

Quantitative assessment of posture stability using computerised dynamic posturography in type 2 diabetic patients with neuropathy and its relation to glycaemic control

Emam A A, Gad A M, Ahmed M M, Assal H S, Mousa S G

ABSTRACT

Introduction: Patients with diabetic neuropathy have an imbalance, which comes with a higher risk of falls. The aim of this study was to assess posture stability using computerised dynamic posturography in type 2 diabetics mellitus patients with neuropathy as well as its relation to glycaemic control.

Methods: 54 type 2 diabetics mellitus patients with peripheral neuropathy were recruited, together with 18 type 2 diabetics mellitus patients without peripheral neuropathy acting as the control group. The first group was divided into two subgroups according to glycaemic control assessed by HbA1c (A1c), the first subgroup comprising 24 patients had good glycaemic control with A1c less than or equal to seven percent and the second subgroup with 30 patients had poor glycaemic control with A1c more than 7 percent. The postural stability was evaluated using dynamic posturography.

Results: The composite equilibrium score, sensory organisation test 1, 2 and 3 conditions were significantly lower in the neuropathic group as compared to the non-neuropathic group (p-value is less than 0.001). A1c was significantly correlated with the composite equilibrium score in the neuropathic group with poor glycaemic control (r-value equal to -0.395) but not correlated in the neuropathic group with good glycaemic control (r-value equal to 0.151).

Conclusion: Posture instability in type 2 diabetic patients with peripheral neuropathy reflects an impairment of the somatosensory system; also, poor glycaemic control resulted in more posture instability. The early detection of imbalance

using dynamic posturography and achieving good glycaemic control may be of great help in the prevention of falls in such patients.

Keywords: glycaemic control, computerised dynamic posturography, diabetes mellitus, glycaemic control, peripheral neuropathy, type 2 diabetes mellitus

Singapore Med J 2009;50(6):614-618

INTRODUCTION

Peripheral neuropathy has a variable prevalence of around 32.3% among type 2 diabetic patients.⁽¹⁾ Although Centomo et al have reported that diabetes mellitus per se has no effect on posture control during quiet standing,⁽²⁾ the ability to maintain an upright posture is compromised in type 2 diabetic patients with peripheral neuropathy.⁽³⁾ Postural control is a common mechanism to maintain the body when it is subjected to unpredictable displacements. As the ability of postural control decreases with ageing or neuropathy, a fall due to imbalance is a common cause of morbidity and mortality.⁽⁴⁾ The loss of sensory perception has a deleterious effect on postural stability.⁽⁵⁾ Patients with diabetic sensory neuropathy and poor postural control are more susceptible to having a high risk of falls,⁽⁶⁾ which can be attributed to the lack of accurate proprioceptive feedback from the lower limbs.⁽⁷⁾

Dynamic posturography is an important test in the evaluation of imbalance in patients with polyneuropathy.⁽⁸⁾ Posturography allows for the early detection of disequilibrium⁽⁹⁾ and has also been used to document postural instability in diabetic patients with peripheral neuropathy.⁽¹⁰⁾ In a study done by Maurer et al, falls by type 2 diabetic patients were associated with poor performance in the Berg Balance Scale test.⁽¹¹⁾ The aim of this study was to assess posture stability using computerised dynamic posturography in type 2 diabetic patients with neuropathy as well as its relation to glycaemic control.

Internal Medicine Department, National Research Center, Cairo 12311, Egypt

Emam AA, MD
Assistant Professor

Assal H S, MD
Assistant Professor

Internal Medicine Department, Al-Azhar University, Faculty of Medicine, Naser City, Cairo 12531, Egypt

Gad AM, MD
Assistant Professor

Mousa SG, MD
Assistant Professor

Rheumatology & Rehabilitation Department

Ahmed MM, MD
Professor

Correspondence to:
Dr Ahmed Alsayed Emam
Tel: (20) 2 3582 7948
Fax: (20) 1379 0265
Email: ahmedemam6@hotmail.com

METHODS

A total of 54 type 2 diabetic patients with peripheral neuropathy (mean age 63.96 ± 3.45 years, mean duration of diabetes mellitus 12.5 ± 7.8 years) were recruited from the diabetic clinic of one hospital, together with 18 age- and gender-matched type 2 diabetic patients without peripheral neuropathy who acted as the control group (mean age 64.33 ± 4.26 years, mean duration of diabetes mellitus 11.2 ± 6.8 years). The patients were not selected sequentially, and a sample size testing and power of study were done, with a value of 0.80 (80%) obtained. All patients were asked to provide their complete medical history and were subjected to a thorough neurological examination.

