STATE OF WASHINGTON
DEPARTMENT OF HEALTH
MEDICAL QUALITY ASSURANCE COMMISSION

FILED
FEB 04 2014
Adjudicative Clerk Office

In the Matter of the License to Practice as a Physician and Surgeon of:	**No. M2010-844**
ZBIGNIEW M. GRUDZIEN, MD License No. MD00039350 Respondent	**STATEMENT OF CHARGES**

The Executive Director of the Medical Quality Assurance Commission is authorized to make the allegations below, which are supported by the evidence contained in file numbers 2010-143782 and 2010-145501. The patients referred to in this Statement of Charges are identified in the attached Confidential Schedule.

1. ALLEGED FACTS

1.1 On December 14, 2000, the state of Washington issued Respondent a license to practice as a physician and surgeon. Respondent's license is currently active.

1.2 During pertinent time frames, Respondent provided medical services to patients as a physician and manager of Hope Medical Holistic Clinic, PLLC, located in Vancouver, Washington.

1.3 Review of Respondent's medical charts for Patients A through K for time frames between September 2006 and March 2010 indicates that Respondent's care for these patients fell below the standard of care in numerous respects detailed below.

FAILURE TO APPROPRIATELY MANAGE CARDIAC CONDITIONS

1.4 Respondent failed to appropriately manage cardiac conditions experienced by Patients A-G.

ORIGINAL

PATIENT A

1.4.1 Patient A presented in September 2007 as a fifty-eight year old female with a complex history of multiple diseases, problems and surgeries. Patient A described tightness or squeezing chest pain, with accompanying nausea, which was precipitated by exercise and relieved when at rest. Patient A's symptoms included edema, fainting and fatigue. She also sought care for diabetes. Patient history included coronary artery disease since 2004, along with irregular heartbeat (cardiac arrhythmias), fainting, congestive heart failure, type two non-insulin dependent diabetes, high blood pressure (hypertension), degenerative joint disease, chronic fatigue, irritable bowel syndrome, colon polyps, abnormal turning of the eyes (strabismus) with right eye vision deficit and goiter. Past surgical history included hernia repair, ovarian surgery, appendectomy and percutaneous transluminal coronary angioplasty (PTCA) to open up blocked coronary arteries in 2005. Respondent provided physician services to Patient A through January 14, 2010. Respondent's management of Patient A's care in response to the history and symptoms presented by Patient A was below standard, both initially and as time progressed, in the following respects:

1.4.1.1 There is no indication that Respondent requested or reviewed Patient A's prior medical records to obtain necessary information on Patient A's cardiac history.

1.4.1.2 Respondent did not determine whether the patient ever had a heart attack, what cardiac diagnostic procedures had been done, whether a stent had been placed earlier during her PTCA, whether she had "at risk" heart wall tissue (myocardium) requiring interventional treatment, what type of irregular heart beat she had, or whether the history of fainting was due to a sudden loss of blood flow and oxygen to the brain due to irregular heart rhythm (cardiac syncope).

1.4.1.3 Patient A's known coronary artery disease and reports of regular exertional chest pain symptoms indicative of inadequate oxygen

ORIGINAL

supply to the heart muscle (angina), clearly indicated a need for aspirin and nitroglycerin, which Respondent did not prescribe until almost two years later, on July 28, 2009.

1.4.1.4 Patient A's hypertension was acknowledged by Respondent but not adequately controlled. While he did alter blood pressure medications on occasion, Respondent did not assertively treat the hypertension to reach the recommended goal. For example, Patient A had blood pressure readings indicating significant hypertension: 170/100 on September 9, 2009, and 180/100 on December 8, 2009, without adjustment of her blood pressure medication by Respondent.

1.4.1.5 Respondent assessed Patient A to have hypertensive heart disease with heart failure, without gathering any information to confirm heart failure, or ordering an echocardiogram to determine this diagnosis.

1.4.1.6 Respondent did not adjust Patient A's medications as would have been appropriate for the heart failure he assessed. Respondent discontinued lisinopril treatment for Patient A's high blood pressure and failed to initiate diuretic treatment. Although the diagnosis of congestive heart failure was carried forth in Patient A's chart, Respondent failed to provide the assessment and treatment indicated for this condition.

1.4.1.7 On October 25, 2007, Patient A reported palpitations, and an electrocardiogram showed some disturbance in her heart rhythm. Despite her reported history of cardiac arrhythmias and past fainting (possible syncope), Respondent failed to order further testing, such as could be provided with an event monitor, to evaluate for possible serious cardiac arrhythmias.

1.4.1.8 Respondent failed to clarify or evaluate Patient A's repeated complaints of dizziness. Respondent failed to ask clarifying questions to determine whether Patient A was experiencing vertigo or

ORIGINAL

lightheadedness that could have been due to unrecognized arrhythmias or other cardiac issues.

1.4.1.9 Although Respondent frequently ordered fasting lipid testing for low-density lipoprotein (LDL) levels in Patient A, he did not respond with appropriate treatment for the consistently abnormal values obtained. The recommended LDL cholesterol level in patients with coronary artery disease is less than 70. On October 4, 2007, Patient A's LDL was 129. Excess cholesterol contributes to plaque formation within arteries, which can reduce the flow of blood to the heart. The chest pain reported by Patient A is one potential symptom of such plaque formation. Respondent recommended a low-fat diet, but did not initiate statin therapy. Statin is a class of drugs that reduces the production of cholesterol by the liver. On February 13, 2008, Patient A had an LDL of 161, but no statin treatment was recommended and hyperlipidemia (excessive fatty substances such as cholesterol in the blood) was not noted in the chart. Between October 4, 2007, and January 7, 2010, Patient A had abnormally high LDL readings ranging from 129 to 188, but Respondent did not respond with effective treatment, which increased Patient A's risk for progressive atherosclerotic cardiovascular disease.

PATIENT B

1.4.2 Patient B presented to Respondent in September 2007 as a seventy year old female patient with complaints of chest pressure worsened by exertion, trouble breathing, palpitations and fatigue. Respondent provided physician services to Patient B through March 10, 2010. According to Respondent's documentation, Patient B had the following medical problems: coronary artery disease, congestive heart failure, history of a stroke in 2006 with resultant weakness on one side of the body (hemiparesis), Type 2 Diabetes Mellitus, "arthritis", history of depression, anxiety, "unspecified Disorder of the Thyroid", and neck pain with associated numbness in bilateral upper extremities.

ORIGINAL

Respondent's management of Patient B's cardiac care in response to the history and symptoms presented by Patient B was below standard, both initially and as time progressed, in the following respects:

1.4.2.1 Respondent documented Patient B's history of coronary artery disease, but did not appropriately gather history necessary to provide adequate treatment. Respondent did not determine whether Patient B had suffered a past heart attack, whether and when she had a heart catheterization or heart stress test, or what the results were.

1.4.2.2 Respondent failed to clarify Patient B's coronary artery anatomy. He failed to determine whether Patient B had remaining "at risk" myocardium. Respondent did not gather information to determine what cardiac arrhythmias Patient B had experienced, or what past treatment or recommendations had been provided for her arrhythmia.

1.4.2.3 Respondent failed to appropriately treat Patient B's symptoms of angina and palpitations, which persisted throughout the time he served as her physician.

1.4.2.4 Respondent did not provide Patient B with recommended treatment for patients with coronary artery disease. Respondent did not prescribe nitroglycerin for Patient B's angina symptoms, with attendant recommendations for her to seek emergent care if chest pain wasn't relieved with three doses of nitroglycerin. In addition, Respondent failed to initiate treatment with angiotensin converting enzyme (ACE) inhibitors, although this is a standard recommendation for secondary prevention of coronary artery disease.

