Chronic Distal Symmetrical Polyneuropathy

I. Overview of the Disorder

A. Pathology

Chronic distal symmetrical polyneuropathy is the most common painful diabetic neuropathy (PDN) in developed countries. The disorder is a debilitating consequence of diabetes mellitus (DM), and it is marked by diffuse, symmetrical, and ascending damage to the small peripheral nerve fibers. It is estimated that up to 50% of diabetes patients develop PDN symptoms at some point during their illness.\textsuperscript{i} However, despite its prevalence, much is still unknown about the pathophysiology of PDN. Given the complexity of DM as a systemic metabolic disease, along with the difficulties in classifying and managing a patient’s individual PDN symptoms, the disorder poses an ongoing therapeutic challenge.\textsuperscript{ii}

Diabetes mellitus is marked by elevated blood glucose levels, also known as hyperglycemia. When the food we ingest is converted into glucose, the pancreas releases the hormone insulin in order for the body’s cells to absorb the glucose molecules for energy. In DM, the body either does not produce a sufficient amount of insulin (type 1), or it develops a resistance to insulin due to chronic overproduction by the pancreas (type 2). Both types lead to a dearth of energy for the body’s cells in the short-term, and can eventually lead to systemic damage to the eyes, kidney, nerves, or heart.

Risk factors for type 2 DM include older age, obesity, physical inactivity, a family history of DM, prior history of gestational DM, impaired glucose tolerance, and race/ethnicity. African Americans, Hispanic/Latino Americans, American Indians, and some Asian Americans
and Pacific Islanders are at particularly high risk for type 2 DM. Type 1 DM is linked with autoimmune, genetic, and environmental factors. Patients with DM may complain of extreme hunger or thirst, frequent urination, unexplained weight loss, lethargy, sudden vision changes, numbness or tingling in the extremities, dry skin, and increased number of infections, or slow-healing sores. Diabetes is diagnosed with a blood test to confirm elevated blood glucose levels.iii

Diabetes is widespread in the United States, and neuropathic pain is its most common and costly complication. According the Centers for Disease Control and Prevention (CDC), over 29 million Americans have DM, up from 26 million in 2010. The CDC estimates that one in four people with DM in the US are unaware that they have the disease. Furthermore, more than one in three Americans over twenty-years-old have prediabetes, defined as elevated blood sugar levels that are approaching the levels seen in type 2 DM. Without a change in lifestyle, including weight loss and a moderate physical activity regimen, 15-30% of people with prediabetes will develop type 2 DM within five years.iv The prevalence of PDN is greater in type 2 diabetes patients, women, and South Asians.v And with approximately 50% of DM patients experiencing neuropathic pain, this symptom alone accounts for 27% of all direct diabetes healthcare costs.vi

Painful diabetic neuropathy impacts the distal peripheral nervous system (PNS). The PNS includes the spinal nerve roots, dorsal root ganglia, peripheral nerve trunks and terminal branches, cranial nerves (except for CN II), and the peripheral autonomic nervous system. Sensory nerves are categorized as either large myelinated fibers (responsible for vibratory sensation and proprioception) or as small unmyelinated fibers that sense pain and temperature. Both large and small fibers are responsible for light touch sensation.

Sensory nerve damage can occur at the level of the axon, myelin sheath, or dorsal root. An injury to the axon due to trauma is called Wallerian degeneration, where the axon and its
myelin sheath distal to the injury site are involved. Injury at the level of the dorsal root ganglion leads to destruction of that specific nerve’s peripheral and central processes. Damage to the myelin sheath (myelinopathies) may be acquired or hereditary, with hereditary myelinopathies presenting with diffuse symptoms and a slow progressive onset. Acquired myelinopathies result in segmental damage that leaves the axons intact, resulting in a complete recovery within a matter of weeks to months. By contrast, most toxic and metabolic injuries produce a “dying-back” or length-dependent pattern, marked by the distal axonal degeneration that characterizes PDN.vii

