagana et al., 2019). Hemoglobin is a complex protein-iron compound that binds with oxygen and carbon dioxide therefore, the diagnostics reflect the oxygen-carrying capacity of the blood (Pagan et al., 2019). Hemoglobin concentrations closely reflect the hematocrit and RBC values, which similarly, is typically decreased in women and the elderly (Pagan et al., 2019). Abnormal hemoglobin levels reflect the same pathological states that abnormal hematocrit and RBC levels do, including anemia (Pagana et al., 2019). Additionally, a metanalysis study revealed that thyroid dysfunction, resulting in hypothyroidism, was also associated with low hemoglobin levels (Wopereis et al., 2018). Decreased hemoglobin concentrations can significantly strain the cardiopulmonary system as the body is unable to maintain sufficient oxygen-carrying capacity to oxygenate the body’s cells (Pagana et al., 2019). This can increase the risk of angina, heart attack, heart failure, and stroke when hemoglobin concentrations become critically low (Pagana et al., 2019). An elderly adult may be recommended a blood transfusion when hemoglobin concentrations are 10g/L below expected ranges (Pagana et al., 2019).

**Platelets – Anemia**

Platelet count is the measurement of circulating platelets available to maintain platelet clotting function (Pagana et al., 2019). Platelets help to initiate the coagulation factor cascade and therefore, are essential to blood clotting (Pagana et al., 2019). Thrombocytosis may be present when platelet counts exceed 400X109/L which is commonly associated with iron-deficient anemia (Pagana et al., 2019). Iron is not necessary for the production of platelets and therefore, platelets can respond to events in the presence of iron deficiency (Pagana et al., 2019). The patients lab diagnostic studies suggest that the patient may have thrombocytosis, a disorder characterized by excess platelets, which is associated with anemia (Pagana et al., 2019; Lewis et al., 2014).

**Hypothyroidism – Anemia**

Two common features of hypothyroidism are anemia and iron deficiency (Wopereis et al., 2018). A metanalysis study observed a cross-sectional relationship between thyroid function and anemia, in which patients with overt hypothyroidism had also been diagnosed anemic (Wopereis et al., 2018). Additionally, this same study revealed that a reduced thyroid function increased the overall risk of developing anemia within the near future (Wopereis et al., 2018). It has been suggested that hypothyroidism may be related to anemia due to the poor production of healthy erythrocytes (Wopereis et al., 2018). This is because thyroid hormones have been shown to promote erythropoiesis by influencing renal production of erythropoietin and by facilitating iron transport and utilization within the body (Wopereis et al., 2018). To further support this, Lewis et al. (2014) indicates that erythrocyte production is influenced by endocrine hormones such as thyroxine, corticosteroids, and testosterone, therefore, hypothyroidism is often associated with anemia. Furthermore, the malnutrition and malabsorption that results from hypothyroidism, may further explain the co-occurrence of hypothyroidism and anemia (Wopereis et al., 2018). Malnutrition and malabsorption are common adaptive responses by the body due to energy deficits (Wopereis et al., 2018). These adaptive responses contribute to deficits in the micronutrients that are critical to erythropoiesis, including iron, folate, and vitamin B12 (Wopereis et al., 2018). As previously mentioned, iron-deficient anemia is the most common form of anemia (Wopereis et al., 2018) and most relevant to our patient. Given this information, it is plausible to assume that the patient’s chronic condition of hypothyroidism is connected to the iron-deficient anemia due to decreased production of erythropoietin, poor facilitation of iron transport, the malabsorption of iron, and malnutrition (Wopereis et al., 2018).

**Strategies, Implementation, and Integration of Collaboration**

**Strategy One**

With concern to her tachycardia, we implemented vital assessments every two hours and sought a consult from the physician as the situation was quickly escalating beyond our scope of knowledge and practice. Along with the primary nurse, both the most responsible physician (MRP) and resident cardiologist were contacted as her pulse has been unresponsive to treatment and has reached levels of increased concern. This strategy proved to be the best course of action. Our client was seen by both the hospitalist and cardiologist; both of whom decided it would be best to move our client to the Critical Care Unit for closer monitoring and treatment.

