

Effect of Balance Training Program on Patient with Balance Deficits Due to Uncontrolled Diabetes with Neuropathy

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ABSTRACT

The purpose of the study was to determine the effect of balance training program on patients with type one un controlled diabetes with neuropathy . Thirty subjects shared in this study assumed two equal groups, fifteen were normal (GI) and fifteen with type one uncontrolled diabetes with neuropathy (GII), Assessment was done by Biodex balance system via the dynamic balance test which including anteroposterior, mediolateral and overall stability index. Group II was trained for two months. The results revealed that there was no significant difference between the balance parameters in the second group post treatment than pre treatment, this indicates that there was no improvement in balance in GII after receiving the balance training program. It can be concluded that performing balance training program does not improve balance in type one uncontrolled diabetic subjects.

Key words: Diabetic neuropathy, balance training, and Biodex system.

INTRODUCTION

Diabetes is a complex group of syndromes that result in malfunction of the beta cell located in the islets of Langerhans of the Pancreas, whose function is the production of insulin¹. The complications of diabetes affect all body systems including the neuromuscular system in the form of sensory, motor and autonomic neuropathies. Motor neuropathy that occurs in diabetes is characterized by muscle atrophy, changes in gait, new pressure points and finally ulceration in foot. Sensory neuropathy is characterized by loss of sensation, bone changes, deformed foot, painless trauma, and finally ulceration in the foot. Autonomic neuropathy is characterized by decrease in perspiration, dry skin cracks fissures, infection, moderate sized areas of gangrene and finally amputation. An individual with diabetes may not experience any pain, even with serious vascular disease, because neuropathy can diminish the feeling or

perception of these symptoms. Neuropathy is associated with the lack of senses of touch and pain that provide gait protection³.

Balance is controlled on the basis of afferent information from the somatosensory, visual and vestibular systems. The first two systems are often affected in the presence of diabetes and also participate in increasing the risk of falling among this population⁵. The somatosensory system is the biggest contributor of feedback for postural control. This sensory system is composed of several different muscle, joint, and cutaneous mechanoreceptors. The information from these receptors is integrated in the central nervous system to produce sensation of joint position and movement. The different receptors do not be seen to equally contribute to kinesthesia. For control of upright posture in individual without pathologic conditions the importance of muscle spindles particularly of the lower leg has been established in numerous studies¹².

Diabetic neuropathy impairs muscle spindle function. As the neuropathic process progresses, its effect on the muscle spindle function increases. This could mean loss of either afferent or gamma efferent nerve to muscle spindle in the lower leg. Diabetes damages the spindle receptors themselves⁶.

People with diabetes can develop nerve problems at any time, but significant clinical neuropathy can develop within the first ten years after receiving diabetes diagnosis so about 60% of people with diabetes have some form of neuropathy⁷.

The aim of this study was to measure balance parameters in normal and type one uncontrolled diabetes with neuropathy. In addition studying the effect of balance program on the control of balance.

MATERIAL AND METHODS

Patients population:

Thirty volunteer subject shared in this study. The normal group (GI) consisted of

fifteen subjects; they have no past history of any musculoskeletal problems, matched in age, sex, weight, height and socio-economic level. The study group (GII) consisted of fifteen patients (males and females). The age of both groups was 50 - 60 years.

Patients in the study group were diagnosed and referred by specialized physician from diabetic outpatient clinic, faculty of Medicine, Cairo University. The chronicity of diabetes was at least twenty years.

Equipment:

Biodex balance system

Is a balance screening and training tool. It consists of a movable balance platform, which provides up to 20 degree of surface tilt in a 360 degree range. (Biodex medical system Inc, Shirley New York, U.S.A . The stability levels available by the system range from a completely firm surface (stability level 8) to a very unstable surface (stability level 1)¹². The computer analyze the patient movements and determine in which directions the patient desire to move or is having difficulty moving.

The dynamic balance test parameters include

- a- Anterior posterior stability index: represent the patient's ability to control their balance in front to back directions. High values represent less stability in all indices of the system.
- b- Mediolateral stability index: represent the patient's ability to control their balance from side to side.
- c- Overall stability index: represent the patient's ability to control their balance in all direction.

Procedure of the study:

Both the control and the study group were assessed. The study group received dynamic balance training for two months three times weekly, at stability level six.