Distal small-diameter cutaneous nociceptive and autonomic nerve fibers are the most vulnerable to metabolic injury, and are therefore the first to be involved in PDN. However, these small-diameter fibers are also the fastest to regenerate, so it follows that the prediabetic and early diabetic patients are the most responsive to the nerve regenerative effects of exercise therapy. Irreversible damage to large myelinated fibers is a late complication of PDN, leading to more pronounced gait ataxia and DM foot ulceration due to the loss of protective touch and proprioceptive function. Insulin resistance, obesity, and hyperglycemia are linked with early damage to small unmyelinated axons, while glucose control is closely related to large fiber loss. Hypertension and smoking are also associated with increased risk of neuropathy.viii

B. Clinical Manifestations & Differential Diagnosis

Patients with PDN complain of a wide range of long-lasting and unremitting sensory symptoms. Sensory complaints specific to small fiber neuropathy include burning, stabbing, pins and needles, and shooting electric shock. These clinical manifestations occur primarily in the distal limbs, and they follow a symmetrical distal-to-proximal “glove-and-stocking” pattern.ix The pain may be exacerbated by non-painful stimuli (such as contact with bed sheets or socks,
known as allodynia), or experienced as an abnormal response to painful stimuli (such as a pinprick examination, known as hyperalgesia). The pain is generally worse at night and during weight bearing.

Large nerve fibers may also be involved in later stages of PDN, which is linked to parasthesia symptoms of intense distal tingling and buzzing. Patients may experience alteration of thermal perception thresholds, autonomic dysfunctions such as hot or cold limbs, and sensory loss. However, motor functions generally remain intact, leaving the patient with relatively normal muscle strength and reflexes. Chronic distal symmetrical polyneuropathy generally advances from an acute phase of a few months, where pain is at the highest intensity, to a long phase of progressive reduction of symptoms and sensory loss.

In comparison to patients with non-painful DM, PDN patients are also more likely to present with comorbidities and an increased risk of mortality. These individuals report significantly impaired quality of life, marked by difficulties with activities of daily living (ADL), anxiety, sleep disruptions, and depression.

Despite the painful nature of PDN, patients may not report their symptoms until they have progressed into severe complaints. For this reason, annual neuropathy screenings for at-risk individuals is advisable. Screenings are fast and simple, involving either a vibratory, pressure, or pain sensation test. Vibratory sensation is tested using a tuning fork on the dorsal bony prominence of the great toe, with the patient identifying when the vibration begins and ends. Pressure is assessed using a monofilament to the plantar surface, with the patient indicating when they detect the pressure sensation. And sharp touch sensation is tested with a pinprick. Each of these assessments have been shown to be effective in identifying neuropathy, and it has been determined that a single screening test is sufficient.
PDN is diagnosed clinically, based on the patient’s history of neuropathic pain and associated deficits, as well as tests and measures supporting the diagnosis. In order to definitively diagnose a patient with PDN, a systematic differential diagnosis must be applied. In the article “An Algorithm for the Evaluation of Peripheral Neuropathy,” A. Poncelet outlines a diagnostic approach to rule out other pathologies (see Appendix A for flowchart). To begin, neuropathy must first be ruled in. Some neuropathies present with motor weakness, just like motor and central nervous system disorders. Hysterical symptoms may also mimic a neuropathy. The first step is to rule out these other etiologies through a thorough patient history and examination. Once neuropathy is identified, other causes of peripheral neuropathy must also be excluded. Looking at the pattern of nerve involvement first, PDN is a polyneuropathy, which affects peripheral nerves diffusely and symmetrically. This pattern may be differentiated from those of focal and multifocal mononeuropathies, which originate in one site or limb asymmetrically, and may affect the entire nerve trunk, including the connective neural tissue and blood supply. Examples of focal mononeuropathies are compressive disorders, such as carpal tunnel, and they present with a specific asymmetrical pattern of symptoms. Other pathological conditions impacting the nerve externally include issues of ischemia or inflammation, also presenting asymmetrically. However, a chronic case of mononeuropathy may mimic PDN, as multiple nerves may be impacted as the disease progresses. A detailed subjective evaluation to chart the onset of the patient’s symptoms is necessary to differentiate between multiple mononeuropathy and PDN.\textsuperscript{xvii,xviii} Moving on to distinguishing between different symmetrical neuropathies, PDN is a distal “dying-back” disorder, predominantly impacting the feet where nerve fibers are the longest. This characteristic is shared by other metabolic and toxic neuropathies, in contrast with ascending but
proximal sensory neuropathies such as in HIV/AIDS. Likewise, PDN does not involve the cranial nerves (e.g., Lyme disease), impact the arms more than the legs (e.g., lead neuropathy), or present with an acute onset (e.g., nerve compression due to hemorrhage).xix

