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# Diabetes-associated cardiac autonomic

*Abstract: Cardiac autonomic neuropathy (CAN) is an underdiagnosed cardiovascular complication associated with diabetes. NPs are in a pivotal position to screen patients for CAN. As the incidence of diabetes increases in an aging population, NPs can help prevent complications associated with diabetes and CAN.*

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Diabetes continues to be one of the most prevalent chronic illnesses addressed by healthcare providers. The CDC estimates in the 2017 National Diabetes Statistics Report that 30.3 million people (9.4% of the US population) have diabetes and 7.2 million (23.8%) of them are undiagnosed.[1] Current data and trends using fasting glucose levels and glycosylated hemoglobin (A1C) tests indicate a significant increase in the number of individuals diagnosed with diabetes across all age groups. Type 2 diabetes mellitus (T2DM) is the most common form of diabetes and its incidence is increasing in children and adolescents.[1] The CDC estimates over 193,000 children and adolescents age 20 and younger have T2DM.[1] However, many individuals with prediabetes or early T2DM are asymptomatic, despite the havoc wreaked by elevated glucose levels on both the macrovascular and microvascular systems.[2] Notwithstanding, with the increasing availability of diabetes treatment, it remains the seventh-leading cause of death in the US with a rate of 24.7 per 100,000 individuals.[1]

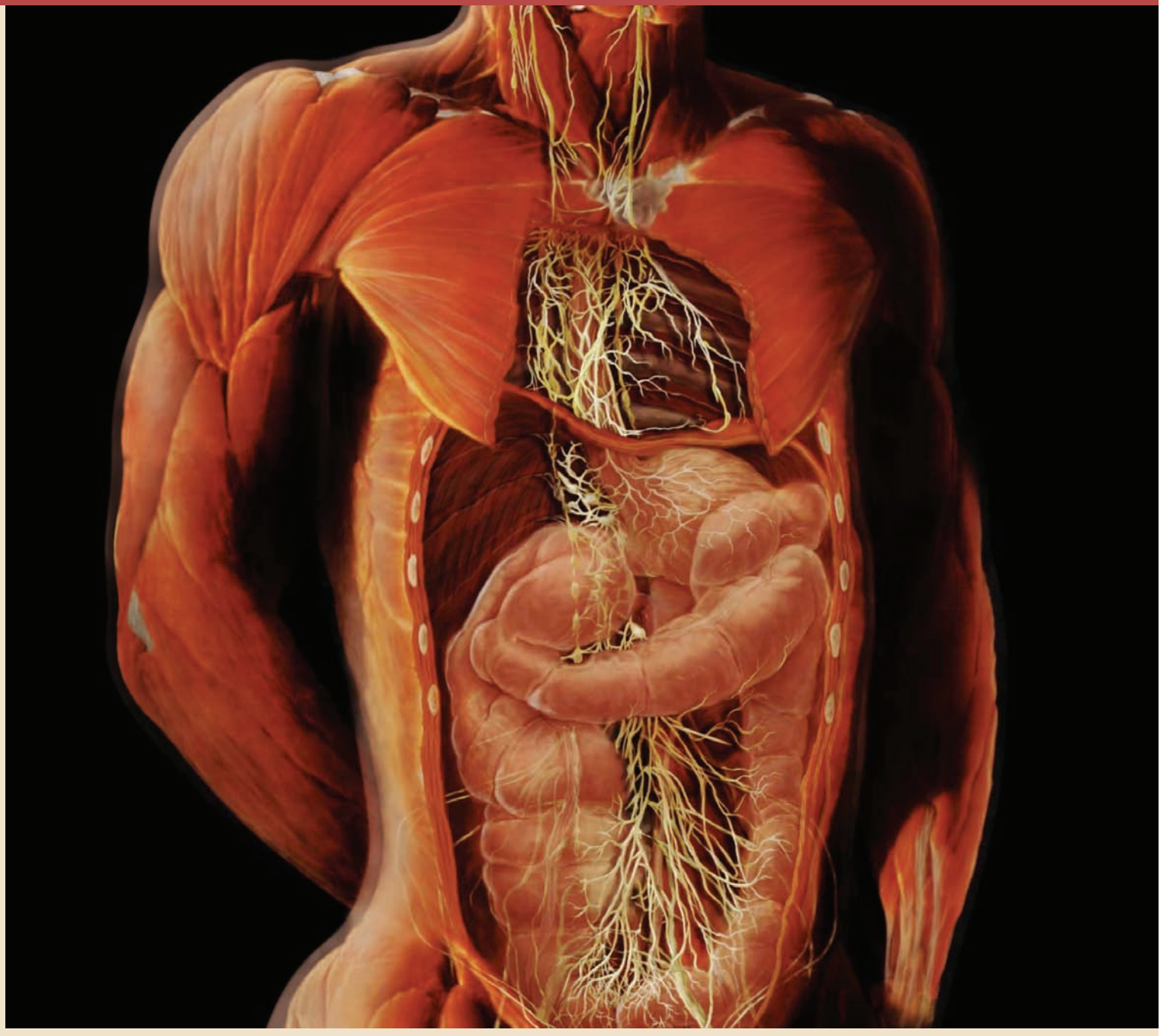
The expanding number of individuals with diabetes, both diagnosed and undiagnosed, is a major concern for NPs. The dynamic relationship between vascular complications and a climbing mortality heightens the need for solutions. Despite the growing demand for early identification of those with prediabetes, patients continue to present with a variety of complex health problems related to beta cell dysfunction resulting in glucose intolerance.[3] This national issue affects patients of all ages throughout the lifespan.