1.4.2.5 In October 2007, Respondent discontinued Patient B's aspirin and lovastatin treatment without discussion in the history or assessment portions of the chart. The noted change in the patient's medication list indicated discontinuation for "dizziness", although no experience of dizziness is charted. As an antiplatelet agent, aspirin was essential medication for secondary prevention of coronary artery disease

and stroke. Respondent failed to replace the aspirin with an alternate antiplatelet medicine, thus placing Patient B at risk for a heart attack or second stroke. Respondent failed to replace the lovastatin that Patient B had been prescribed by an earlier provider to appropriately manage Patient B's hyperlipidemia. The recommended goal for LDL cholesterol levels in a patient with known coronary heart disease is under seventy. This value was never attained during Respondent's care for Patient B.

1.4.2.6 Respondent indicated a diagnosis of heart failure for Patient B starting March 20, 2008, which continued in many subsequent notes. Respondent did not explore the etiology of Patient B's congestive heart failure to determine if it was ischemic in origin, such that augmented medical treatment or interventional treatment for coronary artery disease might have been in order. Respondent did not order an echocardiogram to assess the percentage of blood pumped during each heartbeat (ejection fraction), and did not prescribe standard medications for heart failure. If present, heart failure should have raised concern about continuing to treat Patient B with intravenous (IV) infusions as ordered by Respondent.

1.4.2.7 On March 20, 2008, Respondent began treating Patient B with digoxin, which is used for heart rate control in rapid heart arrhythmias or for strengthening of the heart contraction for patients with congestive heart failure. Respondent did not document an associated diagnosis or indication, as the cardiac rhythm was noted to be regular and there was no new indication of congestive heart failure.

PATIENT C

1.4.3 Patient C presented for management of diabetes and hypertension in December 2008 as a sixty-five year old female. Problems included diabetes mellitus, coronary heart disease, and morbid obesity. Respondent provided physician services to Patient C through January 28, 2010. Respondent's management of Patient C's care in response to the cardiac history and

ORIGINAL

symptoms presented by Patient C was below standard, both initially and as time progressed, in the following respects:

1.4.3.1 Patient C indicated a previous twenty-one day hospital stay related to coronary artery disease. Respondent failed to determine what occurred during that hospital stay. Nor did Respondent explore the status of the coronary artery disease by determining past stress testing or cardiac catheterization results or cardiac ejection fraction, as required by the standard of care. Respondent failed to treat Patient C with statins for cholesterol management or with aspirin for secondary prevention of coronary artery disease.

1.4.3.2 Respondent failed to adequately address Patient C's report of chest pain. The charting shows Patient C's chest pain symptoms were newly reported by Patient C to Certified Physician Assistant (PA-C) Deborah Bryant on September 18, 2009. PA-C Bryant participated under Respondent's supervision in providing patient care under a practice plan approved April 17, 2009. Patient C's description of chest pain symptoms came within the context of the patient's history of coronary artery disease with significant risks for progression of this disease due to diabetes and hypertension. There was inadequate assessment for unstable angina, for which urgent evaluation and treatment would have been indicated. Patient C was not given nitroglycerin to treat possible angina, and was not directed to contact emergent care if chest pain was not relieved by three nitroglycerin tablets. Patient C was not recommended aspirin for myocardial infarction prevention. Much of the history and physical examination for the September 18, 2009, visit was copied directly from prior notes, and related to earlier matters, including a draining, swollen lower leg, resolved in an earlier appointment, instead of the problems reported by Patient C during the September 18 visit. Respondent did not provide adequate follow-up on the reported chest pain. Although a referral

ORIGINAL

to cardiology was ultimately ordered, it was not scheduled for another three months. There is no consult note from cardiology in Patient C's records, and no mention of cardiology recommendations from the cardiology referral to clarify the status of Patient C's heart disease.

PATIENT D

1.4.4 Patient D presented in February 2007 as a seventy-eight year old male with diabetes, coronary artery disease (with a related forty-five day hospitalization in 1996), chronic combined systolic (left ventricle heart pumping capacity) and diastolic (heart ventricle filling capacity) congestive heart failure, hearing loss, benign enlarged prostate, symptoms of chronic fatigue, back pain, history of heart arrhythmia, bladder cancer in 1988, trauma from a fall, and a pleural effusion (build-up of fluid between the layers of tissue that line the lungs and chest cavity). Patient D's family history included a brother with colon cancer. Respondent provided physician services to Patient D through January 28, 2010. Respondent's management of Patient D's cardiac care in response to the history and symptoms presented by Patient D was below standard, both initially and as time progressed, in the following respects:

1.4.4.1 Respondent failed to gather needed details about Patient D's significant history of heart disease with prior hospitalization and heart arrhythmias. Although Respondent noted that old records would be obtained, no release for these records was requested and the old records were not received. Respondent failed to evaluate the congestive heart failure with chest x-ray, echocardiogram or brain-type natriuretic peptide (BNP) level. Respondent was unaware whether Patient D had undergone past cardiac procedures and whether he had "at-risk" myocardium, a compromised heart ejection fraction, or a need for management of heart arrhythmias. The diagnosis of congestive heart failure was noted on a multitude of subsequent visits, and later diagnosis of combined systolic and diastolic heart failure was also documented without echocardiogram

ORIGINAL

support for this conclusion. Even more concerning, Respondent continued to treat Patient D with regular IV infusions that placed him at risk for volume overload and worsening heart failure.

PATIENT E

1.4.5 Patient E presented in March 2007 as an eighty-one year-old female with complaints of shortness of breath accompanied by edema, chest tightness almost daily, dizziness and fatigue. Patient E's conditions included chronic high blood pressure, congestive heart failure, hypothyroidism, hyperlipidemia, colon cancer with tumor removal in 2007, gastroesophageal reflux disease, a history of gastrointestinal track bleeding with a hematocrit of 17.3 noted on March 8, 2007, and a history of malaria. She was noted to have been diagnosed with congestive heart failure six months earlier. Respondent provided physician services to Patient E through March 16, 2010. Respondent's management of Patient E's cardiac care in response to the history and symptoms presented by Patient E was below standard, both initially and as time progressed, in the following respects:

1.4.5.1 Respondent failed to adequately monitor or treat Patient E's heart issues. Respondent did not evaluate the basis for the consistent complaints of chest tightness, and failed to refer for testing. Respondent failed to prescribe medication to treat potential angina. Respondent failed to request records about the diagnosis of congestive heart failure. Respondent failed to request or to order information on Patient E's heart's ability to pump blood (ejection fraction), left and right heart chamber (ventricular) function, or heart valve function. Respondent did not pursue the possibility that coronary artery disease (narrowing in the coronary arteries that limits blood flow to heart muscle) was the cause of the congestive heart failure (ischemic cardiomyopathy), despite the reported symptoms that raise concern for angina. Respondent failed to treat Patient E with medications that would be indicated for a patient with

ORIGINAL

congestive heart failure. Respondent's recommendation for intravenous vitamin and mineral infusions (without indication) resulted in Patient E receiving forty-two such infusions. Given Patient E's advanced age and known congestive heart failure, these treatments placed Patient E at risk for volume overload and worsened the congestive heart failure without treatment benefit.

PATIENT F

1.4.6 Patient F presented in July 2008 as a seventy-eight year-old female with a problem list that included congestive systolic and diastolic heart failure, hypertrophic cardiomyopathy, aortic stenosis, recurrent reports of palpitations, a history of transient ischemic attack (TIA), hypertension, senile dementia with depressive features, a history of depression and reported depression in review of systems, a history of kidney stones, osteoarthritis, metabolic syndrome, myalgia, hearing loss, cataracts and glaucoma. Lab results indicated pre-diabetes that was not mentioned. Patient F was treated with sublingual nitroglycerin to take as needed for chest pain and was on a long-acting nitrate medication. These treatments suggest Patient F had active coronary artery disease, although this was not explicitly stated. Patient F was noted to have intermediate coronary syndrome (also referred to as unstable angina) on one visit, but appropriate evaluation and treatment for this was not instituted.