It is important next to identify whether the damage is occurring in the axon, myelin, or both. This is identified via nerve conduction studies (NCS) and electromyography (EMG). If axonal neuropathy is indicated, what follows is investigating which type of peripheral fibers is primarily implicated. Small fibers are implicated by the painful symptoms of PDN, with little to no motor involvement. This is in contrast with (1) pure sensory involvement, which may be due to drug toxicity or nutritional deficiencies; (2) predominantly motor involvement (e.g., Guillain-Barré); or (3) sensorimotor involvement (e.g., hypothyroidism, uremia).xx

Diabetes, AIDS, and alcoholism can all cause the hallmark “glove-and-stocking” pattern and small-fiber pain specific to PDN. However, involvement of autonomic fibers is less common in AIDS or alcoholism. Therefore, the presence of autonomic symptoms, such as hot or cold limbs, indicates PDN.xxi

Clinical diagnostic tools include a variety of neuropathic pain scales. The Michigan Neuropathy Screening Instrument (MNSI) and Neuropathy Disability Score (NDS) may be used both to screen for the disorder and to assess PDN at the disability level. Scales such as the Brief Pain Inventory and the Neuropathic Pain Questionnaire (NPQ) address the patient’s level of pain, while the Neuropathic Pain Symptom Inventory evaluates PDN symptoms present. Clinicians may also utilize neuropathic-specific quality of life (QOL) scales such as the Neuro-QoL.

The sensitivity of these screening measures is limited by the subjective nature of the scales attempting to measure pain and QOL. And because PDN symptoms are primarily driven by small nerve fiber involvement, the standard quantitative sensory testing protocol — vibration
perception, sensation loss, monofilament exam, and reflexes — is insufficient to conclusively diagnose PDN. More definitive and objective measures for PDN diagnosis and assessment include skin biopsies to measure nerve fiber density,xxii and corneal confocal microscopy to evaluate corneal nerve structures.xxiii

C. General Medical Management

Despite the widespread nature of this disorder and the medical community’s insights into its pathogenesis, many questions remain surrounding the prevention and treatment of PDN. Research supports the premise that hyperglycemia is the primary cause of painful neuropathy. However, there is only a moderate correlation between the two, calling into question whether other pathogenic mechanisms are involved. Current guidelines emphasize hyperglycemic control via medications, diet, and exercise to prevent the onset and progression of PDN. But research also confirms that this single parameter cannot guarantee success, particularly for type 2 diabetes patients.xxiv Because so much remains unclear about the pathophysiology of the disorder, no prophylactic therapy exists to prevent PDN, and all attempts at creating disease-altering PDN therapies have failed in clinical trials.xxv,xxvi A better understanding of the interplay between hyperglycemia and other neuropathy risk factors — including impaired insulin signaling, hypertension, and dyslipidemia — is of paramount importance for improved prevention and medical management.

Once the symptoms are present, it is also unclear which pathogenic mechanisms cause the pain. It has been suggested that nerve regeneration processes are responsible for the symptoms, but that assertion is not well supported by the literature.xxvii Due to this gap in knowledge, there is a dearth of medications specific for the combination of PDN symptoms,
leaving patients at risk for severe polypharmacy. It follows that, compared with DM patients without pain, individuals with PDN have approximately twice the healthcare costs. xxviii

Research indicates that the anticonvulsant Pregabalin is the most consistently effective pain-reliever for PDN, with usage marked by improved sleep, as well as other quality of life markers. Gabapentin and sodium valproate may also be considered as alternative anticonvulsants to manage pain. However, sodium valproate is not a first treatment choice for PDN, as its side effects include weight gain and decreased glycemic control. And it may cause birth defects, so it should be avoided by diabetic women of childbearing age. xxix The antidepressants mirtiptyline, venlafaxine, and duloxetine are also recommended pain treatments. Opioids that are used for PDN symptoms include dextromethorphan, morphine sulphate, tramadol, and oxycodone. And topical pain-relief agents include capsaicin cream, isosorbide dinitrate spray, and the lidoderm patch. xxx