Across the literature, multiple studies have highlighted the correlation between the duration of diabetes and damage to the cardiovascular system.[2-5] Cardiac autonomic neuropathy (CAN) is an underdiagnosed cardiovascular complication associated with diabetic autonomic neuropathy.[3] CAN has a significant impact on morbidity and mortality for patients with diabetes.[3] There is a slightly higher incidence of CAN in patients with T2DM versus type 1 diabetes mellitus (T1DM). This is partially because patients with T2DM are typically older and have a higher incidence of heart-related comorbidities.[3] Cardiovascular disease (CVD) is still the leading cause of death in the US, killing over 366,800 individuals each year, and CAN-related mortality is 27% to 56% over 5 to 10 years after diagnosis.[6,7] CAN is a complication of long-standing diabetes and



**Keywords: autonomic nervous system, cardiac autonomic neuropathy, cardiovascular disease, continuous glucose monitoring system, diabetes mellitus, heart rate variability, resting heart rate**

# neuropathy



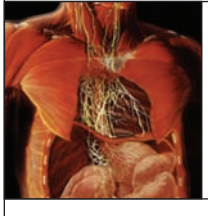
is associated with cardiovascular risk factors such as obesity, smoking, hypertension, and hyperlipidemia. One key commonality between CAN and CVD is a higher incidence of hypertension.[8]

CAN results in patients' heart rates not varying or increasing with moderate exercise, sleep, or stress.[9] This usually indicates complete denervation and a higher risk of a cardiovascular event.[9] Denervation can result in life-threatening, asymptomatic ischemia leading to a possible silent myocardial infarction (MI) in this patient population.[9]

NPs must be on heightened alert in recognizing early vascular changes and neuropathies affecting mortality as well as quality of life. Despite the vast amount of research on diabetes mellitus, a gap exists between diabetes diagnosis and the early detection of CAN.[2,3]

CAN, a classification of diabetic autonomic neuropathy, is associated with a fivefold increased risk of cardiac death.[2] The incidence may even begin in childhood with increasing rates of obesity at an earlier age, impaired glucose tolerance, and progressive insulin resistance.[4,5] The triad of risk factors associated with CAN include the duration of diabetes, glycemic control, and cardiovascular risk factors.[2] If left untreated, these risk factors will result in CVD at an earlier age.[4,5]

The mechanisms contributing to CAN are complex, but NPs are in the position to identify individuals at risk for this devastating disease. Addressing the increasing incidence of childhood obesity will play a key role in reducing the incidence of T2DM at an earlier age. NPs can help prevent this issue by focusing on early detection of T2DM in pediatric and adolescent patients. In a recent study by Andersen and colleagues, researchers found that hyperglycemia, obesity, and hypertriglyceridemia were positively related to CAN.[4] They identified that higher A1C levels, weight, body mass index, and triglyceride levels were correlated not only with T2DM but also with CAN.[4] In addition, heart rate variability (HRV) was found to be an independent prognostic indicator of CAN and sudden cardiac death.[3,10] HRV is defined as the measurement of time between each heartbeat.[3] Heart rate is based on R to R wave measurements during paced deep breathing.[3] Positive variability findings reflect an inability to control the pacing of the heart rate with deep breathing.[2]



***The development of CAN is multifactorial, and forms of diabetic neuropathies may even precede the diagnosis of diabetes.***

The purpose of this article is to educate NPs in early detection and treatment of CAN as well as to discuss avenues for prevention. NPs are in the pivotal position of educating patients on the effects of long-standing diabetes in the development of CAN.