1.4.6.1 Respondent failed to adequately manage Patient F's heart disease issues. Respondent documented a diagnoses of congestive heart failure and narrowing of the heart's aortic valve through which blood flows from the heart to the body (aortic stenosis). However, Respondent did not record information necessary to indicate the severity of heart valve disease and whether the cardiac contractility (ejection fraction) was compromised. Patient F's medication list reflected usual treatment for coronary artery disease, although this diagnosis was not stated. If Patient F did, indeed, have coronary artery disease, requesting past records

ORIGINAL

regarding the extent of her disease, initiating testing if not previously done, and assertive management of hyperlipidemia would have been in order. Respondent did none of these. On September 15, 2008, when Patient F presented for a blood draw, she reported chest pain. Respondent ordered cardiac enzymes [serum creatine kinase (CK) and creatine kinase muscle and brain (CK-MB) levels] and an electrocardiogram (ECG) to determine a diagnosis of intermediate coronary syndrome. This condition, also known as unstable angina, is marked by compromised cardiac perfusion that can progress to a myocardial infarction. Unstable angina may progress to a heart attack; so should be urgently treated to decrease the risk of permanent loss of heart muscle. Respondent failed to treat Patient F's unstable angina with oxygen, aspirin, or nitrates. Moreover, Respondent drew labs in the clinic and then sent the patient home rather than referring her to the hospital. This management placed Patient F at risk for a myocardial infarction. In subsequent visits, coronary artery disease was not listed as a diagnosis and Respondent did not pursue cardiac testing or provide a cardiology referral. Respondent prescribed #10 nitroglycerin tablets plus 3 refills on October 26, 2009. On January 28, 2010 Respondent was contacted by Patient F's pharmacy for a nitroglycerin refill request. Patient F was using a high volume of nitroglycerin indicating poorly controlled angina or inappropriate use of the medication. Respondent did not address this issue.

1.4.6.2 Respondent's ordering of IV vitamin and mineral infusions for Patient F resulted in exacerbated cardiac symptoms for Patient F. On December 3, 2008 when Patient F presented for the vitamin and mineral infusion recommended by Respondent, she developed severe chest pain that coincided with hypotension (blood pressure 80/30) and an oxygen saturation of 93%. Respondent determined a diagnosis of acute coronary insufficiency. He ordered serum CK measurement, myoglobin and troponin tests, but did not perform an electrocardiogram. Respondent

ORIGINAL

initiated IV fluid treatment and administered sublingual nitroglycerin for three doses. When the patient continued to report severe chest pain and her oxygen saturation dropped below 90%, Respondent ordered a rush on the CK tests, and arranged ambulance transport of Patient F to the hospital. When Patient F returned for a visit on December 17, 2008 trigger point injections were performed, but Respondent made no reference to Patient F's prior chest pain episode and hospital evaluation and no reference to a diagnosis of coronary artery disease. On March 12, 2009, Patient F had another episode of hypotension, with a blood pressure of 80/40 that lasted for several hours. Respondent determined Patient F as experiencing vasovagal collapse and did not order an electrocardiogram or consider the possible presence of unstable angina or acute coronary syndrome. On April 3, 2009, Respondent was informed Patient F was being discharged from the hospital after evaluation and treatment of a congestive heart failure exacerbation. Patient F received an IV fluid infusion on March 25, 2009 and again on April 1, 2009. Respondent saw Patient F on April 14, but failed to request inpatient records to review the cardiac evaluation and determine what treatment had been provided. Instead Respondent performed trigger point injections on this date.

PATIENT G

1.4.7 Patient G was seen by Respondent earlier, but review of chart notes begins as of January 31, 2008, when Patient G was a sixty-six year old female. Presenting problems included coronary artery disease, depression, hypertensive heart disease with heart failure, hyperlipidemia, gastroesophageal reflux disease, osteoarthritis, and chronic complaints of neck and head pain, fatigue, insomnia, nausea, diarrhea and constipation. Respondent provided physician services to Patient G through June 6, 2009. Respondent's management of Patient G's care in response to the history and symptoms

presented by Patient G was below standard, both initially and as time progressed, in the following respects:

1.4.7.1 Patient G had a documented diagnosis of coronary artery disease. Additionally, she regularly reported symptoms of retrosternal chest pressure, worsened by walking up a hill or upstairs, that occurred almost daily. These reports of regular chest pain likely represented angina. Respondent failed to treat Patient G with nitroglycerin as needed for chest pain. Moreover, Respondent stopped Patient G's beta-blocker antianginal treatment for no apparent reason. Patient G was not on a long-acting nitrate to substitute for beta-blocker treatment. The records indicate that atenolol was stopped on September 30, 2008, though there was no visit or documentation from that date or any explanation of the discontinuation of this treatment. Atenolol reappeared on medication lists in February 2009. Respondent neglected to treat Patient G with aspirin as would have been clearly indicated to prevent a myocardial infarction. Respondent's records did not include necessary information about the diagnosis and extent of coronary artery disease, whether additional heart muscle was at risk for ischemic injury, whether a past myocardial infarction had occurred, and whether Patient G had any compromise to her ejection fraction.

1.4.7.2 Respondent failed to adequately manage Patient G's hyperlipidemia. Given the diagnosed coronary artery disease, tight cholesterol control was indicated for Patient G. Guidelines recommended an LDL goal of less than 70. On June 4, 2008, Patient G had an LDL of 205. Respondent did not increase her cholesterol lowering statin medication at this time, and did not recheck Patient G's lipids until one year later. The medication list on July 24, 2008 and after indicated that statin treatment had been discontinued. On June 8, 2009, Patient G's LDL was 189, still way above the recommended goal. No alteration in treatment was recommended despite these abnormal results. Assertive

ORIGINAL

titration of cholesterol lowering medication was indicated until Patient G's LDL was documented to be under 70.

1.4.7.3 Respondent included a diagnosis of hypertensive heart disease with heart failure in Patient G's regular visit assessments, but did not note symptoms or signs of heart failure. Respondent did not obtain an echocardiogram to support a diagnosis of heart failure.

SUBSTANDARD MANAGEMENT OF NEUROPSYCHIATRIC CONDITIONS

1.5 Respondent failed to appropriately manage neuropsychiatric conditions experienced by Patients A-J.

PATIENT A

1.5.1 Patient A presented on August 26, 2009, to Respondent with confusion, disorientation, lack of enjoyment in previously pleasurable activities (anhedonia), disturbed sleep, anxiety, and strong mood changes. Respondent made no comments as to the possible cause of these symptoms, and did not attempt to determine whether depression or dementia might be involved. Patient A continued to report these symptoms in subsequent visits, but not until December 8, 2009, did Respondent document a cognitive assessment. Respondent then concluded Patient A had vascular dementia and generalized anxiety disorder. The diagnosis of vascular dementia was unfounded, given available history, the normal neurological examination, and lack of brain imaging showing cerebrovascular disease. Respondent noted that Patient A's speech was garbled, which, if a new symptom, could indicate a recent cerebrovascular event requiring assessment with imaging. Respondent failed to evaluate Patient A for a reversible cause of cognitive decline. Instead, Respondent ordered a Troponin I test and creatine phosphokinase (CPK) isoenzymes (tests for acute coronary syndrome or heart attack), which were not indicated. Though Patient A attested to numerous symptoms of depression, including low energy, insomnia,

ORIGINAL

anhedonia, decreased concentration, sadness of mood, irritability, and anxiety; Respondent did not conclude this diagnosis. More than three months and numerous visits after Patient A first reported these symptoms, Respondent initiated paroxetine (brand name Paxil) treatment, seemingly for generalized anxiety disorder. Such treatment for an anxiety disorder or for depression was indicated at an earlier time.

PATIENT B

1.5.2 Respondent prescribed the anti-depressant paroxetine for Patient B on January 24, 2008, without charting psychiatric symptoms to support this treatment. Patient history did not note psychiatric symptoms, the psychiatric examination results were noted to be unchanged, and there was no assessment of diagnostic criteria for depression or anxiety. In prior and subsequent notes, Respondent did repeat a diagnosis of "anxiety, reaction", without any assessment of treatment efficacy. The May 15, 2008, medication list appears to show paroxetine was discontinued due to ineffectiveness, but it remains on the active and inactive medication list in subsequent notes. No discussion occurs about unresponsive psychiatric symptoms or any consideration of adjusting the paroxetine dosage or alternative anti-depressants.