D. Implications for PT Intervention

Regular aerobic and resistive exercise has been shown to improve neuropathy symptoms and promote distal axon regeneration. xxxi Along with treating pain symptoms, exercise therapy has been shown to target other complaints of PDN patients as well, including decreased muscle strength, decreased sensation in the feet, impaired postural control, exaggerated body sway, and increased falls risk. Foot ulcers can also be addressed in part via exercise training by improving circulation and oxygenation. xxxii

Myriad metabolic benefits are linked with regular exercise training, including improved insulin sensitivity and target organ perfusion, as well as reduced lipid and protein oxidation, production of free fatty acids, and microvascular damage due to humoral inflammation. Regular exercise helps to combat obesity and dyslipidemia, both of which increase neuropathy risk. xxxiii
Furthermore, exercise lowers the risk of heart disease and stroke, improves sleep quality, alleviates stress, improves circulation, helps to maintain joint flexibility, promotes real and perceived functional ability, improves balance to prevent falls, decreases fatigue, and strengthens the heart, muscles, and bones. Besides contributing to the promotion of overall health and wellbeing, it has been shown that these improvements greatly impact the progression of PDN symptoms and its comorbidities.

Patients with early diabetic or prediabetic neuropathy may be particularly responsive to the nerve regenerative capabilities of supervised exercise therapy. Because neuropathy is due to axonal destruction outpacing its regeneration, an increased regeneration rate promoted by intensive exercise therapy can especially benefit the early diabetic neuropathy population. The regenerative effects can also be seen in patients with chronic neuropathy; along with improving other clinically meaningful outcomes, exercise therapy helps to alleviate or slow the progression of PDN symptoms. Simultaneous improvements in BMI, serum lipids, and triglycerides in these patients correspond to a significantly larger reinnervation rate.

While intensive exercise training has been shown to produce effective results, these protocols are difficult for some patients to tolerate and adhere to. For these individuals, reducing sedentary behavior is the highest priority for physical therapy. Sedentary behavior is defined as “activities that do not increase seated or recumbent energy expenditure above the resting level.” As an alternative to prescribed exercise, decreasing sedentary activities alone has been shown to improve insulin sensitivity and lipid management. Prolonged sitting is especially detrimental to this population, as this is linked with decreased insulin sensitivity and elevated blood glucose levels. For this reason, it is important to promote incorporating light-intensity interruptions to sedentary behaviors, ideally in combination with prescribed exercise training.
Exercise therapy includes gait training, sensorimotor and resistance exercises, aerobic and flexibility training, and measures to improve physical activity overall. The greatest effects are seen when the aerobic and resistance programs lead to increased cardiorespiratory and muscular fitness. Because motivation and adherence are such fundamental barriers for this patient population, encouraging patients to undergo regular supervised group exercise training is an excellent approach.

A number of physical therapy modalities are also indicated for PDN patients. Electrotherapy has been shown to improve peripheral nerve microcirculation and muscular oxidative capacity, and to reduce pain symptoms. While different types of electrotherapy may be used, transcutaneous electrical nerve stimulation (TENS) may be particularly suited to this population, as portable devices allow for home treatments. Typically, TENS is recommended as a daily 30-minute treatment for 6-12 weeks, using a low frequency (4 Hz) and intensity (between 5 and 70 mA). Low-level laser therapy has also shown to promote positive effects, including improving cell and nerve tissue function, reducing inflammation, and pain relief.