## Pathophysiology

To prevent and/or treat CAN, NPs must understand the pathophysiology of this devastating complication. The development of CAN is considered multifactorial, with some evidence indicating that forms of diabetic neuropathies may even precede the diagnosis of diabetes.[5] This evidence is related to the underlying pathogenesis of the effects inflammation has on the autonomic nervous system.[5] CAN is linked more specifically to the loss of parasympathetic function but may affect the sympathetic nervous system over time.[11] This loss of parasympathetic function causes the initial presentation of CAN, which is an increase in the patient's resting heart rate.[11]

Autonomic neuropathy associated with CAN is the result of damage to the peripheral nerves associated with diabetes.[11] The peripheral nerves contain not only sensory and motor but also autonomic fibers. Autonomic nerves are small in diameter and prone to damage from the effects of inflammation related to hyperglycemia.[11]

Damage to the autonomic nervous system resulting in CAN affects both the parasympathetic and sympathetic nervous system, causing a struggle to maintain cardiac function.[3,9,11] Literature indicates these two systems often work together to produce life-sustainable functions including control of the heart rate.[3,9,10] For instance, the sympathetic nervous system is known to increase the heart rate, while the parasympathetic nervous system slows the heart rate.[3,9] If both are stimulated at the same time, cardiac output is increased. This response will slow the heart rate, allowing for greater cardiac filling.[3,9,10] Evidence indicates that damage to one system affects the everyday functions needed to maintain homeostasis.[9]

When evaluating and conducting a history and physical exam for possible diabetic autonomic

dysfunction, NPs should ask patients about pre- or syncopal episodes, orthostatic hypotension, heat intolerances, or dysfunction of the bowel, bladder, or sexual organs. In addition, any known history of retinopathy or nephropathy highlights the need to screen for CAN.[9]

## Screening and diagnosis

The American Diabetes Association (ADA) recommends screening for symptoms of neuropathy beginning 5 years after T1DM diagnosis and at the time of T2DM diagnosis.[9,12] Screenings underscore the effects of glycemic excursions on the microvascular system, which typically result in damage to the kidneys and eyes.[12] If CAN is detected early in disease progression, the damage to the cardiovascular system may be reversed.[3] Therefore, screening should occur in all subsequent visits, taking into consideration glycemic control, duration of the diabetes, and complaints related to neuropathic damage.[9,12]

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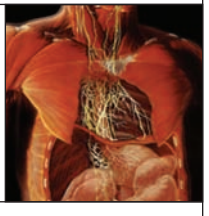


The symptoms related to peripheral neuropathies include, but are not limited to, numbness or tingling in the feet, legs, and hands; frequent indigestion, nausea, or vomiting; dizziness or faintness upon standing or sitting; and problems with urination or erectile dysfunction.[12] Screening should also include symptoms more indicative of CAN; three key complications of CAN include resting tachycardia, orthostatic hypotension, and hypoglycemia unawareness.[13] Patients with hypoglycemia unawareness do not receive appropriate signals of low blood glucose levels from the autonomic nervous system in a timely manner.[12,13] As a result, these patients may present to acute care facilities after an episodic fainting event.

One of the essential steps in diagnosis is to assess for signs and symptoms of cardiovascular function through heart rate and BP measurement, ECG testing, Holter ECG monitoring, baroreflex sensitivity measurement, heart imaging studies, cardiovascular sympathetic tests, and a heart rate turbulence (HRT) test.[2] The HRT test is a newer cardiac electrophysiologic test that examines baroreceptor sensitivity in the parasympathetic nerves of the autonomic nervous system.[2] Although the HRT test has value, it is rarely used in practice.

