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Impact of diabetes mellitus on vestibular function: A scoping review

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Impact of Diabetes Mellitus on Vestibular Function:

A Scoping Review

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List of Abbreviations

- AAR: Amplitude Asymmetry Ratio
- ABR: Auditory Brainstem Response
- ADA: American Diabetes Association
- BPPV: Benign Paroxysmal Positional Vertigo
- BW: Bilateral Weakness
- CN VIII: Cranial Nerve Eight (Vestibulocochlear Nerve)
- cVEMP: Cervical Vestibular-Evoked Myogenic Potentials
- DM: Diabetes Mellitus
- DP: Directional Preponderance
- DPN: Diabetic Peripheral Neuropathy
- EN: Early Nephropathy
- EMG: Electromyography
- ENG: Electronystagmography
- HbA1c: Hemoglobin A1c
- LARP: Left Anterior, Right Posterior SCCs
- MeSH: Medical Subject Headings
- N1: Negative Valley
- OKN: Optokinetic Nystagmus
- oVEMP: Ocular Vestibular-Evoked Myogenic Potentials
- P1: Positive Peak
- PNP: Polyneuropathy
- PRISMA-ScR: Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews
- RALP: Right Anterior, Left Posterior SCCs
- SCC: Semicircular Canal
- SCM: Sternocleidomastoid
- SP: Smooth Pursuit
- SPV: Slow Phase Velocity
- SVV: Subjective Visual Vertigo
- T1DM: Type 1 Diabetes Mellitus
- T2DM: Type 2 Diabetes Mellitus
- TCR: Total Caloric Response
- UW: Unilateral Weakness
- vHIT: Video Head Impulse Testing
- VNG: Videonystagmography
- VOR: Vestibulo-Ocular Reflex

Abstract

Diabetes mellitus (DM) encompasses a group of metabolic diseases that result in high blood sugar (i.e., hyperglycemia). By 2030, it is anticipated that 578 million adults worldwide will have DM, with this number growing at a faster rate in developed areas of the world.[27] If left uncontrolled, DM can cause considerable damage to several areas of the body, including the heart, kidneys, nerves, and ears. When focusing exclusively on the ears, there has been markedly less research on the vestibular system when compared to the auditory system, even though DM is a known risk factor for falling. The purpose of this study was to understand the current state of knowledge regarding DM and vestibular function and to identify gaps in knowledge that need to be explored. A scoping review of the literature was performed and reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) standards.[51] Search terms included medical subject headings (MeSH terms) and keywords related to DM and vestibular function. In total, 326 papers were retrieved and 43 articles met inclusion/exclusion criteria for extensive review. Findings show that studies performed on the vestibular system tend to have smaller sample sizes, inconsistent test batteries, and variable results. There is some evidence to suggest Benign Paroxysmal Positional Vertigo (BPPV) may be more prevalent in individuals with DM, but the exact percentage of those impacted is unknown. The duration and severity of DM was also found to have a significant impact on vestibular test results. As DM becomes more prevalent in our society, it is essential a standardized test battery be developed to

more efficiently evaluate and diagnose vestibular disorders in this population. Findings from this study may help develop a narrower research question which could be used to conduct a systematic review. Findings from this study may also assist in the development of a randomized control trial (RCT) involving individuals with DM.

Introduction

Diabetes mellitus (DM) encompasses a group of metabolic diseases that result in high blood sugar (i.e., hyperglycemia). Hyperglycemia occurs when excess glucose (i.e., sugar) builds up in the bloodstream. This excess sugar is the result of the pancreas not forming enough insulin to circulate throughout the blood. Insulin is a hormone which allows for glucose to enter the body's cells to provide sufficient energy for the brain, muscles, and tissues to function appropriately. The primary ways glucose enters the bloodstream is through food and the liver. When sugar levels fall below normal, the liver begins breaking down its stored glycogen to create glucose for the body to derive energy from.[33]

Types of DM:

The two major types of DM are type 1 and type 2. Type 1 DM, prevalent in 5-10% of individuals with DM, occurs when the body's immune system attacks its own insulin-producing beta cells, causing too much sugar to build up in the bloodstream. Risk factors for developing T1DM include being closely related to someone with the disease and being young in age.[5] Type 2 DM, prevalent in 90-95% of individuals with DM, occurs when the body's cells become insulin-resistant, resulting in excessive sugar to accumulate in the blood. As the sugar accumulates, the pancreas is incapable of producing enough insulin to overcome the body's resistance to the hormone. Risk factors for developing T2DM include being overweight, closely related to someone with the

disease, inactive, 45 years of age or older, part of a racial or ethnic minority group, and having a previous history of pre-diabetes or gestational diabetes.[5] An individual is classified as having pre-diabetes when their blood sugar is above the normal range but not high enough to be diagnosed with T2DM. Gestational diabetes arises during pregnancy when the hormones in a woman's body are highly variable. Hormone variability makes it difficult for the body to process sugar and convert it into energy for the cells. [33]

Global Impact of DM

More than 9.3% of the global population between the ages of 20 and 79 are living with DM, with that number only expected to grow in the coming decades. By 2045, it is anticipated that seven hundred million (10.9%) people will be living with DM.[47] To add to the issue, the prevalence of DM increases significantly with age, and over half of individuals with DM are unaware they even have the disease. Further, the diagnosis of DM occurs disproportionately more in developed areas of the world, with China, India, and the United States of America having the highest known prevalence rates.[47] The growing prevalence of DM worldwide is a public health crisis due to the detrimental effects the disease can have on the entire body. Common complications of DM include retinopathy, neuropathy, kidney damage/failure, heart and blood vessel disease (i.e., microangiopathy), weakened immune system response, bacterial and fungal infections, depression, and an increased risk of developing dementia.[33] Another common, but

significantly less discussed, complication of DM includes inner ear impairments, which will be the primary focus of this paper.

Effects of DM on the Auditory System:

Glucose is the main source of energy for the cochlea and helps to maintain the endocochlear potential.[13] Juhn and Youngs (1972) found that glucose levels in the cochlear fluids are similar to those found in blood. When hyperglycemia occurs, it thickens the capillaries in the stria vascularis and has the ability to negatively impact the cochlear transduction process responsible for the endocochlear potential. When there is a disruption in the cochlea's ability to derive oxygen and glucose from the stria vascularis for the purpose of cochlear transduction, peripheral hearing loss may occur.[13] Other possible impacts of hyperglycemia on the auditory system include demyelination of the vestibulocochlear nerve (CN VIII), poor peripheral nerve conduction resulting from damage to CN VIII, degeneration of inner and outer cells located at the basal turn of the cochlea, narrowing of the internal auditory artery, thickening of the basilar membrane, and damage to cell types in vascular regions of the stria vascularis, spiral ligament, and spiral ganglia which may be more vulnerable to the negative impacts of high blood sugar.[13]

Although DM is a known risk factor for auditory complications, the American Diabetes Association (ADA) has yet to add routine audiometric evaluations as a recommendation for individuals living with disease. Individuals with DM have an increased likelihood of developing mild sensorineural hearing loss when compared to non-diabetic controls. In

addition, patients with T2DM have a greater incidence of high frequency (6000-8000 Hz) hearing loss components, with factors such as increased age, poor glycemic control, and longer disease duration leading to poorer hearing outcomes.[17] Lastly, there is research to suggest that hyperglycemia can lead to retrocochlear or central auditory pathologies, illustrated through Auditory Brainstem Response (ABR) testing. In a study by Vaughan et al. (2007) involving veterans diagnosed with DM, results showed a significant delay in the absolute latencies for Waves I, III, and V of the ABR in the right ear. In addition, all interpeak latencies between Wave I and Wave V (i.e., I-III, I-V, III-V) in both ears were significantly prolonged when compared to non-diabetic controls. After adjusting for increased age, hearing loss, and other diabetic health factors, a significant difference was still observed for the majority of absolute and interpeak latencies measured in diabetic versus non-diabetic controls. This evidence suggests that individuals with DM may be at an increased risk for developing retrocochlear pathologies, such as auditory neuropathy and/or auditory processing disorders.

Effects of DM on the Vestibular System:

While there has been greater research in recent decades regarding the negative impacts of hyperglycemia on the auditory system, there is still little known about the effects of DM on the vestibular system. Due to the vestibular system having similar connective tissues to those observed in the cochlea, it is hypothesized that the biological mechanisms shown to negatively impact auditory function will also have detrimental effects on the vestibular system.[13] Of the research available, studies focus primarily on the otolith end organs,

responsible for perceptions of linear acceleration and static tilt, with limited information derived from animal models. Gioacchini et al. (2018) outlines how metabolic stress from hyperglycemia has been shown to result in demyelination of the vestibulocochlear nerve and Type I hair cell loss in the saccule. In addition, hyperglycemia causes an overproduction in the extracellular matrix which is responsible for the structural support of the body's cells and tissues. In humans, an overproduction of the extracellular matrix may translate to impairment of the connective tissues needed for the health of otolith end organs. Lastly, hyperglycemia may lead to degeneration of the maculae in both the utricle and saccule. Degeneration of the maculae can cause otoconia, or the calcium carbonate crystals that assist with the detection of linear acceleration, to detach and fall into the semicircular canals (SCCs). Otoconia detachment may result in a well-known vestibular condition called Benign Paroxysmal Positional Vertigo (BPPV).[16]

A brief glance at the clinical studies which have assessed the impact of DM on vestibular function show inconsistent test batteries. While some studies assessed the SCCs with caloric measurements[8], others assessed with Rotary Chair[22] or Video Head Impulse Testing (vHIT).[19] While a few studies evaluated the otolith end organs (i.e., utricle and saccule) with both cervical vestibular-evoked myogenic potentials (cVEMP) and ocular vestibular-evoked myogenic potentials (oVEMP),[2, 11] the majority evaluated with the cVEMP only.[20, 25, 26] Further, very few studies appear to have evaluated vestibular function with multiple vestibular measures, preventing the reader from obtaining a full understanding of the detrimental impact DM may be having on the entire system.

When comparing studies which have assessed the impact of DM on vestibular function, test results vary significantly. While some studies found greater dysfunction of the SCCs than the otoliths,[54] others found no abnormality in the SCCs.[22] Similarly, while some studies found prolonged cVEMP latency[30] or reduced cVEMP amplitude,[24] others measured normal cVEMP latencies and amplitudes.[3] An important consideration when evaluating the results of these studies is to consider whether or not severity and duration of DM were accounted for in the statistical analyses. A study by Konukseven et al. (2014) found significantly longer cVEMP and oVEMP latencies in the T2DM group when compared to the pre-diabetic and control groups. In addition, this study found that higher hemoglobin A1c (HbA1c) levels and longer durations of DM were associated with longer cVEMP and oVEMP latency values. These results illustrate how the inclusion of duration and severity of disease in the statistical analysis of vestibular measures may give greater insight into how the disease is functionally impacting the entire system. When duration and severity are not included in statistical analyses, significant vestibular findings may be overlooked.

DM and Benign Paroxysmal Positional Vertigo (BPPV):

One of the only consistent findings found in research on individuals with DM is an increased incidence of BPPV. D'Silva et al (2017) performed a retrospective chart review of vestibular patients and found that 46% of those with both a vestibular and DM diagnosis also had a BPPV diagnosis. In contrast, only 37% of vestibular patients without DM had BPPV. A study by Webster et al. (2015) also found that hyperglycemia increases

the risk for BPPV recurrence, and that those with normal glucose-insulin tests were less likely to have multiple episodes of BPPV. As mentioned earlier, this may be evidence to suggest that well-controlled DM can lessen the negative impacts of the disease on the vestibular system.

DM and Risk of Falls:

DM is an independent risk factor for falling. It is suspected that more than 70% of individuals with DM have a balance difficulty, with the issue being made worse by increased duration and severity of the disease.[41] This increased incidence of balance difficulties in individuals diagnosed with DM occurs independently from the presence of peripheral neuropathy and retinopathy, suggesting the vestibular system is playing a key role.[13] As DM becomes more prevalent in our society, it is essential that a standardized test battery be developed to more efficiently evaluate and diagnose vestibular disorders in this population. The purpose of this study was to conduct a scoping review to examine how research has previously been conducted, identify key vestibular characteristics of individuals diagnosed with DM, and identify gaps in knowledge that need to be explored.