**Strategy Two**

In response to the emergence of incontinence and the fact that she was unaware of her incontinence we sought to include a toileting schedule for our client. This strategy is a joint effort with those to follow; it seeks to increase her mobilization and help improve her control over this emergent incontinence. To prepare for our strategy, we consulted with the physiotherapist on 5 South to ensure that it was safe to ambulate our client – he ensured us that she was safe to ambulate. While we did not have the time to see the outcome of this specific intervention, our client was relieved to have a strategy in place to restore some of her independence. The ultimate goal would be to see her return to a state of continence.

**Strategy Three**

Regarding her decreased mobilization and activity level, we implemented a plan to have her sit in a chair or bedside for all meals. Our consult with physiotherapy let us know that this strategy was safe and effective. Again, having been moved to the Critical Care unit, we did not see the end result of our strategy, but having placed it in her plan of care we would hope to see the continuation of the strategy and a marked improvement to her activity level and mobility status.

**Strategy Four**

With the concern with her decrease in mobility, we see a potential problem with skin breakdown; this concern is further complicated by her incontinence. Our strategy includes a few steps: Proper peri-care, use of extra protective cream on red/excoriated skin, and daily Braden scales. These three steps along with our other mobilization strategies will hopefully prevent breakdown of the skin and development of a pressure injury. Our strategies will be proven successful with continued absence of injury.

**Strategy Five**

While we cannot diagnose depression; we’ve noted that our client seems increasingly hopeless. She is no longer mobilizing, she has stated that she does not know what is going on with her and that no one has answers for her, she is no longer performing self-care tasks, and lastly, she has become incontinent. Our strategy to combat the feelings of hopelessness is to promote participation and maximizing her control: we want her to ring her call bell every time she needs to use the bedpan or commode, to assist with or perform her own self-care, keep an open dialogue throughout care with our client as she is feeling neglected, and lastly encourage our client to seek additional forms of support and provide what resources we have at our disposal. As with our other strategies, it is impossible to now see if our care plan interventions were successful but, we can speculate that should this strategy be successful we would see an increase in mood/outlook, an increase in self-care participation, and an overall decrease in her personal neglect.

**Strategy Six**

We assessed a slight decrease in air entry bilaterally to the base of her lung fields (confirmed with primary nurse) and along with direct assessment of her lungs, noted that she was almost constantly lying flat. Our strategy was to raise the head of the bed to 30 degrees to promote easier respirations and open up her airways. While our client denied shortness of breath and a cough, we felt it was prudent to ensure that with everything else going on that her respiratory system stayed in top shape; as such, to see success in this strategy would be the continued health of her respiratory system, continued absence of adventitious sounds within the lung beds, and hopefully the return of complete air entry to her bilateral lung field bases.

**Conclusion**

Throughout this case study we have thoroughly discussed our patient’s chronic and episodic health challenges, the pharmacology behind the prescribed medications, and the importance of the patient’s lab values. We have elaborated on the connections between all of the indicated areas in order to better understand our patient as a whole. This case study allowed us to understand how all of these categories are all interconnected and how they affect our patient during the recovery phase. After thoroughly researching and linking the relationships between the medications, health challenges, and lab values, we were able to produce six strategies to implement for the patient to help with her recovery in the future.

**Rationale for Sources**

“Esomeprazole, Oral Capsule (Magnesium).” Healthline.com, 2018, www.healthline.com/health/esomeprazole-oral-capsule#side-effects.

This website is one I used to briefly supplement the knowledge of Esomeprazole in conjunction with the textbook. The knowledge on the website is understandable and is closely monitored by experts who update the articles as new information arises. This website article was used to further gain knowledge of the proton pump inhibitor’s side effects to further elaborate on those from the textbook.

“Nexium (esomeprazole magnesium) Delayed-Release Capsules.” *U.S. Food & Drug Administration*, 2006, https://www.accessdata.fda.gov/drugsatfda\_docs/label/2006/021153s022lbl.pdf

The source was obtained through the Food and Drug Association federal agency that provides recent, reliable, and credible information regarding medications. This source was used to further supplement the knowledge of Esomeprazole, specifically related to the pharmacokinetics and drug interactions. The textbook did not have an adequate amount of information relating to the pharmacokinetics of Esomeprazole therefore this external source was needed to be used.