Peripheral neuropathy was diagnosed by Hoffmann's reflex and a Nihon-Kohden Neuropack® electromyograph. The test evaluates the latency and amplitude of the response of a peripheral nerve to an electrical stimulus of increasing intensity applied to the tibial nerve for the detection of peripheral neuropathy.⁽¹²⁾ The diabetic patients with peripheral neuropathy were divided into two subgroups according to their glycaemic control assessed by HbA1c (A1c) concentrations; the first subgroup ($n = 24$) had good glycaemic control with $A1c \leq 7\%$ and the second subgroup ($n = 30$) had poor glycaemic control with $A1c > 7\%$.

The inclusion criteria were: (1) ability to ambulate 25 feet independently; (2) good cognitive functions; and (3) good visual field and acuity. The exclusion criteria were: (1) those with vestibular abnormalities; (2) those with nephropathy and retinopathy; (3) those with a history of antihypertensive medication that might produce postural hypotension, or drug intake that affect postural stability; (4) those with musculoskeletal disorders that contribute to postural instability; and (5) those with cardiac, renal and liver diseases. The patients gave their informed voluntary consent to participate in the study according to the protocol approved by the local ethics committee and in accordance with the ethical standards of the Helsinki declaration.

Fasting venous samples were collected for the estimation of blood sugar, liver and renal functions. The A1c level was measured using high-performance liquid chromatography instruments (HLC-723 GHB IIIs; Tosoh Corporation, Tokyo, Japan) with a reference range of 4.5%–6.2%. All patients were evaluated for postural stability using computerised dynamic posturography. The SMART Balance Master (NeuroCom International Inc, Clackamas, OR, USA) was used for the postural stability assessment. Blood glucose was measured before performing the tests to rule out hypoglycaemia.

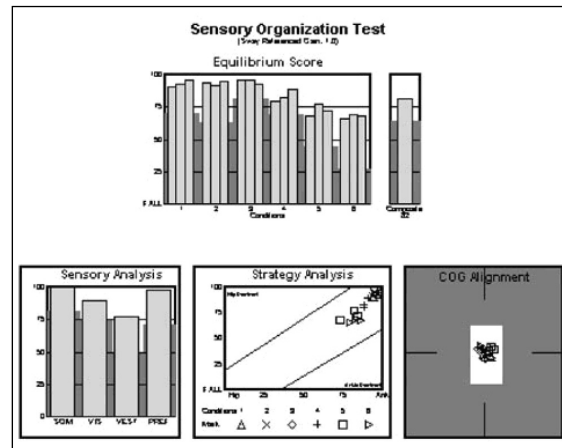


Fig. 1 Sample graphs of normal posturography with sensory organisation test results.

The sensory organisation test (SOT), which was also performed, included six test conditions. The first three conditions (static posturography) involved the patient standing on a fixed platform with eyes open (SOT 1), eyes closed (SOT 2) and using sway-referenced vision (SOT 3). The second three conditions (dynamic posturography) involved the patient standing on a moving platform with the conditions of SOT 4 (eyes open), SOT 5 (eyes closed) and SOT 6 (using sway-referenced vision).

The composite equilibrium score was calculated, and describes the overall level of performance during all the SOT trials. This score is a weighted average of the equilibrium scores from the 18 trials (three for each of the six conditions). Higher scores indicate better postural stability. Equilibrium scores from each of the trials represent a percentage with which the peak amplitude of anterior-posterior sway is compared with a theoretical anterior-posterior limit of stability. The scores ranged from 0 (touching a support surface, shifting feet or falling) to 100(%) (little or no sway). A reprint of normal posturography with a SOT is shown in Fig. 1. The control group without peripheral neuropathy was included, and the observers were not informed about the aim of the work in order to eliminate observer bias.

The Statistical Package for Social Sciences version 12.0 (SPSS Inc, Chicago, IL, USA) was used for data processing. Quantitative data were presented as mean and standard deviation and the Student *t*-test was used for a comparison of the means. A correlation between variables was done and the Pearson correlation coefficient was calculated. All tests were two-tailed and considered to be statistically significant at $p < 0.05$.