Methods

This scoping review was reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) standards.[51]

Search Strategy

In April of 2021, the online databases of PubMed (MEDLINE) and ProQuest- Dissertation and Theses Global were used to identify and compile relevant papers. Search terms included medical subject headings (MeSH terms) and keywords related to DM and vestibular function. The search strategy was discussed with an experienced librarian from James Madison University, Ms. Lara Sapp, who offered further suggestions and revisions. The final search strategy used for the PubMed (MEDLINE) database can be referenced in **Appendix I**, while the search strategy for the ProQuest- Dissertation and Theses Global database can be referenced in **Appendix II**. Search results were exported into Mendeley©, a free reference management software, and all duplicates were removed. Two reviewers worked together to evaluate the titles, abstracts, and, eventually, full-text publications to determine if they met study inclusion criteria. Disagreements on study selection were resolved through discussion with a third reviewer, when necessary. Following the completion of study selection in Mendeley©, a citation review and reference review were performed on all included articles in an attempt to identify further relevant publications.

Study Selection

To be included in this scoping review, studies were required to have individuals with DM (type 1 or 2) as the primary population of interest, have an abstract available for review, be published in the English language, and include direct measures of vestibular function (i.e., VNG/ENG, cVEMP, oVEMP, vHIT, Rotary Chair, etc.).

Papers were excluded if individuals with DM were not the primary population of interest and if indirect measures of vestibular function (i.e., questionnaires, posturography, bedside testing, etc.) were solely used. Studies were also excluded if they lacked an abstract, were review in nature, or were not published in the English language. There were no age or date restrictions applied to this review; papers published during any time frame containing data from children and/or adults with DM could have been included if other selection criteria were met. **Figure I.** describes the search strategy used for this scoping review. In total, 326 articles were recovered during the database searches, reference reviews, and citation reviews, with forty-three studies meeting the inclusion criteria for this scoping review.

Data Extraction

A data-charting Excel© spreadsheet was developed by both reviewers to determine the relevant variables necessary for data extraction. Each reviewer independently extracted and charted the data, discussed the results, and updated the data-charting spreadsheet in a repeated fashion. All included publications had data extracted in relation to study

characteristics (i.e., location, setting, design, sample size), participant factors (i.e., sex, age, DM type & severity, comorbidities), auditory and vestibular tests performed, and significant vestibular test findings.

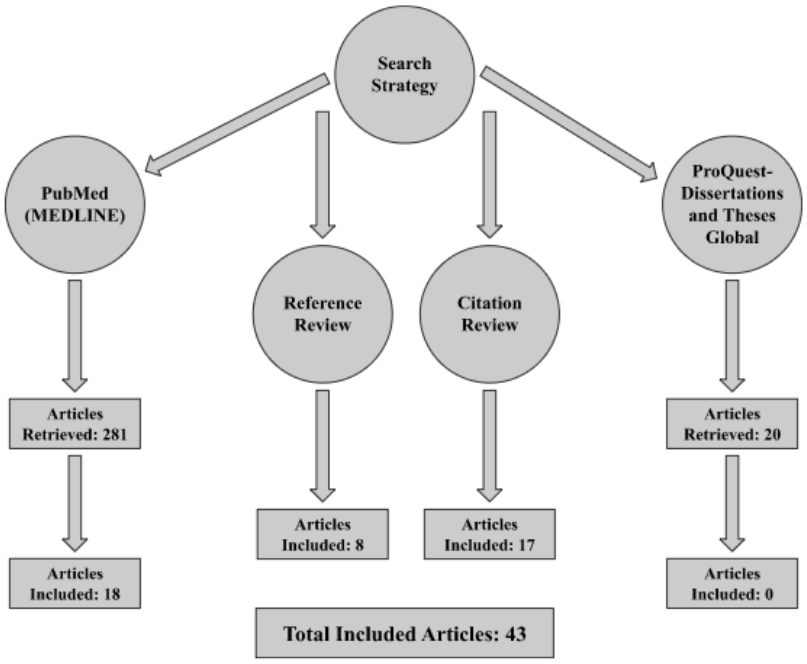

Figure I. Search Strategy

Results

Overview:

After an extensive evaluation of the 326 articles recovered during database searches, reference reviews, and citation reviews, forty-three studies met the inclusion criteria for this scoping review. **Table I** outlines the range of years in which the included articles were published, with the majority being produced in the last ten years.

Table I. Article Year Distribution

Year	Articles: n (%)
1980-1989	1 (2.3)
1990-1999	4 (9.3)
2000-2009	8 (18.6)
2010-2015	11 (25.6)
2016-Present	19 (44.2)

To be included in this scoping review, a study's primary population of interest had to be individuals with DM. Twelve (27.9%) studies evaluated those with T1DM, nineteen (44.2%) with T2DM, and twelve (27.9%) with both T1DM and T2DM. Further, thirty-four (79.1%) studies evaluated adults aged eighteen years or older, one (2.3%) evaluated children aged seventeen years or younger, and eight (18.6%) evaluated both children and adults. The average DM participant age amongst all studies was 45.9 years. The average DM sample size was 50.7, with the largest study having 104 DM subjects,[40] and the smallest having 5 DM subjects.[10]

The vestibular tests performed on study participants can be viewed in **Table II.** Over half (53.5%) of included studies used caloric testing to assess the horizontal SCCs and

superior vestibular nerve. Fewer studies assessed the SCCs with Rotary Chair (4.7%) and vHIT (16.3%). When looking at the assessment of otolith organs, 34.9% of studies assessed the saccule and inferior vestibular nerve with cervical vestibular-evoked myogenic potentials (cVEMP), while 20.9% of studies assessed the utricle and superior vestibular nerve with ocular vestibular-evoked myogenic potentials (oVEMP). Lastly, eighteen (41.9%) studies used other indirect vestibular measures, such as postural stability, bedside testing, and subjective visual vertigo (SVV) to assess vestibular function in individuals with DM.

Twenty-eight (65.1%) studies used one direct measure (VNG/ENG, Rotary Chair, vHIT, cVEMP, oVEMP) to assess vestibular function in individuals with DM, seven (16.3%) used two direct measures, seven (16.3%) used three direct measures, and one (2.3%) used four direct measures.

Table II. Vestibular Test Distribution

Vestibular Test:	Articles: n (%)
VNG/ENG: Ocular Motility	18 (41.9)
VNG/ENG: Positional/Positioning	13 (30.2)
VNG/ENG: Caloric	23 (53.5)
Rotary Chair	2 (4.7)
vHIT	7 (16.3)
cVEMP	15 (34.9)
oVEMP	9 (20.9)
Other: Indirect Vestibular Measures	18 (41.9)

VNG/ENG: Videonystagmography/Electronystagmography
vHIT: Video Head Impulse Testing
cVEMP: Cervical Vestibular-Evoked Myogenic Potentials
oVEMP: Ocular Vestibular-Evoked Myogenic Potentials

VNG/ENG: Ocular Motility

Eighteen studies performed VNG/ENG ocular motility testing on individuals with DM. The mean age of participants was 39.2 in the DM group(s) and 41.9 in the control group. Eight studies evaluated those with T1DM, four with T2DM, and six with either T1DM or T2DM. The average DM sample size was 48.3, with the largest study having 104 DM subjects,[40] and the smallest having 5 DM subjects.[10] Ocular motility measures do not provide a direct assessment of vestibular function, so they were not a primary focus of this scoping review. A table outlining the ocular motility findings from the studies included in this scoping review can be found in **Appendix III.**

VNG/ENG: Positional Testing

Table III. outlines the eight studies that used positional testing (i.e., Dix-Hallpike) and/or questionnaires to determine the presence of posterior SCC Benign Paroxysmal Positional Vertigo (BPPV) in individuals with DM. Five studies used descriptive analyses to outline results, while three studies used descriptive and statistical analyses. The mean age of participants was 45.9 in the DM group(s) and 41.4 in the control group. One study evaluated those with T1DM, five with T2DM, and two with either T1DM or T2DM. The average DM sample size was 56, with the largest study having 104 DM subjects,[40] and the smallest having 25 DM subjects.[12]

Six studies identified the presence of BPPV in individuals with DM,[11, 20, 26, 28, 37, 40] while two studies did not.[12, 24] Ozel et al. (2013) and Ibraheem et al. (2017) found that 7.7% and 8.9% of DM subjects had BPPV, respectively. Kanumuri et al. (2017) and Naik & Tilloo (2018) found that 20% and 22% of T2DM subjects had BPPV, respectively. Lastly, D'Silva et al. (2017) reported the highest prevalence of BPPV, with 46% of T2DM subjects having the condition.

The impact of duration and/or severity of DM on positional VNG/ENG testing was investigated by one study. Ozel et al. (2013) did not find a higher prevalence of BPPV in individuals who had been diagnosed with DM for more than seven years.

Table III. BPPV Findings						
Article	**Study Design**	**Sample Size:**	**Mean Age**	**Diabetes Type**	**Defining Abnormality**	**Results**
D'Silva et al. (2017)	Cross Sectional	T2DM: 19 BPPV: 18 T2DM and BPPV: 14 Control: 20	T2DM: 58.6 ± 5.3 BPPV: 54.9 ± 5.9 T2DM and BPPV: 58.5 ± 5.6 Control: 57.5 ± 5.3	T2DM and T2DM w/ BPPV	Descriptive (%)	**BPPV:** 46% of T2DM subjects
El Shafei et al. (2021)	Cross Sectional	T1DM: 25 Control: 25	T1DM: 10.4 +/- 2.7 Control: 10.11+/- 2.6	T1DM	Descriptive (%) and Statistical Finding (P< .05)	**BPPV:** Not detected in any subjects; No significant difference between T1DM subjects and controls (p>.05)
Ibraheem et al. (2017)	Cross Sectional	T1DM: 15 T2DM-Oral: 15 T2DM-Insulin: 15	T1DM: 33.87 ± 8.47 T2DM-Oral: 43.67 ± 5.33 T2DM-Insulin: 45.4 ± 3.87	T1DM, T2DM treated with oral hypoglycaemic, and T2DM treated with insulin	Descriptive (%)	**BPPV:** 8.9% of all DM subjects; 6.7% of T1DM subjects, 13.3% of T2DM-Oral subjects; 6.7% of T2DM-Insulin subjects
Kalkan et al. (2018)	Cross Sectional	T2DM: 33 T2DM w/ DPN: 33 Control: 35	T2DM: 53.8±7.3 T2DM w/ DPN: 53.8±8.7 Control: 49.6±8.4	T2DM and T2DM w/ Diabetic Peripheral Neuropathy (DPN)	Descriptive (%) and Statistical Finding (P< .05)	**BPPV:** Not detected in any subjects; No significant difference between DM and control groups (p>.05)
Kanumuri et al. (2018)	Cross Sectional	T2DM: 40 Control: 20	T2DM: 20-60 Control: 20-60	T2DM	Descriptive (%)	**BPPV:** 20% of T2DM subjects
Kim et al. (2012)*	Retrospective	T1DM: 10 T2DM: 25	T1DM: 51.1 +/- 15.5	T1DM w/ DPN and T2DM w/	Descriptive (%)	**BPPV Testing Performed:** 57.9% of subjects with DPN (T1DM & T2DM) were diagnosed with vestibular

Table III. BPPV Findings

Article	Study Design	Sample Size:	Mean Age	Diabetes Type	Defining Abnormality	Results
			T2DM: 51.1 +/- 15.5	DPN		dysfunction.
Naik & Tilloo (2018)	Cross Sectional	T2DM: 100	T2DM: 30-60	T2DM	Descriptive (%)	**BPPV:** 22% of T2DM subjects
Ozel et al. (2013)	Cross Sectional	T2DM: 104 Control: 104	T2DM: 50.3 Control: 48.3	T2DM	Descriptive (%) and Statistical Finding (P< .05)	**BPPV:** Higher prevalence of BPPV in T2DM subjects when compared to controls (p=.006); BPPV present in 7.7% of T2DM subjects and 5.8% of control subjects

*No quantitative data provided by study
BPPV: Benign Paroxysmal Positional Vertigo
DM: Diabetes Mellitus
DPN: Diabetic Peripheral Neuropathy
T1DM: Type 1 Diabetes Mellitus
T2DM: Type 2 Diabetes Mellitus

VNG/ENG: Caloric

Table IV. outlines the twenty-three studies that performed caloric testing on individuals with DM to assess the function of the horizontal SCCs and superior vestibular nerve. Nine studies used statistical analyses to outline results, twelve used descriptive analyses, and one used descriptive and statistical analyses. One study did not specify how results were analyzed.[7] The mean age of participants was 42.5 in the DM group(s) and 37.5 in the control group. Seven studies evaluated those with T1DM, eight with T2DM, and eight with either T1DM or T2DM. The average DM sample size was 60.3, with the largest study having 188 DM subjects,[46] and the smallest having 12 DM subjects.[48]

The methods used to conduct the caloric test were inconsistent across studies. Eleven of the studies outlined their chosen caloric methods. Specifically, five studies used bithermal water calorics, four used bithermal air calorics, one used monothermal cool water calorics, and one used trithermal ($10^{\circ}C$, $20^{\circ}C$, and $42^{\circ}C$) air calorics. Twelve of the twenty-three studies did not specify whether they used water or air to stimulate a caloric response. In addition to differences in caloric methods, there was variability across studies in the caloric parameters used to assess vestibular function. Eleven studies assessed unilateral weakness (UW), three assessed bilateral weakness (BW), five assessed directional preponderance (DP), one assessed total caloric response (TCR), and two assessed mean slow phase velocity (SPV). Of these, some described their findings descriptively (e.g., the percentage of patients whose results were outside the normal range as defined in that study), while others did statistical comparisons between a control group

and a group with DM. Twelve studies did not provide specific quantitative data to illustrate further assessment of caloric test results (i.e., UW, BW, DP, etc.).