Additional modes of exercise therapy have also been shown to benefit the PDN patient population. The traditional Chinese martial arts technique of Tai Chi involves slow balanced movements, breathing exercises, and meditation and concentration practices. This combined approach is particularly well suited to PDN patients, as it has been shown to improve skin vasculature, plantar sensation, and balance, along with reduced stress and enhanced coordination, strength, and flexibility. Yoga is another mind-body therapy that provides evidence-backed improvements for patients with PDN. Comprised of meditation, breathing exercises, and physical postures, yoga primarily helps to lower stress, which in turn impacts blood glucose levels. The postures are shown to increase strength, flexibility, balance, and coordination, as well as play a
role in decreasing inflammation and improved sleep quality. Yoga has been shown to help alleviate chronic pain symptoms, and to address the psychological challenges particular to chronic disease.\textsuperscript{xliiv}

The safety of exercise therapy for this patient population has been firmly established by the current evidence. It has been shown that moderate weight-bearing exercise is safe for all PDN patients (barring a severe foot deformity or peripheral vascular compromise), and that exercise therapy involving balance, walking, and lower extremity strength training does not increase the incidence of falls. On the contrary, exercise therapy is linked with a decreased falls risk for this patient population.

One of the most critical physical therapy intervention for this population is providing education. Physical therapists can also emphasize the importance of smoking cessation and diet counseling in successful outcomes to patients, referring them to resources and programs to facilitate these positive developments. And while patients experiencing pain may understandably be concerned about exercise being difficult to tolerate or worsening their symptoms, it is vital to educate patients that therapeutic exercise is linked with decreased pain symptoms. However, to avoid adverse events, it is of paramount importance that exercise therapy be prescribed and monitored by a physical therapist. It is important to monitor the physiologic parameters of PDN patients during exercise, including blood glucose, blood pressure, and heart rate. And minor adverse effects may occur in response to therapeutic exercise, including skin irritation, joint or muscle pain, hypoglycemia, and angina.\textsuperscript{xlv}
II. Case Scenario

A. Patient Profile

ASSESSMENT:
Evaluation (CPT 97001 – Physical therapy evaluation)

Mr. H is a 54-year-old African American male who presents with lower extremity pain that started approximately 6 weeks ago. He has been referred to my outpatient clinic by his primary care provider (PCP), with a diagnosis of early PDN. The pain keeps him awake at night, increases with weight bearing, is the worst by the end of the day, and is exacerbated by any pressure on the toes by his bed sheets. He describes the bilateral foot pain as sharp and needle-like. He characterizes his pain as ranging from a 4-7 out of 10, with 10 being the worst pain imaginable. He began taking pregabalin this week, and prior to this addition to his medications, Mr. H claims that he experienced 6-9/10 pain levels. Mr. H also complains of dry skin on his feet, and he has begun to experience mild numbness and tingling in his feet as of 2 months ago. Mr. H states that offloading and icing his feet in the evenings provides some relief from his symptoms.

His past medical history includes a type 2 DM (NIDDM) diagnosis 6 years ago, as well as an MI 5 years ago, CAD, HTN, morbid obesity, dyslipidemia, atrial fibrillation, and a sedentary lifestyle. He is currently using the following medications: pregabalin (for DPN), aspirin (to prevent blood clots), digoxin (for CAD), capoten (for HBP), glucophage (NIDDM, improves insulin action), and mevocor (for high cholesterol).

Mr. H lives with his wife in a second-story apartment without an elevator. He works as a computer programmer, drives to work and to social engagements, and is sedentary during the day. Mr. H states that the longest distance he regularly walks is the half-mile to his neighborhood park and back with his wife on the weekends (1 mile total), but he has been less willing to make
the trip recently secondary to pain during the half-mile return. He notices that recently he has been losing his balance more frequently during ADL. By the time he returns home in the evenings, he complains of 7/10 pain, leading to difficulties ascending his apartment stairs without taking 2-5 minute breaks. He states that he lacks the motivation to incorporate more physical activities into his lifestyle because of his pain and deconditioning, although his PCP has told him that he should engage in regular exercise. And while he claims he takes his medications as prescribed, Mr. H believes he can be more compliant with the diabetic diet outlined by his PCP.

His medical records indicate that his hemoglobin A1c tends to fall between 8% and 9% (with an A1c of 6.5% indicating DM). He does not currently smoke or drink alcohol. His blood pressure is 110/70, pulse rate is 75, and respiratory rate is 12. There are no murmurs or abnormal heart sounds. He has good dorsalis pedis pulses. He is alert and oriented x 4, and his speech is fluent. Motor strength is MMT grade 5/5 throughout, and all passive and active ROM are within functional limits. Mr. H has difficulty with tandem walking, and while his gait is slightly wide-based, it is steady.