The ADA recommends testing for CAN using the HRV test.[13] Across multiple epidemiologic studies, tachycardia is a major risk factor for cardiovascular death.[7,11] Tachycardia is a resting heart rate greater than 100 beats/minute often recorded on home monitoring systems.[7,11] HRV occurs when the heart rate varies with movement or positional changes.[11] In patients with CAN, there is a noticeable absence of HRV. Screening for HRV consists of the provider or practitioner monitoring for a decreased or fixed heart rate in response to deep breathing signaling a disruption in the parasympathetic and sympathetic nervous system.[13] However, detection of a variance in HRV is not always existent in all patients with CAN, making the diagnosis somewhat difficult.[13]

Other symptoms observed in patients with CAN include postural hypotension, dizziness, faintness, or visual impairment upon standing.[3,13] These symptoms are more likely to occur 2 to 4 hours after meals because of postprandial hypotension.[3,13] Additional screening to consider would include a 24-hour ECG test to check for lengthening of the QT interval due to ventricular arrhythmias.[2,14]

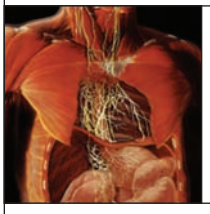
There are three quick cardiovascular screening tests that are easy to perform in the outpatient office setting. The NP can monitor the patient's heart rate during deep breathing (HRV), lying-to-standing, and during the Valsalva maneuver.[15] Another test, the cold pressor test, is performed by immersing the patient's hand into a container of ice water for 1 minute and then measuring changes in BP and heart rate to check vascular response.[15] The cold pressor test has been used to predict major cardiac events and heart rate excitability.[15]

Postural hypotension screenings are also very easy to perform in any office setting. Criteria for postural hypotension include monitoring for a fall in systolic or diastolic BP 1 minute after standing of greater than 10 to 20 mm Hg.[2] A cardiology referral may be indicated if screening results are questionable or abnormal. (See *Potential assessment findings in patients with CAN.*)

## Health consequences

Across the literature, evidence indicates that patients with diabetes and CAN have a significantly higher

incidence of morbidity and mortality compared with adults without CAN.[3] Unfortunately, CAN has been associated with cardiac disorders including myocardial ischemia and MI, orthostatic dysfunction, heart failure, and arrhythmic disorders.[3,14] Therefore, patients with CAN may have a poor quality of life.[14] NPs can reduce the health consequences of CAN through early identification and risk management.



***Continuous glucose monitoring systems aid in reducing the risk of hypoglycemia due to intensive insulin therapy.***

Additional health issues associated with CAN are due to hyperglycemia resulting in vessel and nerve damage throughout the body.[12,14] Vessel and nerve damage often occur in the major organs of the body, including the eyes, kidneys, and heart.[12,14] Patients usually present with changes in vision because of damage to the eyes or complaints of tingling or burning in the extremities because of neuropathy.[12,16] Prolonged hyperglycemia damages the vessels in the retina, leading to hemorrhages, exudates, and retina swelling.[12,16] Edema leads to retinal damage, including blindness.[12,16] NPs play a central role in educating patients and family members on the potential health effects and the role of nonadherence to prescribed treatments that improve quality of life.

## Treatment

One of the main goals of treatment for CAN is symptom control and slowing the progression of the condition. This requires a continual focus on the balance between lifestyle and medication management due to the complexity and progression of this type of neuropathy. Individualized plans of care are based on medical history, symptomology, and diagnostic results. The complexity of this complication requires a multidisciplinary approach to treatment.

Although glycemic control is the mainstay of treatment for CAN, research has shown a greater reduction in morbidity and mortality when combined with interventions focused on controlling other cardiovascular risk factors.[3] Caution must be taken with intensive glycemic control in patients with long-standing uncontrolled diabetes due to increasing mortality associated with strict glycemic control.[12,17,18]

Several studies have provided evidence that mortality rates increase due to sudden death in patients with CAN.[11,12] NPs can affect morbidity and mortality by contributing as a member of an interdisciplinary team. This requires the NP to make frequent adjustments to individualized plans of care.