“Xalatan, Latanoprost ophthalmic solution.” *U.S. Food & Drug Administration,* 2011, [www.accessdata.fda.gov/drugsatfda\_docs/label/2012/020597s044lbl.pdf](http://www.accessdata.fda.gov/drugsatfda_docs/label/2012/020597s044lbl.pdf)

This source was obtained through the Food and Drug Association federal agency that provides recent, reliable, and credible information regarding medications. This source was used to supplement knowledge from the textbook specifically relating to the pharmacokinetics of Latanoprost. Because the textbook provided little information on the absorption, distribution, metabolism, and excretion of the drug, an external source was needed to be used.

“Zopiclone.” *DrugBank.ca,* 2020, [www.drugbank.ca/drugs/DB01198](http://www.drugbank.ca/drugs/DB01198)

**“**pms-Zopiclone.” *Rexall Pharmacy Group,* 2020, www.rexall.ca/articles/view/2763/pms-Zopiclone

“Zopiclone.” *F. A.* *Davis Plus,* 2015, [www.davisplus.fadavis.com/3976/meddeck/pdf/zopiclone.pdf](http://www.davisplus.fadavis.com/3976/meddeck/pdf/zopiclone.pdf)

The online resources used are all credible and reputable. They enhanced the knowledge provided by the textbook by further explaining the pharmacokinetics of Zopiclone. Zopiclone was not adequately explained in the textbook therefore external, online resources were required to further elaborate on the medication.

American Heart Association. (2016). What are the symptoms of atrial fibrillation (afib or af)? Retrieved from <https://www.heart.org/en/health-topics/atrial-fibrillation/what-are-the-symptoms-of-atrial-fibrillation-afib-or-af>

Epidemiology, pathophysiology, and clinical outcomes. *Circulation Research. 120*(9). doi:10.1161/CIRCRESAHA.117.309732

Heart and Stroke Foundation of Canada. (2018). Atrial fibrillation. Retrieved from https://www.heartandstroke.ca/heart/conditions/atrial-fibrillation

Iwasaki, Y., Nishida, K., Kato, T., & Nattel, S. (2011). Atrial fibrillation pathophysiology: Implications for management. *Circulation. 124*(20). doi:10.1161/CIRCULATIONAHA.111.019893

Potter, P. A., Perry, A. G., Stockert, P. A., & Hall, A. M. (2019). *Canadian fundamentals of nursing* (6th ed.). Astle, B. J., & Duggleby, W. (Eds.). Milton, ON: Elsevier Canada

Staerk, L., Sherer, J., Ko, D., Benjamin, E., & Helm, R. (2017). Atrial fibrillation:

These sources were used to supplement the knowledge of Atrial fibrillation. The textbook is sanctioned by Thompson Rivers University and is required for nursing students as the knowledge is detailed, accurate, and credible. The online sources used are all credible and updated with accurate information. There are various associations that are referenced as they are considered to be contributed to by experts on the subject. There websites extensively explain the pathophysiology, prevalence, signs and symptoms, and risk factors of atrial fibrillation.

Brenta, G., Vaisman, M., Sgarbi, J. A., Bergoglio, L. M., Andrada, N. C. de, Bravo, P. P., Orlandi, A. M., & Graf, H. (2013). Clinical practice guidelines for the management of hypothyroidism / Diretrizes clínicas práticas para o manejo do hipotiroidismo. *Arquivos Brasileiros de Endocrinologia & Metabologia, 57*(4), 265–291. doi: 10.1590/S0004-27302013000400003

Chaker, L., Bianco, A. C., Jonklaas, J., & Peeters, R. P. (2017). Hypothyroidism. *The Lancet, 390*(10101), 1550–1562. doi: 10.1016/S0140-6736(17)30703-1

Both of these sources were found using Thompson Rivers University’s Library discover feature, and were further narrowed using exclusive search for scholarly, peer reviewed sources. The use of two articles was to ensure information was consistent between sources as well as ensuring that a complete picture of the disease was formed. Both articles are relevant to the specific disease and are recently published ensuring that the information is up-to-date.