Mr. H enjoys visiting the park with his wife, spending time with friends and family, and participating in his church choir. He states that his pain symptoms are limiting his ability to consistently continue with these activities. His goal is to decrease his pain so that he can improve his quality of life, and return to his prior level of function and sleep quality.

**Differential Diagnosis**

My patient evaluation supports Mr. H’s PCP’s diagnosis of early PDN, linked with his other health issues of NIDDM, hypertension, obesity, and dyslipidemia. Mr. H’s Michigan Neuropathy Screening Instrument (MNSI) results further supports the diagnosis, as a score
greater or equal to 7 has a 99% sensitivity. The following are Mr. H’s responses to the MNSI physical assessment and questionnaire:

**MNSI PHYSICAL ASSESSMENT**
1. **Foot inspection:** Patient presents with dry skin, and callouses on the heel and metatarsal heads.
2. **Vibration sensation:** Reduced (I feel vibration for 12 additional seconds)
3. **Muscle stretch reflexes:** Present
4. **Monofilament test:** Reduced sensation (7 correct responses out of 10 applications)

**MNSI QUESTIONNAIRE**
1. Are your legs and/or feet numb? **Yes**
2. Do you ever have any burning pain in your legs and/or feet? **Yes**
3. Are your feet too sensitive to touch? **No**
4. Do you get muscle cramps in your legs and/or feet? **Yes**
5. Do you ever have any prickling feelings in your legs or feet? **Yes**
6. Does it hurt when the bed covers touch your skin? **Yes**
7. When you get into the tub or shower, are you able to tell the hot water from the cold water? **Yes**
8. Have you ever had an open sore on your foot? **No**
9. Has your doctor ever told you that you have diabetic neuropathy? **Yes**
10. Do you feel weak all over most of the time? **No**
11. Are your symptoms worse at night? **Yes**
12. Do your legs hurt when you walk? **Yes**
13. Are you able to sense your feet when you walk? **Yes**
14. Is the skin on your feet so dry that it cracks open? **No**
15. Have you ever had an amputation? **No**

**FINAL SCORE:** 7 (“Responses of ‘yes’ to items 1-3, 5-6, 8-9, 11-12, 14-15 are each counted as 1 point. A ‘no’ response on items 7 and 13 counts as 1 point. Item #4 is a measure of impaired circulation and item #10 is a measure of general asthenia and are not included in scoring”)

**Diagnosis**

- **Pattern 5G:** Impaired Motor Function and Sensory Integrity Associated with Acute or Chronic Polyneuropathies
- **ICD-10 code E11.42:** Type 2 diabetes mellitus with diabetic polyneuropathy
**Prognosis**

Due to the recent onset of pain symptoms, the patient has good rehabilitation potential. Physical therapy treatment, in conjunction with diet and exercise modifications, is likely to reduce his symptoms and increase his endurance and balance.

**PLAN:**

**Problem List**
1. Pain with ambulation >0.5 mile and ascending 1 flight of stairs
2. Pain level at worst 7/10
3. Decreased endurance secondary to pain
4. Impaired sensation and vibratory sense
5. Loss of balance during daily activities

**Goals (10 weeks)**
1. Increase ambulation tolerance to >1 mile and >1.5 flights of stairs
2. Decrease worst pain level to 5/10
3. Increase balance to achieve negative Romberg Test

**Planned Intervention**

Will see patient 1x/week for 10 weeks. Will instruct patient on activity modification, and educate on skin care, diet, and exercise. Will perform gait training, balance training, resistance training, and aerobic training. Will teach HEP that will improve balance, to decrease the risk of falling. Will refer patient to dietician. Will refer patient to cardiac rehab group exercise program 2x/week for 10 weeks for resistance and aerobic training, to increase strength and endurance, and to decrease pain symptoms.
Treatment Plan