El Shafei et al. (2021) found significantly more T1DM subjects had unilateral weakness when compared to controls. Ibraheem et al. (2017) found T2DM subjects treated with insulin had a larger UW than T2DM subjects treated with oral hypoglycaemic. One study did not find a statistically significant difference in UW between T2DM and control subjects.[36] When looking at descriptive UW test results, two studies found more than 20% of DM subjects had UW,[1, 45] while four studies found less than 10% of DM subjects had UW.[4, 14, 15, 29]

Deshpande et al. (2015) found a significantly worse bilateral weakness in T2DM subjects when compared to controls. While one study found 21.8% of T1DM subjects had BW,[4] another found 33.3% of T1DM subjects had BW.[29] For directional preponderance, three studies found 16% or less DM subjects had DP,[14, 15, 45] while two studies found more than 30% of DM subjects had DP.[4, 9] Total caloric response was evaluated by one study which found T2DM subjects treated with oral hypoglycaemic had a significantly better TCR when compared to T1DM subjects and T2DM subjects treated with insulin.[20] When evaluating the two studies that measured mean slow phase velocity, one did not find a significant difference between T2DM and control subjects,[36] while the other found a significant difference between T1DM and control subjects at 44°C and 30°C in the right ear and 30°C in the left ear.[45]

The impact of duration and/or severity of DM on caloric VNG/ENG results was investigated by sixteen studies. Eleven studies found a positive correlation between increased DM duration and a greater impairment of caloric test results,[1, 4, 9, 14, 20, 31, 32, 42, 43, 46, 50] while three did not.[8, 15, 18] Further, eight studies found a positive correlation between increased DM severity and a greater impairment of caloric test results,[9, 14, 20, 32, 37, 43, 46, 50] while five did not.[4, 15, 18, 31, 49]

Table IV. Caloric Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
Aantaa & Lehtonen (1981)	Cross Sectional	Insulin-Treated Diabetes: 24	Insulin-Treated Diabetes: 34	Insulin-Treated T1DM and T2DM	Unspecified	Descriptive (%)	**UW:** 20.8% of T1DM & T2DM subjects
Biurrun et al. (1991)	Cross Sectional	T1DM: 46 Control: 33	T1DM: 25.9 +/- 8.9 Control: 26.2 +/- 9.4	T1DM	Bithermal Water: 30°C and 44°C	Descriptive (%)	**UW:** 4.3% of T1DM subjects **BW:** 21.8% of T1DM subjects **DP:** 54% of T1DM subjects
Chamyal (1997)*	Cross Sectional	T1DM: 10 T2DM: 20 Control: 30	T1DM: 17-49 T2DM: 17-49 Control: 20-48	T1DM and T2DM	Bithermal Unspecified	Unspecified	**Caloric Performed:** No evidence of vestibular dysfunction detected in any T1DM or T2DM subjects
Deshpande et al. (2017)	Cross Sectional	T2DM: 35 Control: 25	T2DM: 70.6 +/- 4.7 Control: 74.6 +/- 5.4	T2DM w/o DPN	Bithermal Water	Statistical Finding (p<.05)	**BW:** T2DM subjects had a significantly worse bilateral caloric weakness than controls (p=.041)
Dorkar (2015)	Cross Sectional	Uncomplicated DM: 30 DN: 30	Uncomplicated DM: 25-55 DN: Unspecified	Uncomplicated DM and Diabetic Nephropathy	Unspecified	Descriptive (%)	**UW:** 13.3% of uncomplicated DM subjects; 26.7% of DN subjects **DP:** 33.3% of uncomplicated DM subjects; 36.7% of DN subjects
El Shafei et al. (2021)	Cross Sectional	T1DM: 25 Control: 25	T1DM: 10.4 +/- 2.7 Control: 10.11+/- 2.6	T1DM	Bithermal Air: 25°C and 49°C	Statistical Finding (P< .05)	**UW:** Significantly more T1DM subjects had UW when compared to controls (p<.05).
Gawron et al. (2002)	Cross Sectional	T1DM: 95 Control: 44	T1DM: 15.5 +/- 5.1 Control: 16.3 +/- 6.1	T1DM	Bithermal Water: 30°C and 44°C	Descriptive (%)	**UW:** 4.2% of T1DM subjects and 0% of controls **DP:** 7.4% of T1DM subjects and 4.54% of controls

Table IV. Caloric Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
Gawron et al. (2011)	Cross Sectional	T1DM: 59 Control: 33	T1DM: 20 Control: 19.2	T1DM	Bithermal Water: 30°C and 44°C	Descriptive (%)	**UW:** 5% of T1DM subjects and 0% of controls **DP:** 11.6% of T1DM subjects 0% of controls
Herrera-Rangel et al. (2015)*	Cross Sectional	T2DM: 99	T2DM: 52 +/-7	T2DM	Bithermal Unspecified	Descriptive (%)	**Asymmetry or No Response:** 7% of T2DM subjects
Ibraheem et al. (2017)	Cross Sectional	T1DM: 15 T2DM-Oral: 15 T2DM-Insulin: 15	T1DM: 33.87 ± 8.47 T2DM-Oral: 43.67 ± 5.33 T2DM-Insulin: 45.4 ± 3.87	T1DM, T2DM treated w/ oral hypoglycaemic, and T2DM treated w/ insulin	Bithermal Unspecified	Statistical Finding (p<.05)	**UW:** T2DM subjects treated with oral hypoglycaemic had a significantly smaller UW when compared to T2DM subjects treated with insulin (P=.027) **TCR:** T2DM subjects treated with oral hypoglycaemic had a significantly better TCR when compared to T2DM subjects treated with insulin and T1DM subjects (p=.091)
Kim et al. (2012)*	Retrospective	T1DM: 10 T2DM: 25	T1DM: 51.1 +/- 15.5 T2DM: 51.1 +/- 15.5	T1DM w/ DPN and T2DM w/ DPN	Unspecified	Descriptive (%)	**Caloric Performed:** 57.9% of subjects with DPN (T1DM & T2DM) were diagnosed with vestibular dysfunction
Klagenberg et al. (2007)	Cross Sectional	T1DM: 30	T1DM: 25.7	T1DM	Trithermal Air: 10°C, 20°C, and 42°C	Descriptive (%)	**UW:** 6.7% of T1DM subjects **BW:** 33.3% of T1DM subjects **Abnormal Caloric Result:** 60% of T1DM subjects
Kuniyil et al. (2020)*	Cross Sectional	DM: 97	DM: 54.68 +/- 10.68	Unspecified DM type	Bithermal Unspecified: 20°C to 47°C	Statistical Finding (p<.05)	**UW:** More common in subjects with DM > 5 years (p<.001) **Normal caloric results:** More common in subjects with DM </= 5 years (p<.001).
Li et al. (2019)*	Cross Sectional	T2DM: 51 Control: 43	T2DM: 56.1 +/- 10.1 Control: 54.4 +/-7.2	T2DM	Bithermal Air: 23°C and 49°C	Statistical Finding (P< .05)	**Abnormal Caloric Results:** T2DM subjects had statistically more abnormal caloric results than control subjects (p<.05).
Morgan et al. (2015)	Cross Sectional	T2DM: 28 Control: 28	T2DM: 51.64 +/- 6.72	T2DM	Monothermal Water: 30°C	Statistical Finding (p<.05)	**UW:** No significant difference between T2DM and control subjects (p>.05)

Table IV. Caloric Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
			Control: 44.04 +/- 13.92				**Mean SPV:** No significant difference between T2DM and control subjects (p>.05)
Naik & Tilloo (2018)*	Cross Sectional	T2DM: 100	T2DM: 30-60	T2DM	Unspecified	Descriptive (%)	**Caloric Performed:** 70% of T2DM subjects were diagnosed with vestibular dysfunction.
Prakash & Sumathi (2013)*	Cross Sectional	T2DM: 100 Control: 100	T2DM: < 40 Control: <40	T2DM	Bithermal Unspecified	Descriptive (%)	**Caloric Performed:** 42% of T2DM subjects were diagnosed with vestibular dysfunction. 12% of control subjects were diagnosed with vestibular dysfunction.
Ren et al. (2018)*	Cross Sectional	T2DM: 30 T2DM w/ EN: 30 Control: 30	T2DM: 56.40 +/- 8.46 T2DM w/ EN: 58.07 +/- 7.65 Control: 55.33 +/- 6.21	T2DM and T2DM w/ EN	Bithermal Air: 24°C and 50°C	Statistical Finding (p<.05)	**Caloric Performed:** No statistically significant difference between percentage of control, T2DM, and T2DM w/ EN subjects diagnosed with vestibular dysfunction (p>.05).
Rigon et al. (2007)	Cross Sectional	T1DM: 19 Control: 19	T1DM: 8-25 Control: 8-25	T1DM	Bithermal Water: 30°C and 44°C	Descriptive (%) and Statistical Finding (P< .05)	**UW:** 21.05% of T1DM subjects; 0% of control subjects. **DP:** 15.79% of T1DM subjects; 0% of control subjects. **Mean SPV:** Significant difference between T1DM and control subjects for mean SPV at 44°C RE, 30°C RE and 30°C LE (p<.05)
Roy et al. (2019)*	Cross Sectional	T1DM and T2DM: 188	T1DM and T2DM: 51.59 ± 0.76	T1DM and T2DM	Bithermal Unspecified: 30°C and 44°C	Statistical Finding (p<.05)	**Caloric Performed:** Statistically significant association between abnormal vestibular results and duration of diabetes (p=.001).
Scherer & Lobo (2002)*	Cross Sectional	T1DM: 12	T1DM: </= 40	T1DM	Bithermal Air: 20°C and 42°C	Descriptive (%)	**Abnormal Caloric Results:** 66.7% of T1DM subjects.
Sharma et al. (1999)*	Cross Sectional	DM: 25 DM w/ Complications:	DM: 20-50 DM w/ Complications:	Unspecified DM type and unspecified	Unspecified	Statistical Finding (p<.05)	**Caloric Performed:** No evidence of vestibular dysfunction found in DM subjects with or without complications (p>.05).

Table IV. Caloric Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
		25 Control: 25	20-50 Control: 20-50	DM type with complications			
Sumathi et al. (2016)*	Cross Sectional	T2DM: 100	T2DM: 25-40	T2DM	Bithermal Unspecified	Descriptive (%)	**Caloric Performed:** Vestibulopathy (central pathology) was found in 42% of T2DM subjects.