Depakene (valproic acid) Solution Depakene (valproic acid) Capsule, Liquid Filled. (2009, January). Retrieved March 9, 2020, from https://www.accessdata.fda.gov/drugsatfda\_docs/label/2009/018081s047,018082s032lbl.pdf

This source was obtained through the Food and Drug Association federal agency that provides recent, reliable, and credible information regarding medications. Admittedly, this resource is outdated however, it was only used to supplement the information that was already obtained from other resources regarding the medication Valproic Acid. Furthermore, information in this source was constituent with the information obtained within other resources, therefore can be considered accurate and relevant. This article provided in-depth information regarding the pharmacokinetics of Valproic Acid which was necessary to provide a robust description.

Diabetes.co.uk. (2019). Drug induced diabetes. Retrieved from https://www.diabetes.co.uk/drug-induced-diabetes.html

This online resource was used as it is a great reference from the diabetes organization in the United Kingdom. The information from the website used was to supplement the knowledge of the relationship between the use of Metoprolol and how it could impact glucose levels.

Doenges, M. E., Murr, A. C., & Moorhouse, M. F. (2019). *Nursing care plans: Guidelines for individualizing client care across the life span.* Philadelphia, PA: F.A. Davis Company.

This textbook is a sanctioned resource by the Thompson River University School of

Nursing that has been recommended by several professors. This is a credible resource to use for researching potential care plans for our patient. This source thoroughly explains the rationale behind potential nursing care plans, desired outcomes, related health conditions, and the pathophysiology of various health conditions. This was a valuable source for understanding the pathophysiology behind some of our patient’s chronic and episodic health conditions and potential care plans to treat the patient’s conditions.

Dunn C., J., & Jarvis B. (2000). Dalteparin: an update of its pharmacological properties and clinical efficacy in the prophylaxis and treatment of thromboembolic disease. *Drugs, 60*(1), 203–237.

This resource was found using Thompson River University’s Library discover feature, and was further narrowed using exclusive search for scholarly, peer reviewed sources. This resource provided relevant and accurate information regarding the pharmacological properties and clinical efficacy of dalteparin. Admittedly, this article is outdated however, it provided the necessary information regarding the pharmacokinetics of dalteparin. The information obtained from this (2000) article was consistent with the information within the other resources from more recent years. We used this source for supplementary information regarding the distribution of Dalteparin.

FRAGMIN® dalteparin sodium injection. (n.d.). Retrieved March 9, 2020, from https://www.accessdata.fda.gov/drugsatfda\_docs/label/1999/20287s8lbl.pdf

The source was obtained through the Food and Drug Association federal agency that provides recent, reliable, and credible information regarding medications. Unfortunately, this resource did not have a date provided and therefore, was only used to supplement the information that was already obtained from other resources regarding the medication Dalteparin. This article provided information on the elimination process of Dalteparin, which we were unable to obtain from the other sources. This article was required in order to provide a more robust description on the pharmacokinetics of Dalteparin.

Goldberg, S. G. (2019). Narratives of bipolar disorder: Tensions in definitional thresholds. *The Humanistic Psychologist, 47*(4), 359–380. doi: 10.1037/hum0000131.supp

Parker, J. N., & Parker, P. M. (2003). *Bipolar Disorder: A Medical Dictionary, Bibliography, and Annotated Research Guide to Internet References*. Icon Group International, Inc.

Both sources were found using Thompson Rivers University’s Library discover feature, and were further narrowed using exclusive search for scholarly, peer reviewed sources. Again, two sources for information were used to ensure information remained consistent across multiple sources. Parker & Parker (2003) is a dated source admittedly, but we would like to stress the information on bipolar disorder has remained fairly consistent. The major changes come with diagnosis; a subject we are not concerned with. To offset the dated first reference, we made certain to use as recent a reference on the subject as possible. Goldberg (2019) was not only a comprehensive paper on the subject of bipolar disorder but it also reinforced much of the information found by Parker & Parker (2003).

Hazard-Vallerand, A., & Sanoski, C., A., (2018). Davis’s Drug Guide for Nurses (16th ed.). Philadelphia, PA: F.A. Davis Company.