RESULTS

Our results showed that the composite equilibrium score,

Table I. Clinical data, serum HbA1c and parameters of the sensory organisation test in diabetic patients with and without peripheral neuropathy.

	Diabetics with PN (n = 54) mean ± SD (95% CI)	Diabetics without PN (n = 18) mean ± SD (95% CI)	p-value
Age (years)	63.96 ± 3.45 (63.01–64.91)	64.33 ± 4.26 (62.22–66.41)	NS
Duration of diabetes mellitus (years)	12.5 ± 7.8 (10.30–15.76)	11.2 ± 6.8 (9.56–13.67)	NS
No. of falls in last six months	≤ 3 (n = 15) > 3 (n = 7)	– –	– –
Medications:			
Oral hypoglycaemic	n = 37	n = 11	–
Insulin	n = 17	n = 7	–
Fasting blood sugar (mmol/L)	10.4 ± 2.2 (8.62–12.36)	9.6 ± 1.3 (7.61–10.89)	NS
Serum creatinine (umol/L)	112.3 ± 6.1 (106.62–116.36)	110.5 ± 7.6 (107.3–114.8)	NS
HbA1c (%)	7.24 ± 1.17 (6.92–7.97)	7.08 ± 0.50 (6.83–7.34)	NS
Composite equilibrium score (%)	67.77 ± 3.65 (66.77–68.78)	88.97 ± 2.69 (87.64–90.32)	0.001
SOT1	81.04 ± 4.36 (79.81–82.23)	92.61 ± 3.55 (90.85–94.38)	0.001
SOT2	71.51 ± 3.32 (70.60–72.42)	87.50 ± 2.63 (86.19–88.81)	0.001
SOT3	71.22 ± 2.64 (70.23–72.22)	84.69 ± 1.92 (83.34–85.65)	0.001
SOT4	75.75 ± 4.44 (74.82–76.7)	75.38 ± 2.67 (73.05–75.72)	0.12
SOT5	65.77 ± 4.66 (64.51–67.05)	66.08 ± 2.34 (64.92–67.26)	0.76
SOT6	63.45 ± 2.65 (62.73–64.18)	62.65 ± 2.13 (61.56–63.71)	0.24

PN: peripheral neuropathy; SOT: sensory organisation test; NS: not significant; CI: confidence interval

Table II. Clinical data, serum HbA1c concentrations, parameters of the sensory organisation test in diabetic patients with peripheral neuropathy (poor vs. good glycaemic control).

Variables	PN with poor glycaemic control (n = 30) mean ± SD (95% CI)	PN with good glycaemic control (n = 24) mean ± SD (95% CI)	p- value
HbA1c (%)	8.09 ± 0.75 (7.81–8.37)	6.18 ± 0.60 (5.93–6.44)	0.001
Composite equilibrium score (%)	68.08 ± 3.92 (66.61–69.59)	67.38 ± 3.34 (65.79–68.80)	0.49
SOT1	81.60 ± 4.05 (80.9–83.12)	80.34 ± 4.72 (78.35–82.34)	0.29
SOT2	71.32 ± 4.03 (68.81–72.82)	71.75 ± 2.21 (70.81–72.69)	0.64
SOT3	71.91 ± 3.80 (70.47–73.33)	70.37 ± 3.30 (68.97–71.77)	0.12
SOT4	74.96 ± 2.68 (73.95–75.96)	76.75 ± 4.05 (75.04–78.47)	0.06
SOT5	65.66 ± 2.78 (64.62–66.70)	65.92 ± 6.35 (63.24–68.61)	0.89
SOT6	62.80 ± 2.08 (62.02–63.59)	64.02 ± 2.86 (62.96–65.57)	0.08

PN: peripheral neuropathy; SOT: sensory organisation test; CI: confidence interval

Table III. Correlation between serum HbA1c concentrations and the composite equilibrium score of the SOT test in diabetic mellitus patients with peripheral neuropathy (poor vs. good glycaemic control).

	HbA1c (good glycaemic control) (n = 24)	HbA1c (poor glycaemic control) (n = 30)
Composite equilibrium score	r = 0.151	r = -0.395*

* Correlation is significant at the 0.05 level (two-tailed).