**Pharmacologic therapy.** Medication management currently being studied and recommended in clinical trials is based on the pathogenesis of this devastating illness.[3,12,17-19] The Diabetes Control and Complication Trial (DCCT) reported that the incidence of CAN may be reduced by 37% with intensive management.[18] This requires multiple daily injections of long-acting and short-acting insulin with home glucose monitoring at least four times per day.[12] The Epidemiology of Diabetes Interventions and Complications (EDIC) trial was a subsequent study with the participants of the DCCT.[17,18] The EDIC trial evaluated participants 13 to 14 years after completion of the DCCT.[12,17,18] Participants with intensive insulin management continued to have lower rates of autonomic neuropathy (28%) compared with the conventional therapy group (35.2%), respectively.[17] This indicates the need to focus intently on pharmacologic therapy to target glycemic control throughout the lifespan of a patient. However, in patients with T2DM, pharmacologic therapy must also include treatment to reduce cardiovascular risk at the time of diagnosis.[19]

Medication therapy for T1DM focuses on insulin management in combination with lifestyle modifications. In contrast, patients with T2DM have more options. Initially, some patients may need lifestyle

### Potential assessment findings in patients with CAN[3,11,13]

- Resting tachycardia (higher resting heart rate >78 bpm) is a risk factor for CVD.
- Rise in heart rate over time
- Drop in heart rate volume
- Fixed heart rate that does not vary with exercise or sleep
- Exercise intolerance
- Orthostatic hypotension with standing
- Orthostatic tachycardia or bradycardia with standing
- Prolongation of QT intervals
- Increase in nocturnal BP
- Silent MI or cardiac denervation syndrome
- Gold standard: cardiovascular autonomic reflex tests
- Ankle-brachial index >1.2, and/or Doppler sonography

**Treatment options for T2DM**[17,18,20,21]

| | A1C% reduction | Hypoglycemia risk |
|---|---|---|
| **Exercise** | 0.66 | Low-moderate if on sulfonylureas and/or insulin* |
| **Meal planning** | Varies | Low |
| **Medications** | | |
| ***T2DM:*** | | |
| • Metformin | 1.0–2.0 | Low |
| • Sulfonylureas | 1.0–1.5 | Moderate* |
| • Thiazolidinedione | 0.8–1.0 | Low |
| • Dipeptidyl peptidase IV inhibitors | 0.5–0.8 | Low |
| • Glucagon-like peptide-1 mimetic | 0.6–0.8 | Moderate |
| • Sodium-glucose transport protein 2 | 0.5–1.0 | Low |
| • Insulin | >2.5 | Moderate* |
| ***T1DM:*** | | |
| • Insulin | >2.5 | Moderate* |
| **Monitoring** | | |
| ***Self-monitoring blood glucose:*** | Targeting goals: | |
| • One to two times a day at staggered times for T2DM | Fasting, 70–130 mg/dL | |
| • Four or more times a day with meals and bedtime for T1DM | 2-hour postprandial <180 mg/dL | |
| ***Continuous glucose monitoring:*** | Bedtime, 90–150 mg/dL | |
| • Constant monitoring of glucose levels in the interstitial space | | |
| • Monitor with alarms for high and low parameters | | |
| • Monitor without alarms requiring active participation | | |

*Risk increases if patient is not monitoring blood glucose levels frequently.

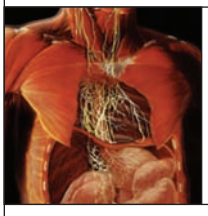
modification with the addition of metformin (if age-appropriate).[12] Additional medication therapy for patients with T2DM should be considered if glycemic goals are not met within 2 to 3 months.[12] The Standards of Medical Care in Diabetes established by the ADA continue to encourage NPs to add a second or third agent at subsequent visits if glycemic control is not achieved.[12] NPs should also consider the risk of hypoglycemia. The ADA promotes an individualized plan of care including frequent adjustments to medication regimens.[12] The "caution" within the Standard of Care is to avoid provider inertia, which can increase the risk of complications associated with poor glycemic control.[12] Clinical inertia refers to a provider opting not to adjust the medication using the ADA Standards of Care.[12] (See *Treatment options for T2DM.*)

It is critical that NPs understand the indications for each class of oral diabetes medications as well as their mechanisms of action.[20] The provider plays a central role in promoting the value of pharmacologic therapy, including the use of insulin in T2DM to prevent complications as well as intensive insulin therapy in T1DM. However, patients need to be instructed on the importance of lifestyle changes to assist in maintaining glycemic control.[12]