PATIENT C

1.5.3 Respondent failed to evaluate Patient C for depression, despite reported numerous symptoms related to depression, including insomnia, low energy, and poor concentration.

PATIENT D

1.5.4 On October 22, 2007, Respondent reported that Patient D was experiencing confusion, forgetfulness and difficulty concentrating. Respondent noted a psychiatric exam which indicated that Patient D's attention span, concentration, awareness of current events, past history, vocabulary, ability to

ORIGINAL

name objects, repeat phrases and speak spontaneously were poor. Intellectual impairment is noted with severe memory loss, and disorientation to time and often place. No abnormalities on neurological exam were noted. Respondent ascertained a diagnosis of chronic organic brain syndrome and cerebrovascular disease without adequate evidence. In contrast, Respondent's prior psychiatric examination on February 21, 2007, noted Patient D was oriented to person, place and time, had intact recent and remote memory normal recall of three objects at five and ten minutes, with good ability to repeat phrases and speak spontaneously and intact abstract thought, judgment and insight. Respondent thus documented a dramatic and very concerning change in cognition over an eight-month period of time. This is not consistent with chronic organic brain syndrome. The normal neurological exam does not support a vascular dementia due to cerebrovascular disease. Respondent failed to assess Patient D for possible reversible causes of cognitive decline such as thyroid dysfunction, B12 deficiency or depression.

PATIENT F

1.5.5 Respondent failed to adequately address mental health issues reported by Patient F. In July 2008, Patient F had no notable cognitive deficits on exam and was noted to have intact orientation, short and long-term memory, spontaneous speech and normal insight and judgment. On September 23, 2008, Patient F reported confusion, disorientation, short-term memory loss, apathy, anhedonia and sleeping difficulty. On September 23, 2008, her exam demonstrated disorientation, poor memory, a poor ability to speak spontaneously and impaired insight and judgment. Respondent determined a diagnosis of senile dementia with depressive features for Patient F. Respondent failed to consider what might have contributed to the patient's rapid cognitive decline. Patient F's symptoms might have been accounted for by uncompensated depression. Respondent did not consider a diagnosis of major depressive disorder or consider treatment for depression. Additionally, Patient F had an

ORIGINAL

elevated thyroid stimulating hormone (TSH). Though hypothyroidism might have contributed to patient's depressive symptoms and increased body mass index ratio, Respondent did not initiate treatment for hypothyroidism. Patient F had known hearing loss, glaucoma and cataracts. These deficits might have played a role in the depression or perceived cognitive decline, though this was not addressed.

PATIENT G

1.5.6 Respondent failed to adequately manage Patient G's depression. Patient G was initially taking an antidepressant medication, citalopram (brand name Celexa). Medication lists show this was discontinued on October 30, 2007. Patient G regularly complained of fatigue, low energy and insomnia, which commonly result from depression. Respondent regularly listed a diagnosis of depression in his assessments, though he did not offer treatment for this.

1.5.7 On May 28, 2009, Respondent documented: "She also presents for evaluation of confusion, disorientation and significant memory loss. Associated symptoms include: anhedonia, disturbed sleep, and incontinence. The patient's behavior is depressed, labile and passive. Symptoms are gradually worsening. Associated signs and symptoms include low energy, physical activity decrease and memory loss." Psychiatric and neurological examinations were not detailed, but were noted to be unchanged from previous visit. Respondent added new diagnoses of cerebral atherosclerosis, hypertensive encephalopathy, and vascular dementia with depressed mood. Documentation did not include a cognitive assessment to support a diagnosis of dementia. The reported symptoms may have been related to untreated depression, rather than dementia. Respondent did not reinitiate antidepressant treatment, despite the noted symptoms of depression.

1.5.8 On June 9, 2009, Respondent initiated treatment with donepezil (brand name Aricept), which is a medication for moderate Alzheimer's dementia, not for vascular dementia. However, a diagnosis of Alzheimer's disease was not

ORIGINAL

substantiated by history or examination. Respondent did not check Patient G's thyroid stimulating hormone (TSH) levels or screen for B12 deficiency, as is recommended in evaluating patients with possible dementia.

PATIENT H

1.5.9 Patient H presented to Respondent in September 2006 as a sixty-five year old male. Patient H's problems included insulin dependent diabetes mellitus, sleep apnea syndrome addressed with continuous positive airway pressure (CPAP) device, hypertension, hyperlipidemia, glaucoma, depression, arthritis with back pain, and a possible history of bladder stones. Respondent provided physician services to Patient H through March 29, 2010. Respondent's management of Patient H's mental health care in response to the history and symptoms presented by Patient H was below standard, both initially and as time progressed, in the following respects:

1.5.9.1 Patient H presented with depressed mood. Respondent did not follow-up to inquire about progression of the depression symptoms, or whether the patient had suicidal or homicidal ideation. Respondent did not reassess Patient H's current antidepressant medication.

1.5.9.2 Respondent inadequately managed Patient H's symptoms of depression, which included the patient's report of depressed mood, lack of energy, and decreased concentration. Respondent attributed low energy to vitamin and mineral deficiencies, despite a multitude of vitamin and mineral infusions and a lack of laboratory data indicating deficiencies. Respondent listed depression in his assessment diagnoses, and this continued to be noted in review of systems and the diagnoses lists, but no additional treatment was provided after discontinuation of sertraline (brand name Zoloft) on May 15, 2007. There was no documented review of depression symptoms or discussion of changes in medications.

ORIGINAL

PATIENT I

1.5.10 Patient I presented in September 2007 as a thirty-one year old female with a problem list that included hypertension, reactive airway disease (asthma), depression, bipolar affective disorder, left ovarian cancer (2003), cervical cancer (1994, 1997, 2003), diabetes, tubal pregnancy, lupus with a past 21-day hospitalization, methamphetamine abuse, increased body mass index of 41, fatigue, three miscarriages, former tobacco dependence (quit 8/07), tubal ligation (2003), a history of thirteen motor vehicle accidents (without documentation of associated injuries), and "progressive poly-arthritis". At first visit, Patient I's medication list included aripiprazole (brand Abilify), prazosin hydrochloride, quetiapine (brand Seroquel), clonazepam, and acyclovir. Respondent provided medical services to Patient I through November 29, 2007. Respondent's management of Patient I's care in response to the history and symptoms presented by Patient I was below standard, both initially and as time progressed, in the following respects:

1.5.10.1 Respondent mismanaged Patient I's psychiatric illness. He discontinued multiple psychiatric medications, including clonazepam and quetiapine at the first visit. Additionally, Respondent doubled Patient I's prazosin dose at the first visit, without any indication as to what prompted this change in dose. Patient I was presumably on prazosin for post-traumatic stress disorder symptoms. Respondent did not communicate with the previous prescriber of Patient I's psychiatric medications and did not request related records. It was unclear whether Patient I was to follow up with her previous psychiatric prescriber.

PATIENT J

1.5.11 Patient J was under Respondent's care beginning December 23, 2009. Patient J was primarily cared for by Deborah Bryant, PA-C, working under Respondent. Patient J's medical problems included Human T-cell lymphotrophic virus (HTLV) type II with associated neuromuscular deficits, Diabetes,

Hypertension, Chronic Obstructive Pulmonary Disease, history of depression, fibromyalgia, tobacco dependence, complaints of fatigue/malaise. Patient J reported being disabled due to HTLV associated neuropathic pain.

1.5.12 Patient J regularly reported depression during her clinic visits (noted in review of systems), but this was never addressed. Patient J also consistently reported exhaustion, fatigue and insomnia, all of which are frequently symptoms of depression.