SOT 1, 2 and 3 conditions were significantly lower in the neuropathic group as compared to the non-neuropathic group. However, in dynamic conditions, there was no significant difference between the two groups, as shown in Table I. In the neuropathic group, there was no significant difference in the composite equilibrium score and all parameters of the SOT test between the good and poor glycaemic control subgroups, as shown in Table II. However, A1c was significantly correlated with the

composite equilibrium score in the neuropathic group with poor glycaemic control, but not correlated in the neuropathic group with good glycaemic control, as shown in Table III and Figs. 2 and 3.

DISCUSSION

The postural control is a complex system that controls the orientation and balance of the body when it is in an upright posture.^(13,14) Imbalance is commonly found in patients

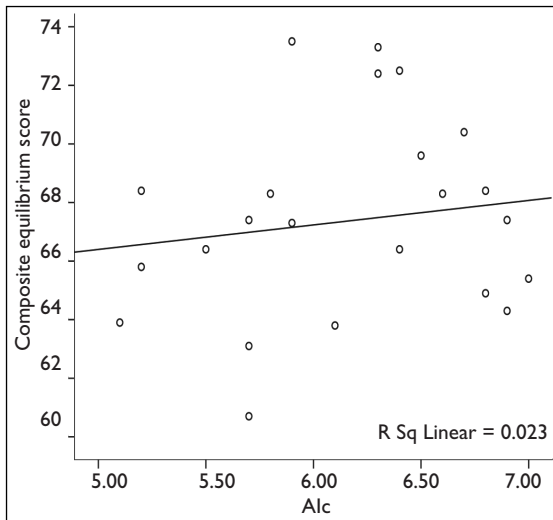


Fig. 2 Scatterplot shows the correlation between the serum A1c concentrations and composite equilibrium score of the SOT test in diabetic mellitus patients with peripheral neuropathy and good glycaemic control.

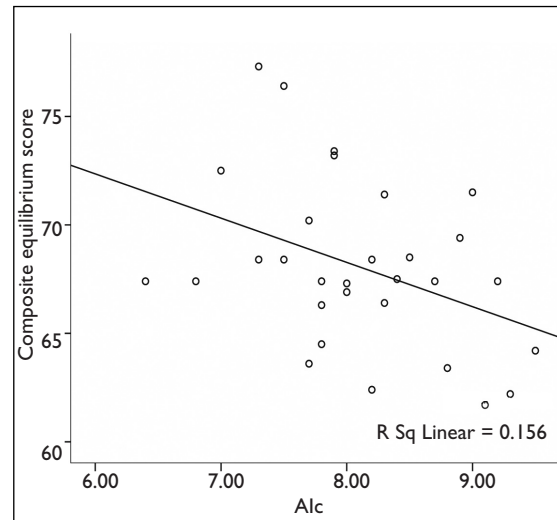


Fig. 3 Scatterplot shows the correlation between the serum A1c concentrations and composite equilibrium score of the SOT test in diabetic mellitus patients with peripheral neuropathy and poor glycaemic control.

with polyneuropathy due to impaired proprioception and motor functions.^(15,16) The afferent sensory input from the proprioceptors as well as the efferent motor nerves must be intact in order to maintain balance.⁽¹⁷⁾ Most, but not all, studies of older adults with diabetes mellitus have shown that peripheral neuropathy is a risk factor for falls.⁽¹⁸⁻²¹⁾ In a study conducted on 77 patients with diabetes mellitus, Tilling et al reported that 50% of those who fell in their study were suffering from diabetic peripheral neuropathy.⁽²²⁾

In the present study, posture stability using computerised dynamic posturography in type 2 diabetic patients with neuropathy and its relation to glycaemic control was evaluated. Our results showed that the composite equilibrium score, SOT 1, 2 and 3 conditions were significantly lowered in neuropathic as compared to non-neuropathic patients. However, in dynamic conditions, there was no significant difference between the two groups. These results may reflect the impairment of the somatosensory system, rather than vestibular and/or visual disorders;⁽⁹⁾ similar findings were reported by Di Nardo et al.⁽²³⁾