Increasing data support the use of continuous glucose monitoring (CGM) systems to aid in reducing the risk of hypoglycemia due to intensive insulin therapy.[22] Therefore, as a provider assists patients in maintaining glycemic control, the dangers of hypoglycemia may outweigh the benefit of tight glycemic control, especially in patients with hypoglycemia unawareness.[7,13,18] CGM systems require education for both patients and their families.[12,22] Several models on the market provide the ability to detect hypoglycemia.[22] CGM systems have the added benefit of offering additional data to providers to assist them in making better-informed decisions regarding diabetes management. The use of beta-blockers for HRV associated with CAN should be used with caution due to the added risk of hypoglycemia.[2,12] Patients taking beta-blockers should be instructed to monitor glucose levels more

frequently or to consider the use of a CGM system for better ongoing monitoring of glucose levels.[2]

Unhealthy lifestyles and risk factors of CVD pose a problem in the management of patients with chronic illness. Medication is usually initiated past a trial of lifestyle modifications, including diet and exercise. The provider may consider prescribing midodrine and/or fludrocortisone for symptomatic orthostatic hypotension.[3,9] In addition, salt tablets are recommended for patients without heart failure but with a history of frequent falls.[3,9]



***Glycemic control provides a protective mechanism in all patients with diabetes, especially those with T1DM.***

Treatment of orthostatic hypotension associated with CAN should include nonpharmacologic interventions including increasing fluid intake, teaching the patient to avoid sudden changes in position, reducing movement that increases intra-abdominal or intrathoracic pressure such as the Valsalva maneuver, and/or bending to assist in lifting objects.[3,9,11,13] In addition, NPs should review patients' medication lists to determine if any should be discontinued because of hypotension or potential adverse reactions. This list should include tricyclic antidepressants, diuretics, and alpha-adrenoreceptor antagonists.[3] Also, the NP must continually assess fluid status for volume depletion, which can significantly increase the risk of orthostatic hypotension.[11] This includes strategies for measuring fluid intake and output, especially for those with recent or repeated falls. Interprofessional practice should include utilization of a licensed pharmacist.

**Nonpharmacologic therapy.** CVD risk factor management should include providing patients with information focused on smoking cessation, weight reduction, management of hypertension, hyperlipidemia, and hyperglycemia, and the use of antiplatelet agents such as a daily low-dose aspirin. Unfortunately, CAN is an independent risk factor for CVD that increases risk more than traditional cardiovascular markers such as age, waist-hip ratio, pulse pressure, and non-high-density lipoprotein cholesterol measures.[3] Across multiple studies on CAN, glycemic control provides a protective mechanism in all patients with diabetes, but especially those with T1DM.[17,18] Moderate-to-intense aerobic activity has been shown to lower mortality in patients with both T1DM and T2DM.[23] Establishment of a supervised physical activity program has been shown to slow the progression of CAN in both early and advanced stages of this disease.[23] The ADA recommends a cardiac stress test prior to initiation of a moderate-to-intense aerobic physical activity program.[12]

In addition to aerobic activity, nonpharmacologic treatment for CAN may also improve strength and balance while other aerobic activities cause the parasympathetic nervous system to reduce HRV.[11] For example, a small controlled crossover study conducted by Motooka and colleagues examined the effect of companionship with a dog on HRV in a population of older adults.[24] This study indicated companionship with an animal significantly improved HRV in this sample.[24] An additional study examined the effects of Tai chi, which improved glycemic control, lipid levels, balance, and neuropathic symptoms in adults with T2DM.[25] However, additional studies are needed to examine the effects of alternative therapies on HRV outcomes. This demonstrates the need for providers to incorporate a multifaceted approach to the treatment of CAN.

## Conclusion

CAN is a serious complication of diabetes and is associated with an increase in mortality. Maintaining glucose control is essential in preventing CAN. Steady control of glucose levels can prevent or delay associated problems, including neuropathies such as CAN. NPs are in a pivotal position to monitor for changes in health status and to educate patients on the importance of lifestyle changes and appropriate medication usage to prevent complications from diabetes. Use of an interdisciplinary treatment approach will improve a patient's quality of life and reduce complications that occur with CAN. NP

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