*No quantitative data provided by study
BW: Bilateral Weakness
DM: Diabetes Mellitus
DP: Directional Preponderance
DPN: Diabetic Peripheral Neuropathy
EN: Early Nephropathy
SPV: Slow Phase Velocity
T1DM: Type 1 Diabetes Mellitus
T2DM: Type 2 Diabetes Mellitus
TCR: Total Caloric Response

Rotary Chair

Of the forty-three articles included in this scoping review, only two utilized the Rotary Chair to assess horizontal SCC function. **Table V.** outlines the findings of these studies. Jauregui-Renaud et al. (2017) used a case-control study to compare 101 individuals with T2DM to 51 healthy controls. The average age of T2DM subjects was 60.3, and the average age of the healthy controls was 56.5. Statistical analyses were used to determine there were no significant differences between T2DM and control subjects for vestibulo-ocular reflex (VOR) gain at 0.16 and 1.28 Hz ($p>.05$). In addition, VOR gain did not vary significantly for subjects with a history of falls when compared to subjects without a history of falls ($p>.01$). A second study by Klagenberg et al. (2007) was descriptive in nature. Thirty T1DM subjects with an average age of 25.7 were all shown to have pre- and post-rotary nystagmus within normal limits.

Table V. Rotary Findings

Article	Study Design	Sample Size	Mean Age	Diabetes Type	Method	Defining Abnormality	Results
Jauregui-Renaud et al. (2017)	Cross Sectional	T2DM: 101 Control: 51	T2DM: 60.3 +/- 9.8 Control: 56.5 +/- 6.8	T2DM	Sinusoidal rotation at 0.16 Hz and 1.28 Hz (60°/sec peak velocity)	Statistical Finding (p<.05)	No significant difference between T2DM and control subjects for gain to sinusoidal rotation at 0.16 Hz and 1.28 Hz (p>.05)
Klagenberg et al. (2007)	Cross Sectional	T1DM: 30	T1DM: 25.7	T1DM	Pendular swing rotatory test with stimulation of all SCCs	Descriptive (%)	Pre- and post-rotatory nystagmus were within normal limits for all T1DM subjects

SCCs: Semicircular Canals
T1DM: Type 1 Diabetes Mellitus
T2DM: Type 2 Diabetes Mellitus

Video Head Impulse Test (vHIT)

Table VI. outlines the seven studies included in this scoping review which used vHIT to assess the function of the SCCs in individuals with DM. All seven studies used a case-control style, with the average age of participants being 41.2 in the DM group(s) and 39.4 in the control group. Three studies evaluated those with T1DM, three with T2DM, and one study with either T1DM or T2DM. The average DM sample size was 30.3, with the largest study having 66 DM subjects,[24] and the smallest having 8 DM subjects.[39]

Looking specifically at vHIT gain, four of seven studies did not find a statistically significant difference between DM and control subjects.[24, 34, 35, 39] Heystek (2018) reported significantly lower left anterior SCC and right posterior SCC gain in people with DM. Ribeiro et al. (2020) also reported significantly lower left anterior SCC gain, as well as lower left posterior and right anterior SCC vHIT gain. Lastly, Ibraheem et al. (2021) reported a significantly lower right and left lateral SCC vHIT gain in subjects with DM. vHIT gain is considered to be within normal limits if it is .8 or better in the lateral canals and .7 or better in the vertical (anterior and posterior) canals. Despite there being significant differences between DM and control groups for mean vHIT gain in certain studies, mean vHIT gain was still within normal limits for all SCCs (right and left ears) in the seven studies which analyzed it.

Of five studies that investigated the presence of overt and covert saccades,[19, 24, 34, 39, 44] all but one did not find a statistically significant difference between DM and control subjects. Minnaar (2017) observed a significantly higher occurrence of overt and covert

saccades in the right lateral SCC of T2DM subjects. Ibraheem et al. (2021) was the only study to evaluate subjects for gain asymmetry, finding higher asymmetries in the lateral and LARP (left anterior, right posterior) canals of people with T1DM and T2DM. Ibraheem et al. (2021) was also the only study to account for diabetic severity in their test results, finding a statistically significant negative correlation between mean Hb-A1c levels and vHIT gain for the lateral SCCs in the right and left ears. A positive correlation between mean Hb-A1c levels and gain asymmetry was also found for the lateral and RALP canals in T1DM subjects and the lateral and LARP canals in T2DM subjects.[21]

Table VI. vHIT Findings						
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Defining Abnormality**	**Results**
Heystek (2018)	Cross Sectional	T1DM: 30 Control: 30	T1DM: 35.2 +/- 12.4 Control: 35.4 +/- 12.4	T1DM	Statistical Finding (p<.05)	**Gain:** T1DM subjects had a significantly lower left ear anterior gain (p<.001) and right ear posterior gain (p=.026) when compared to controls ; No significant difference between T1DM and control subjects for left lateral, left posterior, right lateral, or right anterior gain (p>.05); Despite significant differences observed between groups in the left anterior and right posterior canals, mean gain was within normal limits for all SCCs in the right and left ears **Saccades:** No significant difference in the presence of overt and covert saccades in the anterior, posterior, and lateral canals in the left and right ears of T1DM and control subjects (p>.05)
Ibraheem et al. (2021)	Cross Sectional	T1DM: 15 T2DM: 15 Control: 15	T1DM: 37.8±9.9 T2DM: 40.9±8.4 Control: 34.9±8.1	T1DM T2DM	Statistical Finding (p<.05)	**Gain:** T1DM and T2DM subjects had a significantly lower lateral canal gain than control subjects in the right (p=.001) and left (p=.035) ears; No significant difference between diabetic subjects and controls for anterior and posterior gain in the right and left ears (p>.05); Despite significant differences observed between groups in the lateral canals, mean gain was within normal limits for all SCCs in the right and left ears **Gain Asymmetry:** Lateral canal and LARP canal gain asymmetry is significantly greater in T1DM and T2DM subjects when compared to controls (p<.001); No significant difference between diabetic and control subjects for RALP canal gain asymmetry (p>.05)
Kalkan et al. (2018)	Cross Sectional	T2DM: 33 T2DM w/ DPN: 33 Control: 35	T2DM: 53.8±7.3 T2DM w/ DPN: 53.8±8.7 Control: 49.6±8.4	T2DM and T2DM w/ DPN	Statistical Finding (p<.05)	**Gain:** No significant difference between diabetic subjects and controls for anterior, posterior, and lateral canal gain in the right and left ears (p>.05); Mean gain was within normal limits for all SCCs in the right and left ears **Saccades:** No overt or covert saccades were observed in the anterior,

Table VI. vHIT Findings						
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Defining Abnormality**	**Results**
						posterior, or lateral canals in the right and left ears of T2DM, T2DM w/ DPN, and control subjects
Minnaar (2017)	Cross Sectional	T2DM: 28 Control: 28	T2DM: 49.2 +/- 6.1 Control: 49.0 +/- 6.4	T2DM	Statistical Finding (p<.05)	**Gain:** No significant difference between T2DM subjects and controls for anterior, posterior, and lateral canal gain in the right and left ears (p>.05); Mean gain was within normal limits for all SCCs in the right and left ears **Saccades:** T2DM subjects had a significantly higher occurrence of overt and covert saccades in the right lateral canal when compared to controls (p=..002); No significant difference between T2DM and control subjects for the presence of overt or covert saccades in the anterior and posterior canals of the right and left ears (p>.05)
Moossavi et al. (2021)	Cross Sectional	T1DM: 15 Control: 16	T1DM: 28 +/- 5.80 Control: 26 +/- 2.86	T1DM	Statistical Finding (p<.05)	**Gain:** No significant difference between T1DM subjects and controls for anterior, posterior, and lateral canal gain in the right and left ears (p>.05); Mean gain was within normal limits for all SCCs in the right and left ears
Omar et al. (2018)	Cross Sectional	T2DM: 8 Control: 8	T2DM: 36.8 +/- 11.4 Control: 34.6 +/- 11.0	T2DM	Statistical Finding (p<.05)	**Gain:** No significant difference between T2DM subjects and controls for anterior, posterior, and lateral canal gain in the right and left ears (p>.05); Mean gain was within normal limits for all SCCs in the right and left ears **Saccades:** No overt or covert saccades were observed in the anterior, posterior, or lateral canals in the right and left ears of T2DM and control subjects

Table VI. vHIT Findings

Article	Study Design	Sample Size	Mean Age	Diabetes Type	Defining Abnormality	Results
Ribeiro et al. (2019)	Cross Sectional	T1DM: 35 Control: 100	T1DM: 35.37 +/- 10.98 Control: 46.44 +/- 19.82	T1DM	Statistical Finding (p<.05)	**Gain:** T1DM subjects had significantly lower left anterior, right posterior, and left posterior gain when compared to controls (p<.001); No significant difference between T1DM subjects and controls for left lateral, right lateral, or right anterior gain (p>.05); Despite significant differences observed between groups in the left anterior, right posterior, and left posterior canals, mean gain was within normal limits for all SCCs in the right and left ears **Saccades:** Corrective saccades were not observed in the anterior, posterior, or lateral canals in the right and left ears of T1DM and control subjects

DPN: Diabetic Peripheral Neuropathy
LARP: Left Anterior, Right Posterior SCCs
RALP: Right Anterior, Left Posterior SCCs
T1DM: Type 1 Diabetes Mellitus
T2DM: Type 2 Diabetes Mellitus
vHIT: Video Head Impulse Testing

Cervical Vestibular Evoked Myogenic Potentials (cVEMP)

Table VII. outlines the fifteen studies which used the cVEMP to assess the function of the saccule and inferior vestibular nerve. Fourteen studies used a case-control style, while one compared several types and treatments of DM. The mean age of participants was 47.7 in the DM group(s) and 46 in the control group. Three studies evaluated those with T1DM, ten with T2DM, and two with either T1DM or T2DM. The average DM sample size was 33.8, with the largest study having 66 DM subjects,[24] and the smallest having 8 DM subjects.[39]

When evaluating the methods used to perform cVEMP testing on DM subjects, thirteen studies used a 500 Hz tone burst air conducted stimuli to elicit a response, one used a 750 Hz tone burst air conducted stimuli, and one used a 5 Hz click air conducted stimuli. Ten studies had the subjects in a sitting position during testing, two in a supine position, and two lying down. One study did not specify subject position during testing.

In five studies, cVEMP P1 and N1 latencies were significantly prolonged in DM subjects when compared to controls.[2, 20, 21, 25, 30] Eight studies did not find a statistically significant difference between DM and control groups,[3, 11, 19, 24, 34, 35, 39, 54] and one study did not evaluate cVEMP P1 and N1 latencies.[43] Kanumuri et al. (2018) used a descriptive analysis to illustrate how 20% of T2DM subjects had delayed P1 and N1 latencies when compared to controls. Further, Ibraheem et al. (2017) evaluated subjects with T1DM, T2DM treated with oral hypoglycaemic, and T2DM treated with insulin, and found T2DM subjects treated with insulin had significantly prolonged P1 and N1

latencies when compared to the other two groups. For this scoping review, latencies were considered abnormally prolonged if they were greater than 20 ms for P1, and greater than 28 ms for N1. Despite the significant differences observed between DM and control groups, when using the criteria outlined above to determine abnormality, almost all studies were found to have P1 and N1 latencies within normal limits. Kanumuri et al. (2018) found that 20% of T2DM subjects had delayed P1 and N1 latencies when using the criteria of P1 > 13.2 ± 1.27 and N1 > 22.19 ± 1.54. D'Silva et al. (2017) did not specify mean P1 or N1 latency for DM subjects.