- CPT 97110 - Therapeutic exercise for strength, endurance, ROM and flexibility
- CPT 97116 - Gait training (includes stair climbing)
- CPT 97112 - Neuromuscular re-education of movement, balance, coordination, kinesthetic sense, posture, and/or proprioception for sitting and/or standing
- CPT 97535 - Self-care/home management training (e.g., activities of daily living [ADL] and compensatory training, meal preparation, safety procedures, and instructions in use of assistive technology devices/adaptive equipment), direct one-on-one contact
- CPT 98960 - Education and training for patient self-management by a qualified, nonphysician health care professional using a standardized curriculum, face-to-face with the individual patient (could include caregiver/family)
- CPT 97032 - E-stim manual
- CPT 97799 - Low level laser therapy
- CPT 97010 - Cold pack therapy

B. Recommended PT Program

1. Education

Mr. H is a male over age 45 with a previous history of MI, which deems him a high-risk patient for heart disease according to ACSM. I will emphasize that, by making lifestyle and diet changes to help address his dyslipidemia, hypertension, type 2 diabetes, and sedentary lifestyle, Mr. H can work to improve his PDN symptoms, decrease his modifiable cardiac risk factors, and minimize his reliance on his current medications. I will also explain that he has excellent potential to improve his pain levels: the onset of his symptoms is recent, indicating that the peripheral nerves have only just begun to “die back,” and they can regrow faster with a modified diet and exercise therapy.

I will recommend Mr. H revisit a nutritionist to support the healthy diet adjustments he needs. Weight loss, the DASH eating plan, a dietary sodium reduction, limiting alcohol consumption to a maximum of two drinks per day, and continuing with regular aerobic exercise will help to address the Mr. H’s HTN and elevated cholesterol levels, in addition to his
We will also emphasize good foot hygiene, proper footwear, and skin monitoring to prevent diabetic foot ulcers. Regular skin inspection is of paramount importance: small cracks and ulcers can have huge implications for diabetic patients, quickly progressing into ulcers that may eventually require amputation. Mr. H will be instructed in moisturizing to prevent skin dryness, as this also weakens the integrity of the skin. And he will need to wear protective footwear at all possible times to prevent injury.

2. Aerobic & Resistive Exercise

For high-risk cardiac disease patients such as Mr. H, ACSM recommends 3-7 sessions of aerobic exercise per week for 20-60 minutes as a goal, with a 5-10 minute warm-up and cool-down. It will also be important to incorporate resistance training into Mr. H’s program, to promote axonal regeneration, improve PDN symptoms, and to increase his strength and endurance. Mr. H should follow the ACSM guidelines of 2-3 sessions of resistance training per week. He will begin with light loads, where 11-15 reps are completed comfortably, translating to an RPE of 11-13.

In addition to his weekly physical therapy session, I will recommend two additional aerobic and resistive training sessions per week as tolerated. I will refer Mr. H to a cardiac rehab gym’s group exercise program to supervise and facilitate this aerobic and resistive exercise protocol outside of our therapy sessions. In addition, I will encourage Mr. H to take regular breaks from his sedentary lifestyle, and to use a pedometer to monitor his daily step-count. Ideally he could comfortably work up to the recommended 10,000 daily steps.

During our therapy session, we will begin with resistive exercise. I will increase the loads by 5% increments, monitoring Mr. H to make sure he does not strain, use a tight grip, or exhibit any negative symptoms. We will perform 8-10 exercises, 1-4 sets of 11-15 reps, to train each
major muscle group. As recommended by ACSM, the progression will be slow, increasing the load on arms by 2-5lbs per week, and 5-10lbs per week for the legs. To incorporate this program, I will set up 3-5 stations focusing on isometric and eccentric resistance exercises, which are associated with higher muscle tension and less metabolic demand.

An aerobic exercise session will follow, where Mr. H will use an aerobic machine for 20-60 minutes as tolerated, taking breaks whenever needed. The ACSM recommendation is to incorporate both upper and lower extremities in the exercise prescription, so I would include equipment such as the combination upper/lower extremity ergometer, the elliptical, and the treadmill. I will explain the “talk test” to Mr. H, that during this moderate-intensity exercise he should be able to talk, but not sing. He will have to save the singing for choir performances! I will also instruct Mr. H on stretching exercises for the major muscle groups to conclude the session.