SUBSTANDARD MANAGEMENT OF THYROID DISEASE

1.6 Respondent failed to appropriately manage thyroid disease conditions experienced by Patients B, E, F and I.

PATIENT B

1.6.1 Respondent did not gather information to clarify Patient B's charted "Unspecified Disorder of the Thyroid", although he discontinued her propylthiouracil medication for hyperthyroidism at her initial visit. Respondent did not indicate that Patient B's thyroid function had been tested and did not chart a reason to believe the Patient's presumed diagnosis of hyperthyroidism had spontaneously resolved. Three days after the discontinuation, a thyroid-stimulating hormone test revealed normal thyroid function, but this value would have reflected thyroid levels with the benefit of propylthiouracil, because the response to withdrawal of propylthiouracil would take place slowly, over approximately a six week period.

PATIENT E

1.6.2 Respondent failed to adequately manage Patient E's under-active thyroid gland (hypothyroidism). Respondent discontinued levothyroxine medication for hypothyroidism on August 27, 2007, without performing thyroid function tests to assess current function and despite the patient's known hypothyroidism. The change is reflected on the medication list, but Respondent's

ORIGINAL

treatment note for that day does not explain the discontinuation. Respondent failed to check Patient E's thyroid function level until three months later, on November 19, 2007. This test showed significantly elevated thyroid-stimulating hormone (TSH) value of 237 (normal range is 0.4-5), indicating severely underactive thyroid function. Neither this result, nor a TSH measurement of 188 obtained on November 26, 2007, prompted Respondent to treat Patient E for hypothyroidism. When lab testing on January 7, 2008 showed a still markedly elevated TSH value of 226, Respondent finally prescribed levothyroxine treatment three days later.

1.6.3 By June 11, 2008, Patient E's TSH was measured at less than .05, which indicates an excess of thyroid hormones caused by excessive levothyroxine medication. Nevertheless, Respondent did not decrease levothyroxine dosage or repeat the lab test until February 17, 2009, even though other tests were ordered during this interval. TSH values were measured in February and May of 2009 as continuing under 0.05, but the levothyroxine dose was not changed.

1.6.4 On September 10, 2009, after another TSH result under 0.05, and identified weight loss, Respondent decreased the levothyroxine dosage by 25 micrograms. Measurement of TSH on December 8, 2009, was 0.05 and Respondent did not lower the dose of levothyroxine again until January 5, 2010. At this time, Respondent charted that Patient E's condition was "chronic and progressive and we cannot expect a full recovery, but only to manage quality of life and risk factors." There was no identified progressive disease process at this time, except for the medication-induced hyperthyroidism.

PATIENT F

1.6.5 Patient F's TSH was noted to be high on numerous occasions and her symptoms of confusion, anhedonia, fatigue and constipation might have been due, in part, to hypothyroidism. Though Respondent repeatedly tested Patient F's thyroid function, he did not initiate treatment for hypothyroidism.

ORIGINAL

PATIENT I

1.6.6 Respondent assigned Patient I a diagnosis of a sudden toxic worsening of hyperthyroid symptoms (thyrotoxicosis) on September 26, 2007, despite the lack of an abnormal thyroid exam or available thyroid function test results. Lab results from October 2, 2007, indicated Patient I had normal thyroid function.

MISUSE OF INTRAVENOUS INFUSIONS

1.7 Respondent failed to appropriately manage intravenous infusions experienced by Patients A-F and H-K.

1.8 Respondent typically determined a diagnosis of "vitamin and mineral deficiency" for patients to justify sequential intravenous (IV) infusion of vitamins. These diagnoses were without sufficient basis in patient symptoms or laboratory abnormalities. Respondent routinely and frequently attempted to justify IV vitamin infusions based upon elevated homocysteine levels, although oral vitamin supplementation would suffice to manage any vitamin deficiency contributing to elevated homocysteine levels. Respondent did not follow-up to document efficacy of IV vitamin infusions, and arbitrarily determined a number of infusions without subsequent tracking of the number of infusions received. The frequency of these infusions determined by Respondent varied significantly from patient to patient without clinical basis. Respondent failed to track the number of infusions performed, so that patients often received many more infusions than recommended in chart notes. Respondent's use of IV vitamin infusions was unnecessary and likely ineffective for Patient A-F and H and K. It appears that the use of this treatment was for Respondent's personal gain. The following patients were subjected to Respondent's misuse of IV infusions:

PATIENT A

1.8.1 Respondent initiated IV vitamin and mineral infusions for Patient A on May 28, 2008, with a recommended fifteen (15) infusions. There was no tracking to determine when to stop, and Patient A received twenty two (22) such

infusions. Respondent's notes indicate that Patient A had coronary artery disease and hypertensive heart disease with heart failure, and she had poorly controlled blood pressure. Serial IV fluid infusions placed Patient A at risk of fluid overload and congestive heart failure.

PATIENT B

1.8.2 Respondent diagnosed Patient B with a vitamin and mineral deficiency and recommended twelve (12) sequential intravenous vitamin and mineral infusions. A total of fifty-six(56) such infusions were administered at Respondent's office. This regular administration of IV fluids to an elderly woman with underlying heart disease and a diagnosis of congestive heart failure with unknown ejection fraction placed Patient B at risk for the onset, or exacerbation, of fluid overload. Respondent continued to treat Patient B with IV infusions even after diagnosing heart failure on March 20, 1998.

PATIENT C

1.8.3 Respondent recommended IV vitamin and mineral infusions for Patient C without documenting vitamin or mineral deficiency. The number of infusions appears arbitrary, with a total of thirty (30) provided. Diabetes control for Patient C was not improved by IV infusions and C-reactive protein increased with the infusions.

1.8.4 Respondent charted an autologous blood transfusion for Patient C on January 6, 9, 13, 16, and 20 of 2009, and on many subsequent visits, without any indication of a basis for such transfusion of blood from the patient's own body.

PATIENT D

1.8.5 Patient D was administered excessive IV vitamin infusions without indication and without tracking the total number provided. Respondent informed Patient D that his back pain was related, in part, to vitamin and mineral

deficiency. Patient D had not had any laboratory testing at this point and there was no objective evidence of vitamin and mineral deficiency. Respondent recommended twenty (20) weekly infusions, but Patient D was ultimately administered fifty (50).

1.8.6 Respondent determined that Patient D had congestive heart failure. Repeated IV fluid infusions, without any justifiable indication, placed Patient D at risk of volume overload. Despite the multitude of vitamin and mineral infusions over the prior two years, Patient D was noted to have vitamin and mineral deficiency on a July 9, 2009, assessment. Moreover, Patient D's fatigue and back pain symptoms persisted despite numerous IV vitamin and mineral infusions.

PATIENT E

1.8.7 Respondent ordered an arbitrary number of ten (10) IV vitamin and mineral infusions for Patient E without indication, then failed to track the number administered. Patient E was administered forty-two (42) infusions. Given her age of eighty-one years, and known congestive heart failure, this treatment unreasonably placed Patient E at risk of volume overload and worsened congestive heart failure.

PATIENT F

1.8.8 Respondent's treatment of Patient F with IV mineral and vitamin infusions without indication placed this seventy-eight year old patient, with congestive heart failure and aortic stenosis, at unreasonable risk of volume overload and worsened heart function. An arbitrary number of thirty (30) infusions were recommended. The extra volume from infusions also risked increased cardiac workload and angina symptoms. If Patient F actually had vitamin and mineral deficiency, oral replacement would have been far safer. An arbitrary number of thirty infusions was recommended, and continued even when the patient's weight notably increased. As noted in paragraph 1.4.6.2,

FINAL

Respondent continued to treat Patient F with IV fluid infusions shortly after hospital discharge for a congestive heart failure exacerbation.

PATIENT H

1.8.9 Respondent administered IV vitamins and minerals to Patient H without indication. The initial recommendation on March 5, 2007, was for twelve (12) weekly infusions, and then a reassessment of the treatment plan; however, the number of infusions was not tracked. On November 6, 2008, Respondent attributed Patient H's lack of energy to inadequate nutrition, mineral deficiency and vitamin deficiency. At this point, Patient H had been receiving IV vitamin infusions for over one and one-half years, without an attributed benefit in terms of repletion of vitamins and minerals. On August 19, 2009, Respondent stated the total number of IV vitamin infusions should be thirty (30), but by this time Patient H had already received sixty-eight (68) infusions, and an additional nineteen (19) were administered after this date. Patient H received a total of eighty-seven (87) IV vitamin infusions.