In five studies, cVEMP P1-N1 amplitude was significantly smaller in DM subjects when compared to controls,[2, 20, 24, 43, 54] while seven studies did not find a statistically significant difference between groups.[3, 19, 21, 25, 30, 34, 39] Moossavi et al. (2021) found a significantly lower P1-N1 amplitude in only the left ear of T1DM subjects, with the right ear not reaching statistical significance. Ibraheem et al. (2017) found T2DM subjects treated with insulin had a significantly lower amplitude than T2DM subjects treated with oral hypoglycaemic. Two studies did not evaluate P1-N1 amplitude.[11, 26]

Six out of fifteen cVEMP studies evaluated cVEMP amplitude asymmetry ratio (AAR), with five finding no statistically significant difference between DM and control subjects.[20, 21, 25, 30, 34] Akan et al. (2021) found a significantly higher cVEMP AAR in T2DM subjects with diabetic polyneuropathy (DPN) when compared to controls. Further, Minnaar (2017) determined T2DM subjects have a 1.5 times higher risk of developing an abnormal cVEMP AAR than controls. Looking at the occurrence of abnormal or absent

cVEMP responses, three studies showed statistically significant higher absent/abnormal cVEMP responses in DM subjects when compared to controls.[2, 11, 55] Kanumuri et al. (2018) observed 10% of T2DM subjects had absent bilateral cVEMP responses, while Omar et al. (2018) found 25% of T2DM subjects had absent cVEMP responses. Ren et al. (2018) observed that 6.67% of T2DM subjects without early nephropathy (EN), and 10% of T2DM subjects with EN had bilaterally absent cVEMP responses. Lastly, Minnaar (2017) determined subjects with T2DM had a 2.1 times higher risk of having an absent cVEMP than controls. Eight studies did not discuss the prevalence of abnormal or absent cVEMP responses.[3, 19, 20, 21, 24, 25, 30, 35]

The impact of duration and/or severity of DM on cVEMP results was investigated by twelve studies. Three studies found a positive correlation between increased DM duration and a greater impairment of cVEMP results, [3, 20, 30] while two did not.[35, 54] Nine studies found a positive correlation between increased DM severity and a greater impairment of cVEMP results,[2, 11, 20, 21, 24, 25, 26, 30, 43] while three did not.[3, 35, 54]

Table VII. cVEMP Findings

Article	Study Design	Sample Size	Mean Age	Diabetes Type	Method	Defining Abnormality	Results
Akan et al. (2021)	Cross Sectional	T2DM with DPN: 35 Control: 34	T2DM w/ DPN: 53 +/- 18.3 Control: 51 +/- 11.1	T2DM w/ DPN	Stimuli: 500 Hz Toneburst Level: 97 dB Rate: 5/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**P1 Latency:** Significantly prolonged in the right and left ears of T2DM w/ DPN subjects when compared to controls (p= .001); Despite the difference between groups, mean P1 latency was within normal limits for DPN and control subjects in the right and left ears **N1 Latency:** Significantly prolonged in the right and left ears of T2DM w/ DPN subjects when compared to controls (p= .001); Despite the difference between groups, mean N1 latency was within normal limits for DPN and control subjects in the right and left ears **P1-N1 Amplitude:** Significantly lower in the right and left ears of T2DM w/ DPN subjects when compared to controls (p<.05) **cVEMP AAR:** Significantly higher in T2DM w/ DPN subjects when compared to controls (p= .001) **Absent cVEMP:** T2DM w/ DPN subjects had a significantly higher nonresponse rate for bilateral cVEMP when compared to controls (p<.05)
Bektas et al. (2008)	Cross Sectional	T2DM without PNP: 13 T2DM with PNP: 25 Control: 21	T2DM w/o PNP: 49.16 +/- 9.93 T2DM w/ PNP: 53.16 +/-7.98 Control: 49.38 +/- 4.93	T2DM w/o PNP T2DM w/ PNP	Stimuli: 5 Hz Click Level: 105 dB HL Duration: .1 ms Air Conduction Position: Supine	Statistical Finding (p<.05)	**P1 Latency:** No significant difference in P1 latency between diabetic and control subjects for the right or left ear (p>.05); Mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1 Latency:** No significant difference in N1 latency between diabetic and control subjects for the right or left ear (p>.05); Mean N1 latency was within normal limits for DM and control subjects in the right and left ears

Table VII. cVEMP Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
							P1-N1 Amplitude: No significant difference in P1-N1 amplitude between diabetic and control subjects for the right or left ear (p>.05)
D'Silva et al. (2017)	Cross Sectional	T2DM: 19 BPPV: 18 T2DM and BPPV: 14 Control: 20	T2DM: 58.6 ± 5.3 BPPV: 54.9 ± 5.9 T2DM and BPPV: 58.5 ± 5.6 Control: 57.5 ± 5.3	T2DM and T2DM w/ BPPV	Stimuli: 500 Hz Toneburst Level: 95 dB HL Rate: 5/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**P1 Latency:** No significant difference in P1 latency between T2DM, BPPV, T2DM w/ BPPV, and control subjects (p>.05); Mean P1 latency was unspecified for DM subjects **N1 Latency:** No significant difference in N1 latency between T2DM, BPPV, T2DM w/ BPPV, and control subjects (p>.05); Mean N1 latency was unspecified for DM subjects **Threshold:** No significant difference in threshold between T2DM, BPPV, T2DM w/ BPPV, and control subjects (p>.05) **Abnormal cVEMP:** T2DM, BPPV, and T2DM w/ BPPV subjects were significantly more likely to have abnormal cVEMP responses when compared to controls (p<.05)
Heystek (2018)	Cross Sectional	T1DM: 30 Control: 30	T1DM: 35.2 +/- 12.4 Control: 35.4 +/- 12.4	T1DM	Stimuli: 500 Hz Toneburst Level: 95 dB nHL Air Conduction Position: Sitting	Statistical Finding (p<.05)	**P1 Latency:** No significant difference in P1 latency between T1DM and control subjects for the right or left ear (p>.05); Mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1 Latency:** No significant difference in N1 latency between T1DM and control subjects for the right or left ear (p>.05); Mean N1 latency was within normal limits for DM and control subjects in the right and left ears **P1-N1 Amplitude:** No significant difference in P1-N1 amplitude between T1DM and control subjects for the right or left ear (p>.05)

Table VII. cVEMP Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
Ibraheem et al. (2017)	Cross Sectional	T1DM: 15 T2DM-Oral: 15 T2DM-Insulin: 15	T1DM: 33.87 ± 8.47 T2DM-Oral: 43.67 ± 5.33 T2DM-Insulin: 45.4 ± 3.87	T1DM, T2DM treated with oral hypoglycaemic, and T2DM treated with insulin	Stimuli: 500 Hz Toneburst Level: 99 dB nHL Rate: 7.1/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**P1 Latency:** T2DM w/ insulin subjects had a significantly longer P1 latency when compared to T2DM w/ oral and T1DM subjects (p<.05); Despite the difference between groups, mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1 Latency:** T2DM w/ insulin subjects had a significantly longer N1 latency when compared to T2DM w/ oral and T1DM subjects (p<.05); Despite the difference between groups, mean N1 latency was within normal limits for DM and control subjects in the right and left ears **P1-N1 Amplitude:** T2DM w/ insulin subjects had a significantly reduced amplitude when compared to T2DM w/ oral subjects (p<.05) **cVEMP AAR:** No significant difference in cVEMP AAR between diabetic groups (p>.05)
Ibraheem et al. (2021)	Cross Sectional	T1DM: 15 T2DM: 15 Control: 15	T1DM: 37.8±9.9 T2DM: 40.9±8.4 Control: 34.9±8.1	T1DM T2DM	Stimuli: 500 Hz Toneburst Level: 100 dB nHL Rate: 5/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**P1 Latency:** T1DM and T2DM subjects had significantly longer P1 latency when compared to controls in the right and left ear (p<.001); Despite the difference between groups, mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1 Latency:** T1DM and T2DM subjects had significantly longer N1 latency when compared to controls in the right and left ear (p<.001); Despite the difference between groups, mean N1 latency was within normal limits for DM and control subjects in the right and left ears **P1-N1 Amplitude:** No significant difference between diabetic and control subjects for P1-N1 amplitude in the right and left

Table VII. cVEMP Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
							ear (p>.05) **cVEMP AAR:** No significant difference between diabetic and control subjects for cVEMP AAR (p>.05)
Kalkan et al. (2018)	Cross Sectional	T2DM: 33 T2DM w/ DPN: 33 Control: 35	T2DM: 53.8±7.3 T2DM w/ DPN: 53.8±8.7 Control: 49.6±8.4	T2DM and T2DM w/ DPN	Stimuli: 500 Hz Toneburst Level: 105 dB nHL Rate: 5/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**P1 Latency:** No significant difference in P1 latency between diabetic and control subjects for the right or left ear (p>.05); Mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1 Latency:** No significant difference in N1 latency between diabetic and control subjects for the right or left ear (p>.05); Mean N1 latency was within normal limits for DM and control subjects in the right and left ears **P1-N1 Amplitude:** T2DM w/ DPN subjects had a significantly lower P1-N1 amplitude than T2DM and control subjects (p<.05)

Table VII. cVEMP Findings

Article	Study Design	Sample Size	Mean Age	Diabetes Type	Method	Defining Abnormality	Results
Kamali et al. (2013)	Cross Sectional	T1DM without PNP: 14 T1DM with PNP: 10 Control: 24	T1DM w/o PNP: 15-40 T1DM w/ PNP: 15-40 Control: 15-40	T1DM w/o PNP T1DM w/ PNP	Stimuli: 500 Hz Toneburst Level: 95 dB Rate: 5.1/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**P1 Latency:** T1DM w/ PNP subjects had a significantly longer P1 latency than T1DM w/o PNP and control subjects in the right and left ear (p<.05); No significant difference between T1DM w/o PNP and control subjects for the right or left ear (p>.05); Despite the differences observed between groups, mean P1 latency was within normal limits for all DM and control subjects in the right and left ears **N1 Latency:** T1DM w/ PNP subjects had a significantly longer P1 latency than T1DM w/o PNP (right ear) and control (right and left ear) subjects; No significant difference between T1DM w/o PNP and control subjects for the right or left ear (p>.05); Despite the differences observed between groups, mean N1 latency was within normal limits for all DM and control subjects in the right and left ears **P1-N1 Amplitude:** No significant difference between diabetic and control subjects for P1-N1 amplitude in the right or left ear (p>.05) **cVEMP AAR:** No significant difference between diabetic and control subjects for cVEMP AAR (p>.05)
Kanumuri et al. (2018)	Cross Sectional	T2DM: 40 Control: 20	T2DM: 20-60 Control: 20-60	T2DM	Stimuli: 500 Hz Toneburst Level: 105 dB nHL Rate: 5.1/sec Air Conduction Position: Lying down	Descriptive (%)	**Normal Response:** 70% of T2DM subjects had normal cVEMP responses **Delayed Response:** 20% of T2DM subjects had delayed P1 and N1 latencies (P1 > 13.2 ± 1.27, N1 > 22.19 ± 1.54) **Absent cVEMP:** 10% of T2DM subjects had absent bilateral cVEMP responses

Table VII. cVEMP Findings

Article	Study Design	Sample Size	Mean Age	Diabetes Type	Method	Defining Abnormality	Results
Konukseven et al. (2014)	Cross Sectional	T2DM: 30 Pre-Diabetic: 30 Control: 31	T2DM: 43.9 +/- 9.2 Pre-Diabetic: 46.4 +/- 9.2 Control: 45.0 +/- 8.5	T2DM Pre-Diabetic	Stimuli: 500 Hz Toneburst Level: 95 dB nHL Rate: 5.1/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**P1 Latency:** T2DM subjects had a significantly longer P1 latency when compared to pre-diabetic and control subjects (p<.001) ; Despite the differences observed between groups, mean P1 latency was within normal limits for all DM, pre-diabetic and control subjects in the right and left ears **N1 Latency:** T2DM subjects had a significantly longer N1 latency when compared to pre-diabetic and control subjects (p<.001) ; Despite the differences observed between groups, mean N1 latency was within normal limits for all DM, pre-diabetic and control subjects in the right and left ears **P1-N1 Amplitude:** No significant difference in P1-N1 amplitude between T2DM, pre-diabetic, and control subjects (p>.05) **cVEMP AAR:** No significant difference in cVEMP AAR between T2DM, pre-diabetic, and control subjects (p>.05)
Minnaar (2017)	Cross Sectional	T2DM: 28 Control: 28	T2DM: 49.2 +/- 6.1 Control: 49.0 +/- 6.4	T2DM	Stimuli: 500 Hz Toneburst Level: 97 dB nHL Air Conduction Position: Sitting	Descriptive (%) and Statistical Finding (P< .05)	**P1 Latency:** No significant difference in P1 latency between T2DM and control subjects (p>.05); Mean P1 latency was within normal limits for DM and control subjects **N1 Latency:** No significant difference in N1 latency between T2DM and control subjects (p>.05); Mean N1 latency was within normal limits for DM and control subjects **P1-N1 Amplitude:** No significant difference in P1-N1 amplitude between T2DM and control subjects (p>.05) **cVEMP AAR:** No significant difference in cVEMP AAR between T2DM and control subjects (p<.05) **Absent cVEMP:** T2DM subjects had a 2.1 times higher risk of having an absent cVEMP than controls.