During therapy, I will monitor him for signs of hypoglycemia, along with his blood glucose, blood pressure, and heart rate. And if his pre- or post-exercise blood glucose is <100mg/dl, he should immediately consume 20-30g of carbohydrates.

3. Other Activities

I would recommend Mr. H participate in other group movement practices, including Tai Chi and yoga. Tai Chi’s balance, coordination, and concentration practices will help to train Mr. H’s proprioception, balance, strength, flexibility, and plantar sensation. Yoga is another recommended activity to work on these same elements of therapy. As community exercise practices, both Tai Chi and yoga capitalize on the “group effect” to promote adherence to a consistent lifestyle change. And both practices promote stress reduction, which positively impacts blood pressure and blood glucose levels.
4. **Home Program**

In addition to his two additional aerobic and resistance training sessions at the cardiac rehab facility, I will instruct Mr. H on a simple and short home exercise program to help address his balance and gait issues (see Appendix B). As Mr. H’s balance improves, these exercises can be progressed in difficulty by incorporating head movements and closing the eyes.

We will also incorporate a daily electrotherapy (TENS) treatments into Mr. H’s home program, in order to promote peripheral nerve microcirculation and muscular oxidative capacity, and to reduce pain symptoms. Using a portable home TENS device, I will recommend a daily 30-minute treatment for 6-12 weeks, using a low frequency (4 Hz) and intensity (between 5 and 70 mA).

5. **Modalities**

In addition to his daily TENS home program, we will include low-level laser therapy to his PT sessions, in order to further improve cell and nerve tissue function, reduce inflammation, and relieve pain.
Appendix A: An Algorithm for the Evaluation of Peripheral Neuropathy
Appendix B: HEP

SINGLE LEG STANCE - SLS

With a stable chair in front of you for support if necessary, stand on one leg and maintain your balance.

Progressions: (1) move head left and right, (2) move head up and down, and (3) close the eyes.

Repeat on other leg.

TANDEM STANCE AND WALK

With a wall next to you (to provide support if necessary), stand with one foot directly in front of the other, so that the toes of one foot touches the heel of the other. Ensure that the area in front of you is free from any obstacles.

Maintain your balance.

Progressions: (1) move head left and right, (2) move head up and down, (3) close the eyes, and (4) take steps with your heel touching your toes with each step.

Repeat with other leg leading.

SIGNLE LEG STANCE - CLOCKS

With a wall next to you (to provide support if necessary), start by standing on one leg and maintain your balance. Imagine a clock on the floor, where your stance leg is in the center.

Then, lightly touch position 1 as illustrated with your non-stance foot. Then return that leg to the starting position.

Next, touch position 2 and return. Continue this all the way to position 6.

Maintain a slightly bent knee on the stance side.

Repeat on other leg.
References


Herman, WH et al. Use of the Michigan Neuropathy Screening Instrument as a Measure of Distal Symmetrical Peripheral Neuropathy in Type 1 Diabetes: Results from the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications. (2012) *Diabetic Medicine, 29*(7), 937-944.


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**Endnotes**


viii Singleton, p.2.

ix Javed, p.15.

x Javed, p.3.


xiv Lawson, p.7.
xv Lawson, p.6.
xvi Huizinga, p.6-7.
xvii Poncelet, p.756.
xviii Marchettini, p.175-6.
xix Poncelet, p.759-60.
x x Poncelet, p.756-7.
x xi Poncelet, p. 758.
x xii Singleton, p.2.
x xiii Javed, p.16.
x xv Singleton, p.3.
x xvi Lawson, p.15.
x xvii Singleton, p.17.
x xii Poncelet, p.759-60.
x xiii Singleton, p.756-7.
x xiv Poncelet, p.758.
x xv Singleton, p.17.
x xvi Newton, p.1.
x xvii Singleton, p.1.
x xviii Lawson, p.6.
x x x Law, p.1.
x x x Singleton, p.1.
x x xiv Lawson, p.64.
x x x v White, p.5.
x x x vi White, p.1.
x x x vii Singleton, p.3.
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x x x x ACSM (2010), p.219-221.


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