PATIENT I

1.8.10 Respondent recommended an arbitrary number of twelve (12) IV vitamin and mineral infusions for Patient I without any valid indication.

PATIENT J

1.8.11 Patient J was determined to have a vitamin and mineral deficiency that was not corroborated by laboratory testing, and she was inappropriately treated with IV vitamin and mineral infusions.

PATIENT K

1.8.12 Patient K presented on September 29, 2008, as a 50 year-old female with a history of type 2 diabetes mellitus diagnosed in 2000, hypertension, heart problems resulting in a 2005 hospitalization, history of a motor vehicle

accident, and a family history of heart disease. Patient K's blood cell count results revealed the presence of anemia. Respondent continued to provide health care services to Patient K through June 2009.

1.8.13 Respondent ordered IV iron infusions for Patient K's anemia, rather than pursuing the safer course of treating Patient K with oral iron replacement, as would have been indicated if iron deficiency anemia were present (which had not been confirmed with testing). There was no indication that Patient K had a malabsorption problem that would interfere with oral iron replacement, nor did Patient K suffer from other indicators calling for intravenous iron infusions such as continued gastrointestinal bleeding, inflammatory bowel disease with severe intolerance to oral iron preparations, hemodialysis, or failure to respond to oral iron preparations.

1.8.14 The Dexferrum (high molecular weight iron dextran) formula ordered for Patient K is associated with the highest incidence of adverse reactions. During the second IV iron infusion, Patient K developed itching and hives. During the next infusion, Patient K suffered a severe reaction of hypotension and unresponsiveness, requiring four doses of epinephrine and 1500 mg of IV fluids to regain a stable blood pressure. Despite this severe and potentially life-threatening reaction requiring IV epinephrine, Respondent did not call 911 or otherwise arrange emergent care for Patient K. Thereafter, Respondent ordered weekly IV vitamin and mineral infusions for Patient K for folate deficiency and other vitamin and mineral deficiencies. These were not supported by laboratory or other evidence of folate or vitamin deficiency.

SUBSTANDARD MANAGEMENT OF DIABETES

PATIENTS A,B,C, and K

1.9 Respondent failed to appropriately manage diabetes conditions experienced by Patients A, B, C and K. Potential eye diseases that may arise as a complication of diabetes include damage to the blood vessels in the retina (diabetic retinopathy) that

could, ultimately, result in blindness. Respondent did not refer these patients to an ophthalmologist for a standard yearly eye screening for diabetic retinopathy.

1.10 Respondent also failed to provide Patients A, B, C and K, with other standard diabetes health care maintenance such as yearly urine microalbumin screening for diabetic kidney disease (diabetic nephropathy) and yearly monofilament examination for screening of diabetic neuropathy.

PATIENTS H and J

1.11 Respondent failed to order yearly screening for diabetic retinopathy and nephropathy for Patients H and J. Respondent's treatment of Patient H increased his risk of kidney damage. Respondent actually scheduled long-term treatment with non-steroidal anti-inflammatory medication (nabumetone), in disregard of the added risk for kidney disease with this regime. Respondent failed to raise an objection to Patient H continuing 1500 mg of daily nabumetone, despite an associated risk of renal dysfunction in a patient predisposed to kidney disease.

MISMANAGEMENT OF OTHER MEDICAL PROBLEMS

1.12 Respondent failed to appropriately manage other medical problems experienced by Patients A, C, D, E, F, H, I, J and K.

PATIENT A

1.12.1 Respondent never clarified the history of past ovarian surgery reported by Patient A. As a result, Respondent never determined whether Patient A had a history of ovarian cancer or misplaced uterine tissue (endometriosis) that could require follow-up assessment and care or that might have been contributing to her abdominal symptoms.

1.12.2 When Patient A reported tenderness in her left and central upper abdomen (epigastric pain) on July 9, 2009, Respondent did not ask follow-up questions, or inquire about the presence of blood in the stool that could result from inflammation of the stomach (gastritis), or peptic ulcer disease. Respondent initiated a proton pump inhibitor, omeprazole (brand name Prilosec), which

inhibits acid production in the stomach. This medicine would be started for presumed gastritis or peptic ulcer disease, but, although Patient A's symptoms persisted for approximately two years, Respondent failed to refer Patient A for an upper endoscopy, as was indicated to determine whether she had gastritis or peptic ulcer disease and would guide additional indicated symptoms evaluation and treatment.

1.12.3 Patient A continued to report epigastric pain during the next two years, despite treatment with a proton pump inhibitor medication. Moreover, Respondent recommended that Patient A take Advil or aspirin for pain control in almost every visit. Non-steroidal anti-inflammatory medications were contraindicated in a patient with undiagnosed epigastric pain which might represent peptic ulcer disease or gastritis. In fact, gastritis or peptic ulcer disease could actually have been caused by the anti-inflammatory medications recommended by Respondent.

1.12.4 Respondent determined Patient A had Helicobacter Pylori (H. Pylori, a bacteria associated with gastritis and peptic ulcer disease), but did not order an H. Pylori serum test to confirm this diagnosis, as was indicated. When subsequent testing confirmed that Patient A had a positive serum H. Pylori reading, Respondent continued long-term treatment with double antibiotics, plus twice daily dosage of proton pump inhibitor, even though treatment for H. Pylori is only recommended to continue for 10-14 days. Respondent repeatedly ordered serum H. Pylori antibody test values, which remained positive for an extended period, although this is to be expected after exposure to H. Pylori. Respondent did not order stool antigen testing for H. Pylori, as would be necessary if he sought confirmation of infection resolution.

1.12.5 Respondent failed to appropriately assess Patient A's continual complaints of numbness and tingling. These were documented on February 10, 2009, and might have represented damage to the peripheral nervous system (peripheral neuropathy) or dysfunction of a nerve root of the cervical spine

ORIGINAL

(cervical radiculopathy). Respondent repeatedly treated these symptoms with trigger point injections instead of pursuing appropriate clinical assessment.

PATIENT C

1.12.6 On January 28, 2010, Respondent referred Patient C for a fiber-optic examination of the upper gastrointestinal track (gastroscopy), but failed to chart a basis for this referral. The review of systems reported no gastrointestinal (GI) symptoms, the abdominal examination was benign, and there was no assessment or treatment plan discussion of the need for gastroscopy. The referral form is dated November 3, 2009, requesting evaluation of the upper GI tract for complaints of increased frequency of upper central abdomen pain and difficulty swallowing. None of this information is included in the associated visit note. Appropriate questions about the swallowing difficulty were not included, such as whether the difficulty was with fluids only, or also solids.

1.12.7 Respondent had been treating Patient C with three ibuprofen daily, as indicated in the September 18, 2009, January 28, 2010, and many other notes. Respondent did not stop this anti-inflammatory medication, although it can cause or exacerbate gastritis or peptic ulcer disease that might have been the cause of Patient C's upper-central abdominal pain. Continuing the ibuprofen in these circumstances placed Patient C at risk for worsened gastritis or peptic ulcer disease, with possible GI bleeding complications. Regardless of the reported symptoms, long-term treatment with non-steroidal anti-inflammatory medication such as ibuprofen also placed Patient C at risk for renal insufficiency, which is of particular concern in a diabetic with pre-existing risks for kidney disease.

PATIENT D

1.12.8 Respondent failed to inquire about Patient D's brother's age at the time of diagnosis with colon cancer, which would have informed Respondent of the level of increased colon cancer risk for Patient D. Respondent did not discuss or provide colon cancer screening.