Table VII. cVEMP Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
Moossavi et al. (2021)	Cross Sectional	T1DM: 15 Control: 16	T1DM: 28 +/- 5.80 Control: 26 +/- 2.86	T1DM	Stimuli: 500 Hz Toneburst Level: 97 dB nHL Rate: 5/sec Air Conduction Position: Unspecified	Statistical Finding (p<.05)	**P1 Latency:** No significant difference in P1 latency between T1DM and control subjects in the right or left ears (p>.05); Mean P1 latency was within normal limits for DM and control subjects **N1 Latency:** No significant difference in N1 latency between T1DM and control subjects in the right or left ears (p>.05); Mean N1 latency was within normal limits for DM and control subjects **P1-N1 Amplitude:** T1DM subjects had a significantly smaller P1-N1 amplitude in the left ear when compared to controls (p=.018); No significant difference in P1-N1 amplitude between T1DM and control subjects in the right ear (p>.05)
Omar et al. (2018)	Cross Sectional	T2DM: 8 Control: 8	T2DM: 36.8 +/- 11.4 Control: 34.6 +/- 11.0	T2DM	Stimuli: 750 Hz Toneburst Level: 100 dB SPL Rate: 5/sec Air Conduction Position: Sitting	Descriptive (%) and Statistical Finding (P< .05)	**P1 Latency:** No significant difference in P1 latency between T2DM and control subjects (p>.05); Mean P1 latency was within normal limits for DM and control subjects **N1 Latency:** No significant difference in N1 latency between T2DM and control subjects (p>.05); Mean N1 latency was within normal limits for DM and control subjects **P1-N1 Amplitude:** No significant difference in P1-N1 amplitude between T1DM and control subjects (p>.05) **Absent cVEMP:** 25% of T2DM subjects had absent cVEMP responses.

Table VII. cVEMP Findings

Article	Study Design	Sample Size	Mean Age	Diabetes Type	Method	Defining Abnormality	Results
Ren et al. (2018)	Cross Sectional	T2DM: 30 T2DM with EN: 30 Control: 30	T2DM: 56.40 +/- 8.46 T2DM w/ EN: 58.07 +/- 7.65 Control: 55.33 +/- 6.21	T2DM and T2DM w/ EN	Stimuli: 500 Hz Toneburst Level: 95 dB SPL Rate: 3/sec Air Conduction Position: Lying down	Descriptive (%) and Statistical Finding (P< .05)	**P1-N1 Amplitude:** T2DM and T2DM w/ EN subjects had a significantly lower P1-N1 amplitude than controls (p<.05); No significant difference observed between T2DM and T2DM w/ EN subjects for amplitude (p>.05) **Absent cVEMP:** 6.67% of T2DM subjects and 10% of T2DM w/ EN subjects showed bilaterally absent cVEMP responses
Ward et al. (2015)	Cross Sectional	T2DM: 25 Control: 25	T2DM: 64.7 +/- 7.6 Control: 63.8 +/- 8.7	T2DM	Stimuli: 500 Hz Toneburst Level: 125 dB SPL Rate: 5/sec Air Conduction Position: Supine	Statistical Finding (p<.05)	**P1 Latency:** No significant difference in P1 latency between T2DM and control subjects (p>.05); Mean P1 latency was within normal limits for DM and control subjects **N1 Latency:** No significant difference in N1 latency between T2DM and control subjects (p>.05); Mean N1 latency was within normal limits for DM and control subjects **P1-N1 Amplitude:** T2DM subjects had a significantly lower P1-N1 amplitude than controls (p<.05) **Absent cVEMP:** T2DM subjects had significantly more absent cVEMP responses than controls (p=02)

AAR: Amplitude Asymmetry Ratio
BPPV: Benign Paroxysmal Positional Vertigo
cVEMP: Cervical Vestibular-Evoked Myogenic Potentials
DPN: Diabetic Peripheral Neuropathy
EN: Early Nephropathy
N1: Negative Valley
P1: Positive Peak
PNP: Polyneuropathy

Ocular Vestibular Evoked Myogenic Potentials (oVEMP)

Table VIII. outlines the nine studies which used oVEMPs to assess the function of the utricle and superior vestibular nerve. All studies used a case-control style, with the average age of participants being 47.6 in the DM group(s) and 45.8 in the control group. Two studies evaluated those with T1DM and seven evaluated those with T2DM. The average DM sample size was 30, with the largest study having 66 subjects,[24] and the smallest having 8 subjects.[39]

When evaluating the methods used to perform oVEMP testing on DM subjects, seven studies used a 500 Hz tone burst air conducted stimuli to elicit a response, one used a 750 Hz tone burst bone conducted stimuli, and one used a tap reflex hammer on each subjects' forehead. Seven studies had the subjects in a sitting position during testing, and one study had subjects in a supine position. One study did not specify subject position during testing.

In four studies, oVEMP N1 latency was significantly prolonged in DM subjects when compared to controls,[24, 34, 35, 39] while four studies did not find a statistically significant difference between groups.[2, 11, 19, 30] Ward et al. (2015) used a descriptive analysis to illustrate 18% of T2DM subjects had delayed N1 latency when compared to controls. oVEMP P1 latency was significantly prolonged in 3 studies,[2, 30, 35] while four studies did not find a statistically significant difference between groups.[11, 19, 24, 34] Two studies did not evaluate P1 latency.[39, 54] For this scoping review, latencies were considered to be abnormally prolonged if they were greater than 14 ms for N1, and greater than 19 ms for

P1. Despite the significant differences observed between DM and control groups, when using the criteria outlined above to determine abnormality, almost all studies were found to have N1 and P1 latencies within normal limits. Ward et al. (2015) found that 18% of T2DM subjects had a delayed N1 latency when using the criteria of N1 > 10.3 ms or more than 2 standard deviations of the control mean latency. D'Silva et al. (2017) did not specify mean N1 or P1 latency for DM subjects.

In four studies, oVEMP N1-P1 amplitude was significantly smaller in DM subjects when compared to controls,[2, 19, 24, 54] while four studies did not find a statistically significant difference between groups.[30, 34, 35, 39] One study did not discuss N1-P1 amplitude.[11] Three studies evaluated oVEMP amplitude asymmetry ratio (AAR), with all finding no statistically significant difference between DM and control subjects.[2, 30, 34]

When looking at the occurrence of abnormal or absent oVEMP responses, two studies showed significantly higher absent/abnormal oVEMP responses in DM subjects when compared to controls,[2, 54] while one study did not observe a difference between groups.[11] Minnaar (2017) used a descriptive analysis to determine T2DM subjects have a 1.3 times higher risk of developing abnormal or absent oVEMP responses when compared to controls. Five studies did not evaluate DM subjects for abnormal or absent oVEMP responses.[19, 24, 30, 35, 39]

The impact of duration and/or severity of DM was investigated by five oVEMP studies. Two studies found a significant correlation between increased DM duration/severity and impaired oVEMP results,[2, 30] while three did not.[11, 35, 54] Akan et al. (2021) found that

oVEMP results became increasingly more impaired as subjects developed greater diabetic severity. Konukseven et al. (2014) mentioned a statistically significant correlation between increased HbA1c levels and prolonged oVEMP n1 latency.

Table VIII. oVEMP Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
Akan et al. (2021)	Cross Sectional	T2DM with DPN: 35 Control: 34	T2DM w/ DPN: 53 +/- 18.3 Control: 51 +/- 11.1	T2DM w/ DPN	Stimuli: 500 Hz Toneburst Level: 97 dB Rate: 5/sec Air Conduction Position: Sitting	Statistical Finding (P< .05)	**N1 Latency:** Significantly prolonged in the right and left ears of T2DM w/ DPN subjects when compared to controls (p<.003); Despite the difference between groups, mean N1 latency was within normal limits for DPN and control subjects in the right and left ears **P1 Latency:** Significantly prolonged in the right and left ears of T2DM w/ DPN subjects when compared to controls (p= .001); Despite the difference between groups, mean P1 latency was within normal limits for DPN and control subjects in the right and left ears **N1-P1 Amplitude:** Significantly lower in the right and left ears of T2DM w/ DPN subjects when compared to controls (p<.05) **oVEMP AAR:** No significant difference in oVEMP AAR between T2DM w/ DPN and control subjects (p>.05) **Absent oVEMP:** T2DM w/ DPN subjects had a significantly higher nonresponse rate for bilateral oVEMP when compared to controls (p<.05)
D'Silva et al. (2017)	Cross Sectional	T2DM: 19 BPPV: 18 T2DM and BPPV: 14 Control: 20	T2DM: 58.6 ± 5.3 BPPV: 54.9 ± 5.9 T2DM and BPPV: 58.5 ± 5.6	T2DM and T2DM w/ BPPV	Stimuli: 500 Hz Toneburst Level: 125 dB SPL Rate: 5/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**N1 Latency:** T2DM subjects had a significantly longer latency when compared to controls (p=.03); No significant difference in N1 latency between BPPV and control subjects (p>.05); Mean N1 latency was unspecified for DM subjects **P1 Latency:** No significant difference in P1 latency between T2DM, BPPV, T2DM w/ BPPV, and control subjects (p>.05); Mean P1 latency was unspecified for DM subjects

Table VIII. oVEMP Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
			Control: 57.5 ± 5.3				**Threshold:** No significant difference in threshold between T2DM, BPPV, T2DM w/ BPPV, and control subjects (p>.05) **Abnormal oVEMP:** No significant difference in the presence of abnormal oVEMP responses in T2DM, BPPV, and T2DM w/ BPPV subjects when compared to controls (p>.05)
Heystek (2018)	Cross Sectional	T1DM: 30 Control: 30	T1DM: 35.2 +/- 12.4 Control: 35.4 +/- 12.4	T1DM	Stimuli: 500 Hz Toneburst Level: 95 dB nHL Air Conduction Position: Sitting	Statistical Finding (p<.05)	**N1 Latency:** T1DM subjects had a significantly longer N1 latency in the right ear when compared to controls (p=.036); No significant difference in N1 latency between T1DM and control subjects for the left ear (p>.05); Despite the differences observed between groups, mean N1 latency was within normal limits for DM and control subjects in the right and left ears **P1 Latency:** No significant difference in P1 latency between T1DM and control subjects for the right or left ear (p>.05); Mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1-P1 Amplitude:** T1DM subjects had a significantly smaller N1-P1 amplitude for the right and left ear when compared to controls (p<.05)
Kalkan et al. (2018)	Cross Sectional	T2DM: 33 T2DM w/ DPN: 33 Control: 35	T2DM: 53.8±7.3 T2DM w/ DPN: 53.8±8.7 Control: 49.6±8.4	T2DM and T2DM w/ DPN	Stimuli: 500 Hz Toneburst Level: 105 dB nHL Rate: 5/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**N1 Latency:** No significant difference in N1 latency between diabetic and control subjects for the right or left ear (p>.05); Mean N1 latency was within normal limits for DM and control subjects in the right and left ears **P1 Latency:** No significant difference in P1 latency between diabetic and control subjects for the right or left ear (p>.05); Mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1-P1 Amplitude:** Control subjects had a significantly higher

Table VIII. oVEMP Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
							N1-P1 amplitude than diabetic subjects (p<.05); T2DM w/ DPN subjects had a significantly lower N1-P1 amplitude than T2DM and control subjects (p<.05)
Konukseven et al. (2014)	Cross Sectional	T2DM: 30 Pre-Diabetic: 30 Control: 31	T2DM: 43.9 +/- 9.2 Pre-Diabetic: 46.4 +/- 9.2 Control: 45.0 +/- 8.5	T2DM Pre-Diabetic	Stimuli: 500 Hz Toneburst Level: 95 dB nHL Rate: 5.1/sec Air Conduction Position: Sitting	Statistical Finding (p<.05)	**N1 Latency:** T2DM subjects had a significantly longer N1 latency when compared to pre-diabetic and control subjects (p<.001); Despite the differences observed between groups, mean N1 latency was within normal limits for all DM, pre-diabetic and control subjects in the right and left ears **P1 Latency:** T2DM subjects had a significantly longer P1 latency when compared to pre-diabetic and control subjects (p<.001); Despite the differences observed between groups, mean P1 latency was within normal limits for all DM, pre-diabetic and control subjects in the right and left ears **N1-P1 Amplitude:** No significant difference in P1-N1 amplitude between T2DM, pre-diabetic, and control subjects (p>.05) **oVEMP AAR:** No significant difference in oVEMP AAR between T2DM, pre-diabetic, and control subjects (p>.05)
Minnaar (2017)	Cross Sectional	T2DM: 28 Control: 28	T2DM: 49.2 +/- 6.1 Control: 49.0 +/- 6.4	T2DM	Stimuli: 500 Hz Toneburst Level: 97 dB nHL Air Conduction Position: Sitting	Descriptive (%) and Statistical Finding (P< .05)	**N1 Latency:** No significant difference in N1 latency between T2DM and control subjects (p>.05); Mean N1 latency was within normal limits for DM and control subjects in the right and left ears **P1 Latency:** No significant difference in P1 latency between T2DM and control subjects (p>.05); Mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1-P1 Amplitude:** No significant difference in N1-P1