The mechanisms by which peripheral neuropathy leads to postural instability are complex. Peripheral neuropathy has been shown to impair ankle strength and balance recovery⁽²⁴⁾ as well as walking stability in diabetics.⁽²⁵⁾ The higher vibration threshold perceptions in type 2 diabetic patients with peripheral neuropathy who had a history of falls significantly suggest that sensory deficit is an important contributory factor in falling,⁽²⁶⁾ and a shift from physiological ankle control to hip postural control has also been documented.⁽²⁷⁾ Moreover, Lafond et al have demonstrated that ankle motor activities are

affected in patients with diabetic sensory neuropathy during quiet standing.⁽⁶⁾

Our results also showed that in the neuropathic group, there was no significant difference in the composite equilibrium score and all parameters of the SOT test between those with good and those with poor glycaemic control. However, A1c was significantly correlated with the composite equilibrium score in the neuropathic group with poor glycaemic control, and this correlation did not exist in those with good glycaemic control. Our results were consistent with Tilling et al who reported an increased fall risk with poor glycaemic control (A1c > 7%),⁽²²⁾ however, Miller et al reported a lack of this association.⁽²⁸⁾ Schwartz et al found no association between poor glycaemic control and an increased fall risk, and this association was only found in insulin-treated type 2 diabetics with good glycaemic control (A1c < 6%), which may be related to hypoglycaemic episodes. However, good glycaemic control with oral hypoglycaemic medications was not associated with more frequent falls.⁽²⁹⁾

The presence of postural instability in poorly-controlled diabetics could be associated with severe nerve damage due to long-standing hyperglycaemia. Hyperglycaemia is associated with the glycosylation of antioxidant enzymes that make the oxygen free radical scavenger system less efficient; the resulting high oxidative stress plays an important role in the pathogenesis of diabetic microangiopathy.⁽³⁰⁾ Moreover, chronic hyperglycaemia is associated with the activation of the polyol pathway and the accumulation of sorbitol and fructose in nerve cells, which lead to (Na⁺/K⁺)-ATPase dysfunction⁽³¹⁾ and subsequent demyelination of the peripheral nerves. Thus, good

glycaemic control is the best approach to minimising the prevalence and severity of diabetic polyneuropathy and for the prevention of postural instability. The small sample size from only one centre is a limitation of the current study.

In conclusion, computerised dynamic posturography is an important tool in the assessment of posture instability and allows for early disclosure of the failure of the postural control system. Our data suggests that the impairment of posture stability in type 2 diabetic patients with peripheral neuropathy reflects an impairment of the somatosensory system. In addition, poor glycaemic control resulted in more frequent posture instability. The early detection of posture instability using computerised dynamic posturography and achieving good glycaemic control could be of great help in the prevention of fall-related morbidity and mortality in diabetics with peripheral neuropathy.

ACKNOWLEDGEMENTS

We are grateful to all the staff at the diabetes and balance clinics for their support. We would also like to thank all the patients who participated in this study.