Skidmore-Roth, L. (2019). Mosby’s 2019 Nursing Drug Reference (32nd edition). Littleton, CO: Elsevier Inc.

These drug guides are sanctioned resources by the Thompson River University School of Nursing that many of our clinical instructors recommend that we refer to when conducting research regarding patient medications. These are credible and reliable sources that we regularly use within our clinical practice for educative and research purposes. We used this source to research relevant information for the various medications that the client has been prescribed.

Isosource® 1.5. (n.d.). Retrieved from https://www.nestlehealthscience.ca/en/brands/isosource/isosource1-5-hcp

While researching for tube feed information we were unable to find any information in our drug guides or within a paper relating to our specific tube feed. Contacting pharmacy at the hospital did not pan out for us and as such we went to the source. Royal Inland Hospital uses Nestle brand Isosource; as such, we decided that the official information listed on their webpage would suffice as official information.

Johnstone, C. C. (2018). Pathophysiology and nursing management of acute pancreatitis. *Nursing standard (Royal College of Nursing (Great Britain): 1987)*.

This source is a double-blind peer-reviewed article that discusses the pathophysiology of acute pancreatitis and the necessary nursing considerations regarding the care of a patient with acute pancreatitis. This scholarly article was accesses used the Thompson Rivers University’s Library discover feature. This was a valuable resource because it not only discussed the pathophysiology behind pancreatitis, but also how to manage pancreatitis, the common signs and symptoms, and who is most at risk.

Lewis, S. L., Heitkemper, M. L., Bucher, L., Dirksen, S., & Camera, I. M. (2014). *Medical-surgical nursing in Canada: Assessment and management of clinical problems.* (M. A. Barry, S. Goldsworthy, & D. Goodridge, Eds.) (3rd ed.). Toronto: Elsevier.

This textbook is a sanctioned and required textbook by the Thompson River University School of Nursing. This textbook serves as a credible and reliable resources to research and obtain information regarding a variety of medical conditions and includes the necessary nursing diagnosis and interventions, pathophysiology, diagnostic rationales, and signs and symptoms,

and risk factors associated with the relevant health conditions.

Lewis, J. L. (2018). Hyponatremia. *Merck Manual.* Retrieved from [www.merckmanuals.com/professional/endocrine-and-metabolic-disorders/electrolyte-disorders/hyponatremia](http://www.merckmanuals.com/professional/endocrine-and-metabolic-disorders/electrolyte-disorders/hyponatremia)

This online resource was used as it was found to be quite difficult to find one particular source to adequately explain the relationship between sodium and chloride as they usually are explained as one collective unit. I was further looking for information for how sodium levels affect chloride levels independently. This article is credible and reputable and provides as a great resource for explaining the relationship.

Lilley, L. L., Collins, S. R., Snyder, J. S., & Swart, B. (2016). *Pharmacology for Canadian health care practice* (3rd edition). Toronto, ON: Mosby ELSEVIER

This textbook is a sanctioned resource by the Thompson River University School of Nursing that was recommended by several professors. This is also a credible resource to use for researching medication information. This source thoroughly explains the mechanisms of action, indications, pharmacokinetic considerations, as well as client teaching points. This was a valuable source for understanding the medications for our client.

Pagana, K. D., Pagana, T. J., & Pike-MacDonald, S. A. (2019). *Mosbys Canadian manual of diagnostic and laboratory tests*(2nd ed.). Toronto, ON: Elsevier.

This textbook is a sanctioned resource by the Thompson River University School of Nursing that was recommended by several professors. This is a credible resource to use for researching lab diagnostics and test results. This source thoroughly explains the rationale behind the tests, information regarding the different types of tests, and what each result may indicate. This was a valuable source for understanding the lab results and diagnostic studies for our client.