Table VIII. oVEMP Findings							
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Method**	**Defining Abnormality**	**Results**
							amplitude between T2DM and control subjects (p>.05) **oVEMP AAR:** No significant difference in oVEMP AAR between T2DM and control subjects (p>.05) **Abnormal oVEMP:** T2DM subjects had a 1.3 times higher risk of having an abnormal/absent oVEMP than controls.
Moossavi et al. (2021)	Cross Sectional	T1DM: 15 Control: 16	T1DM: 28 +/- 5.80 Control: 26 +/- 2.86	T1DM	Stimuli: 500 Hz Toneburst Level: 97 dB nHL Rate: 5/sec Air Conduction Position: Unspecified	Statistical Finding (p<.05)	**N1 Latency:** No significant difference in N1 latency between T1DM and control subjects in the right or left ears (p>.05); Mean N1 latency was within normal limits for DM and control subjects in the right and left ears **P1 Latency:** T1DM subjects had a significantly longer P1 latency in the right and left ears when compared to controls (p=.004); Despite the observed difference between groups, mean P1 latency was within normal limits for DM and control subjects in the right and left ears **N1-P1 Amplitude:** No significant difference in N1-P1 amplitude between T1DM and control subjects in the right or left ears (p>.05)
Omar et al. (2018)	Cross Sectional	T2DM: 8 Control: 8	T2DM: 36.8 +/- 11.4 Control: 34.6 +/- 11.0	T2DM	Stimuli: 750 Hz Toneburst Level: 50 dB nHL Rate: 5/sec Bone Conduction Position: Sitting	Statistical Finding (p<.05)	**N1 Latency:** No significant difference in N1 latency between T2DM and control subjects (p>.05); Mean N1 latency was within normal limits for DM and control subjects in the right and left ears **N1 Amplitude:** No significant difference in peak-to-base amplitude between T2DM and control subjects (p>.05)
Ward et al. (2015)	Cross Sectional	T2DM: 25 Control: 25	T2DM: 64.7 +/- 7.6	T2DM	Stimuli: Tap Reflex Hammer	Descriptive (%) and Statistical	**N1 Latency:** 18% of T2DM subjects had a delayed N1 latency (Delayed Latency: N1 > 10.3 ms or more than 2 standard

Table VIII. oVEMP Findings

Article	Study Design	Sample Size	Mean Age	Diabetes Type	Method	Defining Abnormality	Results
			Control: 63.8 +/- 8.7		to Forehead Position: Supine	Finding (P< .05)	deviations of the control mean latency) **N1 Amplitude:** T2DM subjects had a significantly smaller N1 amplitude when compared to controls (p=.04) **Absent oVEMP:** T2DM subjects had significantly more absent oVEMP responses than controls (p=04)

AAR: Amplitude Asymmetry Ratio
BPPV: Benign Paroxysmal Positional Vertigo
DPN: Diabetic Peripheral Neuropathy
N1: Negative Valley
oVEMP: Ocular Vestibular-Evoked Myogenic Potentials
P1: Positive Peak

Discussion

Overview

The purpose of this scoping review was to understand the current state of knowledge regarding DM and vestibular function and to identify gaps in knowledge that still need to be explored. Only recently has there been increased interest into how DM is impacting the vestibular system. Of the forty-three studies included in this scoping review, approximately 70% were published in the last thirteen years. DM sample sizes were relatively small, with included studies having an average sample size of 50.7 DM participants. The largest study had 104 DM subjects,[40] while the smallest had 5 DM subjects.[10] Further, the majority (79.1%) of studies evaluated adults 18 years of age or older, with the average participant age being 45.9 years. Only one study[12] focused their research solely on children with DM, leaving little information known about how the disease is impacting vestibular function in the youngest members of our society.

When looking at the vestibular diagnostic measures used to assess individuals with DM, approximately 65% of studies only used one direct measure of vestibular function (VNG/ENG, vHIT, Rotary Chair, cVEMP, oVEMP, etc.). The majority of included studies did not use multiple direct measures to obtain a full understanding of how DM may be impacting the entire system. For example, over half of studies used the caloric test to assess the horizontal SCC and superior vestibular nerve, while much fewer studies assessed the SCCs with the Rotary Chair or vHIT. Further, a little over one-third of

studies used cVEMP to assess the saccule and inferior vestibular nerve, while only one-fifth of studies used oVEMP to assess the utricle and superior vestibular nerve.

Vestibular Diagnostic Assessments in Subjects with DM

The most common vestibular measure utilized by studies included in this scoping review was VNG/ENG. The most frequently used diagnostic assessment within the VNG/ENG was the caloric test which assesses the horizontal SCC and superior vestibular nerve. While the caloric test was performed by over half (53.5%) of the included studies in this scoping review, the findings revealed significant variability in relation to how the tests were performed and how the results were analyzed. Over half (12/23 or 52.2%) of studies did not outline the methods used to conduct the study, did not provide specific quantitative data in relation to caloric test results, and/or did not provide enough information for future replicability. As a result, it is difficult to determine the prevalence of caloric impairments in individuals with DM.

Some studies showed no significant difference between DM and control subjects for the caloric measures of UW and BW. In contrast, other studies found significant differences between DM and control groups, with upwards of 21.05% of DM subjects being diagnosed with UW and 33.3% of subjects being diagnosed with BW. Due to the variability in how the studies were performed and analyzed, none of them can be compared to determine why so many inconsistencies exist in the test findings.

When looking at the eight studies which used positional testing (i.e., Dix-Hallpike) and/or questionnaires to determine the presence of posterior SCC BPPV, all but two identified the presence of BPPV in individuals with DM. In the six (75%) studies which identified BPPV in DM subjects, the percentage of those impacted ranged from 7.7% to 46%. While the number of impacted individuals varies widely, these findings suggest BPPV may be more prevalent in individuals with DM. As a result, this pathology should be investigated more thoroughly in future research.

Only two of the forty-three studies included in this scoping review utilized the rotary chair to assess vestibular function in individuals with DM. While the findings are minimal, they consistently showed no significant differences between DM and control subjects for VOR gain[22] and pre-/post-rotary nystagmus.[29] Rotary chair assesses the horizontal SCCs and superior vestibular nerves of both ears simultaneously. As a result, if there is a compensated unilateral loss, it will not be apparent on the rotary chair examination if only evaluating VOR gain. VOR phase is a more sensitive measure of the VOR, but none of the studies analyzed this. Due to the limited knowledge available, future research should utilize the rotary chair and include measures of VOR phase so that information regarding possible unilateral and/or bilateral weaknesses resulting from DM can be better understood.

vHIT is a direct measure of all six SCCs and the superior and inferior vestibular nerves. Only one study reported a higher occurrence of overt and covert saccades in subjects with DM, and it was only in the right lateral SCC.[34] Of the seven studies which measured

vHIT gain, four did not find a statistically significant difference between DM and control subjects. The three studies which identified significant differences between DM and control subjects varied in which SCCs they identified as abnormal. Some studies found lower left anterior SCC [19,44] vHIT gain, while others identified lower right posterior SCC [44] or lower left lateral SCC [21] vHIT gain. Although there were significant differences identified between DM and control groups, mean vHIT gain was within normal limits for all six SCCs in all seven studies. The normal vHIT gain measured in all DM and control subjects limits the clinical significance of the statistically significant differences observed between these groups.

cVEMP testing was used by fifteen studies to assess the saccule and inferior vestibular nerve of individuals with DM. A third (5/15) of cVEMP studies identified significantly prolonged P1 and N1 latencies in DM subjects when compared to controls. Although there were significant differences identified between these groups, mean P1 and N1 latencies were found to be within normal limits for both the DM and control groups in almost all studies which measured it. The normal P1 and N1 latencies measured in almost all DM and control subjects limits the clinical significance of the statistically significant differences observed between these groups.

When looking at cVEMP P1-N1 amplitude, five studies found it to be significantly smaller in DM subjects when compared to controls. Seven studies did not find a statistically significant difference between groups, and two studies did not evaluate for P1-N1 amplitude. It should be noted that if electromyography (EMG) was not corrected

for when measuring absolute cVEMP amplitudes, then the responses should be interpreted with caution. Outside factors, such as muscle flexion from the sternocleidomastoid (SCM) muscle, can result in greater levels of EMG, which can lead to larger cVEMP amplitude responses. It could be argued that the most sensitive diagnostic index of the cVEMP is the interaural amplitude asymmetry ratio (AAR). Of the six studies which measured AAR, five did not find a statistically significant difference between DM and control subjects. However, Minnaar (2017) determined that individuals with T2DM have a 1.5 times higher risk of developing an abnormal cVEMP AAR than control subjects.

oVEMP testing was used by nine studies to assess the utricle and superior vestibular nerve of individuals with DM. Less than half (4/9) of the oVEMP studies identified significantly prolonged N1 latencies in DM subjects when compared to controls. Three of the oVEMP studies identified significantly prolonged P1 latencies in DM subjects when compared to controls. Although there were significant differences identified between groups, mean N1 and P1 latencies were found to be within normal limits for both the DM and control groups in almost all studies which measured it. The normal N1 and P1 latencies measured in almost all DM and control subjects limits the clinical significance of the statistically significant differences observed between these groups.

When looking at oVEMP N1-P1 amplitude, four studies found it to be significantly smaller in DM subjects when compared to controls. Four studies did not find a statistically significant difference between groups, and one study did not evaluate for N1-

P1 amplitude. Reduced oVEMP amplitudes suggest smaller responses, but this does not typically hold clinical significance. It could be argued that the most sensitive diagnostic index of the oVEMP is the interaural amplitude asymmetry ratio (AAR). Of the three studies which measured AAR, none found a statistically significant difference between DM and control groups.

Duration and Severity of DM

The majority of caloric studies (11/14, 78.6%) found a positive correlation between increased DM duration and a greater impairment of caloric test results, while 8/13 (61.5%) found a positive correlation between increased DM severity and a greater impairment of caloric findings. Further, 1/1 (100%) studies found a positive correlation between increased DM severity and abnormal vHIT test findings.

Additionally, 3/5 (60%) studies found a positive correlation between increased DM duration and a greater impairment of cVEMP test results and 9/12 (75%) found a positive correlation between increased DM severity and a greater impairment of cVEMP findings. Finally, 2/5 (40%) studies found a positive correlation between increased DM severity and a greater impairment of oVEMP test findings.

In summary, 20/27 (74%) studies which investigated the impact of DM duration and/or severity on vestibular measures found at least one positive correlation between increased DM severity and/or duration and abnormal vestibular test findings. This suggests that,

when severity and duration of DM are accounted for in vestibular analyses, they tend to have a significant impact on the results obtained.

Conclusions

The most consistent finding of this scoping review was an increased prevalence of BPPV in individuals with DM and an association between disease duration and/or severity with abnormal vestibular findings. The percentage of those impacted with BPPV ranged widely from 7.7% to 46%. The large range of individuals with DM suspected to have BPPV supports the need for future research in this area. Caloric testing, rotary chair, and horizontal vHIT all assess the lateral SCCs with different frequency ranges and types of stimulation. With relatively normal rotary chair and horizontal vHIT findings observed in this study, it would be unusual for DM to only have a negative impact on caloric test results. If the caloric test were to be the only abnormal measure of the lateral SCCs, it would suggest an isolated low frequency vestibular impairment in individuals with DM. It should be noted, however, that caloric findings were inconsistent across studies and there was no uniform reporting of methods to allow for meaningful comparisons between studies. For this reason, the prevalence of caloric impairments in individuals with DM is still not well understood.