REFERENCES

- Fedele D, Comi G, Coscelli C, et al. A multicenter study on the prevalence of diabetic neuropathy in Italy. *Italian Diabetic Neuropathy Committee. Diabetes* 1997; 20:836-43.
- Centomo H, Termoz N, Savoie S, Béliveau L, Prince F. Postural control following a self-initiated reaching task in type 2 diabetic patients and age-matched controls. *Gait Posture* 2007; 25:509-14.
- Yamamoto R, Kinoshita T, Momoki T, et al. Postural sway and diabetic peripheral neuropathy. *Diabetes Res Clin Pract* 2001; 52:213-21.
- Kim BJ, Robinson CJ. Effects of diabetic neuropathy on body sway and slip perturbation detection in older population. *Int J Occup Saf Ergon* 2006; 12:241-54.
- Simoneau GG, Ulbrecht JS, Derr JA, Becker MB, Cavanagh PR. Postural instability in patients with diabetic sensory neuropathy. *Diabetes Care* 1994; 17:1411-21.
- Lafond D, Corriveau H, Prince F. Postural control mechanisms during quiet standing in patients with diabetic sensory neuropathy. *Diabetes Care* 2004; 27:173-8.
- Oppenheim U, Kohen-Raz R, Alex D, Kohen-Raz A, Azarya M. Postural characteristics of diabetic neuropathy. *Diabetes Care* 1999; 22:328-33.
- Ledin T, Odkvist LM, Vrethem M, Möller C. Dynamic posturography in assessment of polyneuropathic disease. *J Vestib Res* 1990-1991; 1:123-8.
- Uccioli L, Giacomini PG, Monticone G, et al. Body sway in diabetic neuropathy. *Diabetes Care* 1995; 18:339-44.
- Boucher P, Teasdale N, Courtemanche R, Bard C, Fleury M. Postural stability in diabetic polyneuropathy. *Diabetes Care* 1995; 18:638-45.
- Maurer MS, Burcham J, Chen H. Diabetes mellitus is associated with an increased risk of falls in elderly residents of a long-term care facility. *J Gerontol A Biol Sci Med Sci* 2005; 60:1157-62.
- Kandel ER, Schwartz JH, Jessel TM. Principles of neurology science. In: Dumitru D. *Electrodiagnostic Medicine*. New York: McGraw-Hill, 2000: 730-1.
- Macpherson JM, Fung J, Jacobs R. Postural orientation, equilibrium, and the spinal cord. *Adv Neurol* 1997; 72:227-32.
- Horak FB, Macpherson JM. Postural orientation and equilibrium. In: Rowell LB, Shepherd JT, eds. *Handbook of Physiology. Exercise: Regulation and Integration of Multiple Systems*. New York: American Physiological Society, 1996.
- Voorhees RL. Dynamic posturography findings in central nervous system disorders. *Otolaryngol Head Neck Surg* 1990; 103:96-101.
- Shepard NT, Telian SA, Smith-Wheelock M. Habituation and balance retraining therapy: A retrospective review. *Neurol Clin* 1990; 8:459-75.
- Nasher LM. Adapting reflexes controlling the human posture. *Exp Brain Res* 1976; 26:59-72.
- Strotmeyer ES, Cauley JA, Schwartz AV, et al. Nontraumatic fracture risk with diabetes mellitus and impaired fasting glucose in older white and black adults. *Arch Intern Med* 2005; 165:1612-7.
- Richardson JK, Hurvitz EA. Peripheral neuropathy is a risk factor for falls. *J Gerontol A Biol Sci Med Sci* 1995; 50:M211-5.
- Volpato S, Leveille SG, Blaum C, Fried LP, Guralnik JM. Risk factors for falls in older disabled women with diabetes: the Women's Health and Aging Study. *J Gerontol A Biol Sci Med Sci* 2005; 60:1539-45.
- Franklin GM, Kahn LB, Baxter J, Marshall JA, Hamman RF. Sensory neuropathy in non-insulin-dependent diabetes mellitus. The San Luis Valley Diabetes Study. *Am J Epidemiol* 1990; 131:633-43.
- Tilling LM, Darawil K, Britton M. Falls as a complication of diabetes mellitus in older people. *J Diabetes Complications* 2006; 20:158-62.
- Di Nardo W, Ghirlanda G, Cercone S, et al. The use of dynamic posturography to detect neurosensorial disorder in IDDM without clinical neuropathy. *J Diabetes Complications* 1999; 13:79-85.
- Menz HB, Lord SR, St George R, Fitzpatrick RC. Walking stability and sensorimotor function in older people with diabetic peripheral neuropathy. *Arch Phys Med Rehabil* 2004; 85:245-52.
- Wallace C, Reiber GE, LeMaster J, et al. Incidence of falls, risk factors for falls and fall related fractures in individuals with diabetes and a prior foot ulcer. *Diabetes Care* 2002; 11:1983-6.
- Patel S, Hyer S, Tweed K, et al. Risk factors for fractures and falls in older women with type 2 diabetes mellitus. *Calcif Tissue Int* 2008; 82:87-91.
- Giacomini PG, Bruno E, Monticone G, et al. Postural rearrangement in IDDM patients with peripheral neuropathy. *Diabetes Care* 1996; 19:372-4.
- Miller DK, Lui LY, Perry HM 3rd, Kaiser FE, Morley JE. Reported and measured physical functioning in older inner-city diabetic African Americans. *J Gerontol A Biol Sci Med Sci* 1999; 54: M230-6.
- Schwartz AV, Vittinghoff E, Sellmeyer DE, et al. Diabetes-related complications, glycemic control, and falls in older adults. *Diabetes Care* 2008; 31:391-6.
- Head KA. Peripheral neuropathy: pathogenic mechanisms and alternative therapies. *Altern Med Rev* 2006; 11:294-329.
- Raccach D, Coste T, Cameron NE, et al. Effect of the aldose reductase inhibitor tolrestat on nerve conduction velocity, Na/K ATPase activity, and polyols in red blood cells, sciatic nerve, kidney cortex and kidney medulla of diabetic rats. *J Diabetes Complications* 1998; 12:154-62.