Takata, S., & Yasui, N. (2001). Disuse osteoporosis. *The Journal of Medical Investigation.* Retrieved from [www.medical.med.tokushima-u.ac.jp/jmi/vol48/text/v48\_n3-4\_p147.html](http://www.medical.med.tokushima-u.ac.jp/jmi/vol48/text/v48_n3-4_p147.html)

Thong, B., Ima-Nirwana, S., & Chin, K. (2019). Proton pump inhibitors and fracture risk: A review of current evidence and mechanisms involved. *International Journal of Environmental Research and Public Health. 16*(9), 1571. doi:10.3390/ijerph16091571

These online journals were used to further explain the relationship between use of Esomeprazole and the increased risk for fractures. Due to the patient being bed ridden, there was a need to elaborate on the rationale for the risk imposed from use of Esomeprazole and osteoclast activity.

Surgical Critical Care. (2017). Stress ulcer prophylaxis. Retrieved from http://www.surgicalcriticalcare.net/Guidelines/stress%20ulcer%20prophylaxis%202017.pdf

This online source was used to further elaborate on the purpose of using a proton pump inhibitor to inhibit gastric acid secretion post-surgical. This particular source was used as it is a credible source based on the surgical critical care guidelines.

Wang, C. (2013). The relationship between type 2 diabetes mellitus and related thyroid diseases. *Journal of Diabetes Research*. Retrieved from [www.ncbi.nlm.nih.gov/pmc/articles/PMC3647563/](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3647563/)

The online Journal of Diabetes Research was used as the source to further elaborate on the relationship between hypothyroidism and glucose levels. Unfortunately, there was difficulty finding a scholarly source that indicated hypothyroidism as a potential cause for increased glucose therefore only one scholarly source could be used for this particular connection.

Weinreb, R. N., Aung, T., & Medeiros, F. A. (2014). The Pathophysiology and Treatment of Glaucoma. *Jama, 311*(18), 1901. doi: 10.1001/jama.2014.3192

This source is a peer-reviewed article from the American Medical Association, accessed through the Thompson Rivers University’s Library discover feature. This article discussed the pathophysiology and treatment of glaucoma and was used for clinical review and educational purposes. This was a valuable source because it discussed risk factors, signs and symptoms, and how to prevent and treat glaucoma.

Wensel, T. (2009). Administration of proton pump inhibitors in patients requiring enteral nutrition. *Pharmacy and Therapeutics: A Peer-Reviewed Journal for Managed Care and Hospital Formulary Management. 34*(3), 143-160. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2697083/>

It was deemed necessary to explain the purpose of a proton pump inhibitor to suppress gastric acid secretion during enteral therapy. I used this source to lead into the rest of my paragraph explaining the purpose of taking esomeprazole in conjunction with a jejunostomy tube. This is a peer-reviewed scholarly source that accurately outlines the information needed to make the connection for the client.

Wopereis, D. M., Du Puy, R. S., van Heemst, D., Walsh, J. P., Bremner, A., Bakker, S. J. L., Bauer, D. C., Cappola, A. R., Ceresini, G., Degryse, J., Dullaart, R. P. F., Feller, M., Ferrucci, L., Floriani, C., Franco, O. H., Iacoviello, M., Iervasi, G., Imaizumi, M., Jukema, J. W., & Khaw, K.-T. (2018). The Relation Between Thyroid Function and Anemia: A Pooled Analysis of Individual Participant Data. *Journal of Clinical Endocrinology & Metabolism*, N.PAG. doi:10.1210/jc.2018-00481

This resource was found using Thompson Rivers University’s Library discover feature, and was further narrowed using exclusive search for scholarly, peer reviewed sources. This source conducted a meta-analysis study on the relationships between thyroid function and anemia. This resource served to be a credible and reliable resource to obtain information regarding the relationship between hypothyroidism, anemia, and the related blood diagnostics. The information from this scholarly article was required to supplement the information obtained in other sources to ensure that the data collected was consistent.

World Health Organization (WHO). (2012). *Micronutrient deficiencies: Iron deficiency anaemia.* Retrieved from http://www.who.int/nutrition/topics/ida/en/index.html

The World Health Organization (WHO) is an international health organization that directs international health within the United Nation’s system. The WHO tracks and monitors the prevalence and incidences of diseases and conditions worldwide and provides credible, reliable, and accurate information regarding these health conditions. This source was used to obtain information regarding the prevalence of iron-deficient anemia to provide a robust description of the health condition. The information obtained from the source was consistent with the information that we obtained in other resources however, this source presented as the most recent source.

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