Lastly, 74% of studies which investigated the impact of DM duration and/or severity on vestibular measures found at least one correlation between increased DM severity and/or duration and a greater likelihood of developing abnormal vestibular test results. As DM becomes more prevalent in our society, it is essential a standardized test battery be developed to more efficiently evaluate and diagnose vestibular disorders in this population. Findings from this study may help develop a narrower research question

which could be used to conduct a systematic review. Findings from this study may also assist in the development of a randomized control trial (RCT) involving individuals with DM.

Appendices

Appendix I. PubMed (MEDLINE) Search Strategy

Diabetes[tiab] OR “Diabetes Mellitus”[tiab] OR “Type 2 diabetes”[tiab] OR "Diabetes Mellitus, Type 2/complications"[Mesh] OR "Diabetes Mellitus, Type 2/epidemiology"[Mesh] OR "Diabetes Mellitus, Type 2/etiology"[Mesh] OR "Diabetes Mellitus, Type 2/pathology"[Mesh] OR "Diabetes Mellitus, Type 2/physiopathology" OR "Diabetes Mellitus, Type 2/prevention and control"[Mesh] OR "Diabetes Mellitus/complications"[Mesh] OR "Diabetes Mellitus/diagnosis"[Mesh] OR "Diabetes Mellitus/epidemiology"[Mesh] OR "Diabetes Mellitus/etiology"[Mesh] OR "Diabetes Mellitus/pathology"[Mesh] OR "Diabetes Mellitus/physiopathology"[Mesh]

AND

Vestibular[tiab] OR "Vestibular Diseases/classification"[Mesh] OR "Vestibular Diseases/diagnosis"[Mesh] OR "Vestibular Diseases/epidemiology"[Mesh] OR "Vestibular Diseases/etiology"[Mesh] OR "Vestibular Diseases/pathology"[Mesh] OR "Vestibular Diseases/physiology"[Mesh] OR "Vestibular Diseases/physiopathology"[Mesh]

Number of Results: 281

Appendix II. ProQuest-Dissertation and Theses Global Search Strategy

Diabetes OR “Diabetes Mellitus” OR “Type 1 Diabetes” OR “Type 2 Diabetes”

AND

Vestibular

Number of Results: 20

Appendix III. Ocular Motility Findings

Ocular Motility Findings						
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Defining Abnormality**	**Results**
Aantaa & Lehtonen (1981)	Cross Sectional	Insulin-Treated Diabetes: 24	Insulin-Treated Diabetes: 34	Insulin-Treated T1DM and T2DM	Descriptive (%)	**SP:** Normal in 100% of T1DM & T2DM (insulin treated) subjects **OKN:** Normal in 100% of T1DM & T2DM (insulin treated) subjects
Biurrun et al. (1991)	Cross Sectional	T1DM: 46 Control: 33	T1DM: 25.9 +/- 8.9 Control: 26.2 +/- 9.4	T1DM	Descriptive (%)	**Spontaneous Nystagmus:** 15.2% of T1DM subjects
Darlington et al. (2000)	Cross Sectional	T1DM: 26 T2DM: 27 Control: 21	T1DM: 20-84 T2DM: 20-84 Control: 20-84	T1DM and T2DM	Statistical Finding (p<.05)	**Gaze-Holding in Darkness:** Significantly worse in T1DM (p<.05) and T2DM (p<.0005) subjects when compared to controls **OKN:** Significantly higher mean SPV for T1DM subjects when compared to controls (p<.05); No significant difference in mean SPV for T2DM subjects when compared to control subjects
Doyle (2005)	Cross Sectional	T2DM: 5 Control: 9	T2DM: 55.8 Control: 48.9	T2DM	Statistical Finding (p<.05)	**SP:** Significantly lower high frequency gain in T2DM subjects when compared to controls (p<.05); No significant difference in low frequency gain between T2DM and controls **Gaze-Holding:** No significant difference between T2DM and control subjects **Saccades:** Significantly lower rightward velocity in T2DM subjects when compared to controls (p<.05); No significant difference between T2DM and control subjects for accuracy and latency **OKN:** No significant difference between T2DM and control subjects

Ocular Motility Findings						
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Defining Abnormality**	**Results**
El Shafei et al. (2021)	Cross Sectional	T1DM: 25 Control: 25	T1DM: 10.4 +/- 2.7 Control: 10.11+/- 2.6	T1DM	Statistical Finding (P< .05)	**Spontaneous Nystagmus:** No significant difference between T1DM subjects and controls (p>.05) **SP:** No significant difference between T1DM subjects and controls (p>.05) **Saccades:** T1DM subjects had significantly longer latencies than controls (p<.0001); T1DM subjects had significantly slower velocities than controls (p=.042); No significant difference in accuracy between T1DM subjects and controls **OKN:** No significant difference between T1DM subjects and controls (p>.05)
Gawron et al. (2002)	Cross Sectional	T1DM: 95 Control: 44	T1DM: 15.5 +/- 5.1 Control: 16.3 +/- 6.1	T1DM	Descriptive (%)	**Spontaneous Nystagmus:** 25% of T1DM subjects and 0% of controls **SP:** Impaired in 34.7% of T1DM subjects and 4.55% of controls **OKN:** Impaired in 37.9% of T1DM subjects and 6.8% of controls
Gawron et al. (2011)	Cross Sectional	T1DM: 59 Control: 33	T1DM: 20 Control: 19.2	T1DM	Descriptive (%) and Statistical Finding (P< .05)	**Spontaneous Nystagmus:** 11% of T1DM subjects and 0% of controls **SP:** Significantly high phase value in T1DM subjects when compared to controls (p<.05) **Saccades:** Significantly decreased accuracy in T1DM subjects when compared to controls (p<.05) **OKN:** No significant difference in asymmetry or mean SPV between T1DM subjects and controls (P>.05)

Ocular Motility Findings						
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Defining Abnormality**	**Results**
Ibraheem et al. (2017)	Cross Sectional	T1DM: 15 T2DM-Oral: 15 T2DM-Insulin: 15	T1DM: 33.87 ± 8.47 T2DM-Oral: 43.67 ± 5.33 T2DM-Insulin: 45.4 ± 3.87	T1DM, T2DM treated with oral hypoglycaemic, and T2DM treated with insulin	Statistical Finding (p<.05)	**SP:** Gain was significantly better for T2DM subjects treated with oral hypoglycaemic when compared to T1DM and T2DM subjects treated with insulin (p=.000) **Saccades:** Accuracy was significantly better for T2DM subjects treated with oral hypoglycaemic when compared to T1DM and T2DM subjects treated with insulin (p<.05); No significant difference between diabetic groups for latency and velocity **OKN:** Gain was significantly greater for T2DM subjects treated with oral hypoglycaemic and significantly smaller for T2DM subjects treated with insulin (p=.000)
Kalkan et al. (2018)	Cross Sectional	T2DM: 33 T2DM w/ DPN: 33 Control: 35	T2DM: 53.8±7.3 T2DM w/ DPN: 53.8±8.7 Control: 49.6±8.4	T2DM and T2DM w/ DPN	Descriptive (%)	**Spontaneous/Gaze Nystagmus:** Absent in all subjects
Kim et al. (2012)*	Retrospective	T1DM: 10 T2DM: 25	T1DM: 51.1 +/- 15.5 T2DM: 51.1 +/- 15.5	T1DM w/ DPN and T2DM w/ DPN	Descriptive (%)	**Spontaneous/Gaze Testing:** 57.9% of subjects with DPN (T1DM & T2DM) were diagnosed with vestibular dysfunction.
Klagenberg et al. (2007)	Cross Sectional	T1DM: 30	T1DM: 25.7	T1DM	Descriptive (%)	**Spontaneous Nystagmus:** Normal in all T1DM subjects **SP:** Normal in all T1DM subjects **OKN:** Normal in all T1DM subjects
Kuniyil et al. (2020)	Cross Sectional	DM: 97	DM: 54.68 +/- 10.68	Unspecified DM type	Statistical Finding (p<.05)	**Gaze-Holding:** Normal in all DM subjects **Saccades:** Normal in all DM subjects **OKN:** Normal in all DM subjects

Ocular Motility Findings						
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Defining Abnormality**	**Results**
Nicholson et al. (2002)	Cross Sectional	T1DM: 18 T2DM: 23 Control: 45	T1DM: 62.7 +/- 21.1 T2DM: 65.4 +/- 10.5 Control: 60.9 +/- 8.2	T1DM and T2DM	Statistical Finding (p<.05)	**Gaze-Holding in Darkness:** Rightward gaze was significantly worse for T2DM subjects than controls (p<.05); Leftward gaze was significantly worse for T1DM subjects than controls (p<.01); Leftward gaze was significantly worse for T1DM subjects than T2DM subjects (p<.05) **OKN:** SPV for CW and CCW rotation was significantly lower for T1DM subjects than controls (p<.01); SPV for CW rotation was significantly lower for T2DM subjects than controls (p<.0001); Quick phase amplitude for CCW rotation was significantly smaller for T1DM subjects than controls (p<.05)
Ozel et al. (2013)	Cross Sectional	T2DM: 104 Control: 104	T2DM: 50.3 Control: 48.3	T2DM	Statistical Finding (p<.05)	**SP:** Significantly more T2DM subjects had impaired SP than controls (p<.001) **Gaze-Holding:** Significantly more T2DM subjects had impaired gaze-holding than controls (p=.002) **Saccades:** Significantly more T2DM subjects had impaired saccades than controls (p<.001) **OKN:** Significantly more T2DM subjects had impaired OKN than controls (p<.001)
Ren et al. (2018)*	Cross Sectional	T2DM: 30 T2DM w/ early nephropathy: 30 Control: 30	T2DM: 56.40 +/- 8.46 T2DM w/ early nephropathy: 58.07 +/- 7.65 Control: 55.33 +/- 6.21	T2DM and T2DM w/ early nephropathy	Statistical Finding (p<.05)	**Spontaneous Nystagmus:** Within normal range for T2DM and T2DM with early nephropathy subjects **SP:** Within normal range for T2DM and T2DM with early nephropathy subjects **Gaze-Holding:** Within normal range for T2DM and T2DM with early nephropathy subjects **Saccades:** Within normal range for T2DM and T2DM with early nephropathy subjects **OKN:** Within normal range for T2DM and T2DM with early nephropathy subjects

Ocular Motility Findings						
Article	**Study Design**	**Sample Size**	**Mean Age**	**Diabetes Type**	**Defining Abnormality**	**Results**
Rigon et al. (2007)	Cross Sectional	T1DM: 19 Control: 19	T1DM: 8-25 Control: 8-25	T1DM	Statistical Finding (P< .05)	**Spontaneous Nystagmus:** No significant difference between T1DM subjects and controls (p>.05) **SP:** No significant difference between T1DM subjects and controls (p>.05) **Gaze-Holding:** No significant difference between T1DM subjects and controls (p>.05) **OKN:** No significant difference between T1DM subjects and controls (p>.05)
Scherer & Lobo (2002)	Cross Sectional	T1DM: 12	T1DM: </= 40	T1DM	Descriptive (%)	**Spontaneous Nystagmus:** Not present in anyT1DM subjects **SP:** Type I Pendulum Tracking observed in 58.3% of T1DM subjects; Type II Pendulum Tracking observed in 41.7% of T1DM subjects **OKN:** No asymmetry present in any T1DM subjects
Virtaniemi et al. (1993)	Cross Sectional	T1DM: 53 Control: 42	T1DM: 33.0 +/- 6.0 Control: 31.4 +/- 5.2	T1DM	Statistical Finding (p<.05)	**SP:** Maximum eye movement velocity was significantly reduced at all target velocities for T1DM subjects when compared to controls (p<.05) **Saccades:** Reaction time was longer and accuracy was decreased more significantly in T1DM subjects than controls (p<.01)

*No quantitative data provided by study
CW: Clockwise
CCW: Counterclockwise
DPN: Diabetic Peripheral Neuropathy
OKN: Optokinetic Nystagmus
SP: Smooth Pursuit
SPV: Slow Phase Velocity
T1DM: Type 1 Diabetes Mellitus
T2DM: Type 2 Diabetes Mellitus

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