PATIENT E

1.12.9 Respondent inadequately managed Patient E's colon cancer. He did not request information related to the stage of Patient E's colon cancer to determine whether the tumor extended beyond the colon, or what follow-up surveillance and treatment was indicated. Respondent failed to identify what provider was managing Patient E's colon cancer and failed to adequately coordinate care with this provider. When Respondent determined Patient E to have abnormal weight loss and failure to thrive on September 10, 2009, (after an extended period of unattended medication-induced hyperthyroidism that could well have caused the weight loss), he referred her for gastroscopy and colonoscopy. The results of these studies were not referred to in Respondent's records for Patient E, and no copy of the reports of these procedures were obtained by Respondent.

1.12.10 Respondent failed to determine the basis for Patient E's reported weakness and fatigue. He consistently attributed these symptoms to vitamin and mineral deficiency without substantiating this conclusion. During this time frame, Patient E was struggling with profound under-active and then over-active thyroid function, which likely contributed significantly to her weakness and fatigue. The colon cancer may also have been a contributor, but Respondent did not check on its activity status. Congestive heart failure may have also played a role in the fatigue.

PATIENT F

1.12.11 Patient F had an eye condition that could lead to damage to the optic nerve and blindness (glaucoma), but Respondent failed to refer her to an eye clinic for eye pressure measurements to properly monitor and treat this condition.

//

//

PATIENT H

1.12.12 Respondent inadequately addressed Patient H's elevated blood pressure which was documented on numerous occasions. There is no indication of use of blood pressure medications, but only a reference to Respondent's discontinuation of losartan (Brand name Cozaar), a medication used to lower blood pressure and improve blood flow.

1.12.13 Respondent frequently documented patient education about a low cholesterol diet, but Patient H's low-density lipoprotein (LDL) level for the first three years of Respondent's oversight was higher than the goal for diabetics, which is less than one hundred. Respondent initiated the cholesterol lowering medication, pravastatin (brand name Pravachol), on September 27, 2006, when the patient's LDL level reached 136, but failed to assess the efficacy of treatment until one year later. Pravastatin is used to reduce levels of LDL, called "bad" cholesterol and triglycerides in the blood, while increasing levels of high-density lipoprotein (HDL), called "good" cholesterol. LDL level should have been monitored at approximately six weeks to determine any need for treatment changes.

1.12.14 Pravastatin was discontinued by June 20, 2007, without laboratory results or other indication or explanation in the chart. On September 14, 2007, the LDL was one hundred fifty eight, but no cholesterol-lowering medication was initiated. Pravachol was reinitiated on August 12, 2008, but without a note discussing the change. Patient H's LDL finally reached the goal of less than one hundred on May 7, 2009.

1.12.15 Test results indicated Patient H had anemia, with repeated hematocrit readings between 35-36, when normal range is 40-50. Respondent failed to mention, assess or work-up Patient H for anemia, which should have included looking for evidence of occult gastrointestinal bleeding. Patient H's hematocrit normalized August 2009.

1.12.16 Patient H also had an eye condition that could lead to damage to the optic nerve and blindness (glaucoma); however, Respondent failed to refer

ORIGINAL

him to an eye clinic for eye pressure measurements to properly monitor and treat this condition.

PATIENT I

1.12.17 Respondent failed to request prior records confirming Patient I's documented history of lupus. He also failed to obtain information on her prior work-up or rheumatologic recommendations for her care.

1.12.18 Respondent did not request records regarding Patient I's history of ovarian cancer, which was the diagnosis of most concern. In fact, Respondent failed to address the ovarian cancer history at all in his records. Patient I's history of cervical cancer was also not addressed. Patient I was noted to have had a pap smear performed in July of 2007, but the results were not documented. Respondent ordered IV vitamin and mineral infusions for Patient I's complaints of fatigue and malaise, but did not consider that a recurrence of her cancer might be contributing to these symptoms.

PATIENT J

1.12.19 Patient J reported having a chronic infection with Human T-cell lymphotrophic virus type II. HTLV-II is a retrovirus that can be associated with neurologic disorders and chronic pulmonary infections. Though this infection often coexists with HIV infection (as they share the same mode of transmission), Deborah Bryant, PA-C, did not test Patient J for HIV. Though Patient J was on disability, in part, due to neurologic manifestations of HTLV-II and she regularly noted symptoms of nerve discomfort, this issue was not addressed and the patient was not referred to a neurologist or infectious disease physician to further evaluate her neurologic symptoms and chronic HTLV-II infection.

1.12.20 Patient J also consistently reported a cough during clinic visits (noted in the review of systems sections of visit notes), but consideration of an HTLV-II related pulmonary infection was not entertained or evaluated. Though the

HTLV-II diagnosis was at the top of the list in her visit assessments, the infection was never addressed.

PATIENT K

1.12.21 Respondent's treatment of Patient K's low red blood cell count (anemia), revealed in initial her laboratory test results, was below the standard of care, and placed the patient at risk of gastrointestinal bleeding. Initially, lab test results revealed the presence of anemia with a hematocrit of thirty (30) and low mean corpuscular volume (MCV). Respondent determined iron deficiency anemia and initiated iron infusions without checking iron studies to confirm the diagnosis. Respondent should have, but failed to explore the source of the iron deficiency he assessed, to rule out causes such as gastrointestinal bleeding, for example. Respondent did not inquire about a history of intestinal bleeding and failed to order stool tests for blood to appropriately assess an etiology of iron deficiency anemia. Further, if gastrointestinal bleeding was present, Respondent should have discontinued Patient K's nonsteroidal anti-inflammatory naproxen treatment, which can cause gastrointestinal bleeding.

SUBSTANDARD USE OF LABORATORY TESTS

1.13 Respondent frequently ordered identical laboratory tests for patients without clinical justification, including Patients A, B, C, E and K. This included routine ordering of full chemistry panels, complete blood count testing and blood count differentials, lipid panels and inflammatory markers for patients, despite lack of symptoms and despite having previously obtained consistently normal values for these patients.

1.14 Respondent routinely ordered complete urinalysis testing without any stated urinary symptoms or other indication. When abnormal laboratory test results were received for patients, including Patients A, E, H, I and K, Respondent often failed to provide appropriate interpretation and indicated care. Respondent's substandard use

of laboratory analysis also included a failure to order lab tests for patients when such testing and analysis was indicated.

PATIENT A

1.14.1 Respondent's substandard use of laboratory tests for Patient A included his misuse of H. Pylori serum testing, and failure to obtain stool antigen testing described in paragraph 1.12.4. Respondent's inadequate treatment response to consistently abnormal LDL lab measurements for Patient A is described in paragraph 1.4.1.9 above. Respondent did not adequately address Patient A's abnormal electrocardiogram readings. Respondent failed to provide adequate treatment for Patient A's consistently high blood pressure readings. Respondent failed to order muscle enzyme testing to rule out niacin-induced muscle inflammation for Patient A. Other laboratory testing frequently was ordered without indication.

PATIENT B

1.14.2 Respondent frequently ordered identical laboratory tests for Patient B without clinical justification, for example, repeated testing of blood cell differentials and liver function tests despite consistently normal values.

PATIENT C

1.14.3 Respondent frequently ordered laboratory tests for Patient C that were not indicated.

PATIENT E

1.14.4 Patient E was directed to frequent lipid panel testing, although Respondent had discontinued her statin treatment and was not providing other treatment on the basis of the lipid panel results. In addition, urinalysis testing was repeatedly ordered for Patient E without indication.

PATIENT I

1.14.5 Respondent assigned Patient I with a diagnosis of thyrotoxicosis on September 19, 2007, but did not order thyroid function tests to evaluate this diagnosis. Lab results from October 2, 2007, indicated Patient I had normal thyroid function.

PATIENT K

1.14.6 Respondent failed to provide appropriate interpretation and care when abnormal laboratory result were received for Patient K. Respondent also frequently ordered laboratory testing that was not indicated.

SUBSTANDARD CHARTING

1.15 Respondent's charting of patient care are filled with repetitive and extraneous Electronic Health Record entries that obscure pertinent information to the point of making it difficult to glean reliable information about what happened at each patient visit. Respondent repeatedly cuts and pastes verbatim from prior and/or other patient visits, including fields for presenting illness, examination and treatment plan. The language is generally identical over subsequent visits and is also used between patients. Sometimes repetitive language directly contradicts individualized information that appears to be more tailored to a specific patient. For example, the following phrase appeared in many of Respondent's patient charts: "Since the beginning of symptoms she reports no changes in bowel movements or urination habits." This phrase generally followed a myriad of patient chief complaints that were, typically, completely unrelated to bowel and urination functions.

1.16 The medical charting by Respondent was so grossly inaccurate and misleading that it amounted to a misrepresentation of the patient consultations and evaluations, which compromised patient care from the standpoint of hampering other providers' ability to understand the patients' history and treatment. Repetitive cutting and pasting of entries obscures what information is unique to a particular visit. Replication of wording in numerous different patient charts demonstrates a lack of

individualized information. Respondent often failed to provide particularized information to address abnormal history or examination findings in the visit assessment and treatment portion of the electronic record. The irregularities in documentation render Respondent's chart notes of very little use, and it is unlikely that many of the items documented were actually performed. In addition to the deficiencies noted above, other examples of Respondent's charting deficiencies include, but are not limited to the following:

PATIENT C

1.16.1 Respondent stated a diagnosis of myalgia and myositis at Patient C's first visit without any documented basis. No specific area of muscle pain was noted, the musculoskeletal review of systems was negative, and the musculoskeletal exam results were normal. Much of the history and physical examination for the September 18, 2009, visit was copied directly from prior notes, and related to earlier matters, including a draining, swollen lower leg, resolved in an earlier appointment, instead of the problems reported by Patient C during the September 18th visit.

PATIENT G

1.16.2 Respondent included a diagnosis of hypertensive heart disease with heart failure in Patient G's regular visit assessments, but did not document symptoms or signs of heart failure.

PATIENT I

1.16.3 Respondent assigned Patient I a diagnosis of a sudden toxic worsening of hyperthyroid symptoms (thyrotoxicosis) on September 26, 2007, despite the lack of an abnormal thyroid exam or available thyroid function test results. Lab results from October 2, 2007, indicated Patient I had normal thyroid function.

ORIGINAL

BILLING WITHOUT JUSTIFICATION

1.17 Respondent failed to appropriately manage billing for patients, and overused treatment and counseling modalities to generate billing without clinical justification. Respondent routinely describes repeated exhaustive physical examinations at serial follow-up patient visits, even without a new history or complaint to suggest a need. For the same patient visits, Respondent typically describes providing in-depth lifestyle and dietary counseling to patients, which is then repeated verbatim at every subsequent patient visit. Such repetition of detailed information at every visit is not clinically indicated, but their coding would generate higher-level billings and misrepresented the level of care provided. It is unlikely Respondent would have time to complete such exhaustive examinations and counseling as described for each visit.

1.18 Respondent ordered frequent, identical laboratory tests without indication. He routinely ordered complete urinalysis testing without any stated symptoms or other indications. He routinely ordered full chemistry panels, complete blood count and blood count differentials, lipid panels and inflammatory markers without any indication, and in patients who had consistently normal prior values on these tests. This excessive laboratory testing, without indication, resulted in billing for venipuncture and laboratory testing that was not clinically relevant.

1.19 Respondent directed and scheduled blood draws as clinician visits instead of venipuncture visits to obtain blood samples from patients for laboratory testing. The blood was usually drawn by a health care assistant but was generally billed as a physician visit, rather than a lesser venipuncture code, as would have been appropriate when patients only presented for blood draws.

1.20 Respondent often scheduled and billed for a blood draw visit adjacent to an office visit, instead of obtaining the sample as part of the primary visit. The blood draw visits unnecessarily included review of patient histories, family and social histories, medications, allergies and vital sign checks. There was no clinical indication of a need for these and no orders were specified for these additional services, many of which closely followed an extended physician visit during which these items were also reviewed.

ORIGINAL

1.21 Respondent ordered unnecessary fasting lipid panels with most blood draws, despite the lack of medical indication for such testing. Inclusion of a "fasting" requirement ostensibly authorized billing for a separate early morning visit, although the blood could have been drawn during the regular patient visit with the doctor (which was billed separately).

1.22 Respondent billed telephone calls with pharmacies, patients, and patients' family members as direct patient care visits. Respondent notes from these phone calls included an "objective" section where direct exam findings are stated, although there was no patient present to examine.

1.23 Although without clinical justification, the Respondent's approach to IV vitamin infusions was accompanied by significant billing for associated procedures, visits and intravenous solutions.

2. ALLEGED VIOLATIONS

2.1 Based on the Alleged Facts, Respondent has committed unprofessional conduct in violation of RCW 18.130.180(1), (4), (13) and (16), which provide:

> **RCW 18.130.180 Unprofessional conduct.** The following conduct, acts, or conditions constitute unprofessional conduct for any license holder or applicant under the jurisdiction of this chapter:
>
> (1) The commission of any act involving moral turpitude, dishonesty, or corruption relating to the practice of the person's profession, whether the act constitutes a crime or not. If the act constitutes a crime, conviction in a criminal proceeding is not a condition precedent to disciplinary action. Upon such a conviction, however, the judgment and sentence is conclusive evidence at the ensuing disciplinary hearing of the guilt of the license holder of the crime described in the indictment or information, and of the person's violation of the statute on which it is based. For the purposes of this section, conviction includes all instances in which a plea of guilty or nolo contendere is the basis for the conviction and all proceedings in which the sentence has been deferred or suspended. Nothing in this section abrogates rights guaranteed under chapter 9.96A RCW;
>
> . . .

(4) Incompetence, negligence, or malpractice which results in injury to a patient or which creates an unreasonable risk that a patient may be harmed. The use of a nontraditional treatment by itself shall not constitute unprofessional conduct, provided that it does not result in injury to a patient or create an unreasonable risk that a patient may be harmed;

. . .

(13) Misrepresentation or fraud in any aspect of the conduct of the business or profession;

. . .

(16) Promotion for personal gain of any unnecessary or inefficacious drug, device, treatment, procedure, or service;

. . .

2.2 The above violations provide grounds for imposing sanctions under RCW 18.130.160.

//

//

//

//

//

//

//

//

//

ORIGINAL

3. NOTICE TO RESPONDENT

The charges in this document affect the public health, safety and welfare. The Executive Director of the Commission directs that a notice be issued and served on Respondent as provided by law, giving Respondent the opportunity to defend against these charges. If Respondent fails to defend against these charges, Respondent shall be subject to discipline and the imposition of sanctions under Chapter 18.130 RCW.

DATED: January 31, 2014.

STATE OF WASHINGTON
MEDICAL QUALITY ASSURANCE COMMISSION

MARYELLA JANSEN
EXECUTIVE DIRECTOR

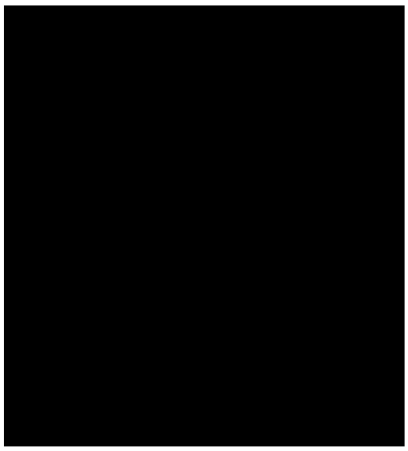
KRISTIN G. BREWER, WSBA #38494
ASSISTANT ATTORNEY GENERAL

ORIGINAL

CONFIDENTIAL SCHEDULE

This information is confidential and is NOT to be released without the consent of the individual or individuals named herein. RCW 42.56.240(1)

Patient A:
Patient B:
Patient C:
Patient D:
Patient E:
Patient F:
Patient G:
Patient H:
Patient I:
Patient J:
Patient K:



ORIGINAL