Balance training program

The Biodex training program was performed in standing position as well as testing. The subject was instructed to focus on the visually feedback screen directly in front of him and attempt to maintain the cursor at the center of the screen while standing on the unstable platform (stability level six) for a period of five minutes. The treatment session was five minutes, three times weekly for two months.

RESULTS

By using the paired t test (OA, AP and ML stability index) at two levels of stability eight and six during the dynamic balance test. Reassessment was done for the study group at two levels of stability eight and six during the dynamic balance test and then compared with the control group . Measurement was done at two levels of stability eight and six.

Table (1): Stability indices for the normal control group at stability level eight and six.

| Stability index (SI) | Level eight | Level six |
|---------------------------|--------------|--------------|
| | X± SD | X± SD |
| Overall stability | 3.35 ± 1.12 | 3.57 ± 1.17 |
| Anteroposterior stability | 2.82 ± 1.11 | 2.96 ± 1.15 |
| Mediolateral stability | 2.14 ± 0.732 | 2.42 ± 0.714 |

Overall stability index: The mean values of OA index of the control group at stability level eight and six were 3.35 ± 1.12 and 3.57 ± 1.17 respectively.

Anteroposterior stability index: The mean values of AP stability of the control group at stability level eight and six were 2.82 ± 1.11 and 2.96 ± 1.15 respectively.

Mediolateral stability index: The mean value of ML stability of the control group at stability level eight and six were 2.14±0.732 and 2.42 ± 0.714 respectively.

Table (2): Stability indices for the study group at stability level eight.

| Level eight Stability Index (SI) | X±SD | | t value | Sign. |
|----------------------------------|---------------|---------------|---------|--------|
| | Pre | Post | | |
| Overall stability | 11.406 ± 1.44 | 11.306 ± 1.43 | 0.001 | P>0.05 |
| Anteroposterior stability | 9.39 ± 1.25 | 9.28 ± 1.20 | 0.001 | P>0.05 |
| Mediolateral stability | 8.39 ± 1.14 | 8.29 ± 1.11 | 0.000 | P>0.05 |

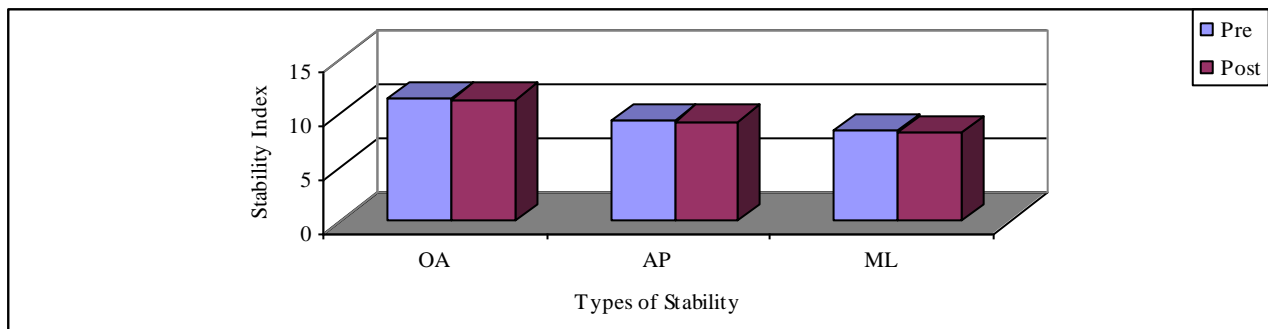


Fig. (1): Stability indices for the study group at stability level eight.

Overall stability index: The mean values of OA index at stability level eight for the study group pre and post treatment were 11.406 ± 1.44 and 11.306 ± 1.43 respectively.

Anteroposterior stability index: The mean values of AP index for the study group pre and

post treatment were 9.39 ± 1.25 and 9.28 ± 1.20 respectively.

Mediolateral stability index: The mean value of ML index for the study group pre and post treatment were 8.39 ± 1.14 and 8.29 ± 1.11 respectively.

Table (3): Stability indices for the study group at stability level six.

| Level six Stability Index (SI) | X±SD | | t value | Sign. |
|--------------------------------|-------------|-------------|---------|--------|
| | Pre | Post | | |
| Overall stability | 11.73± 1.39 | 11.58± 1.32 | 0.006 | P>0.05 |
| Anteroposterior stability | 9.6 ± 1.2 | 9.52 ± 1.22 | 0.002 | P>0.05 |
| Mediolateral stability | 8.6 ± 1.1 | 8.47 ± 1.07 | 0.000 | P>0.05 |

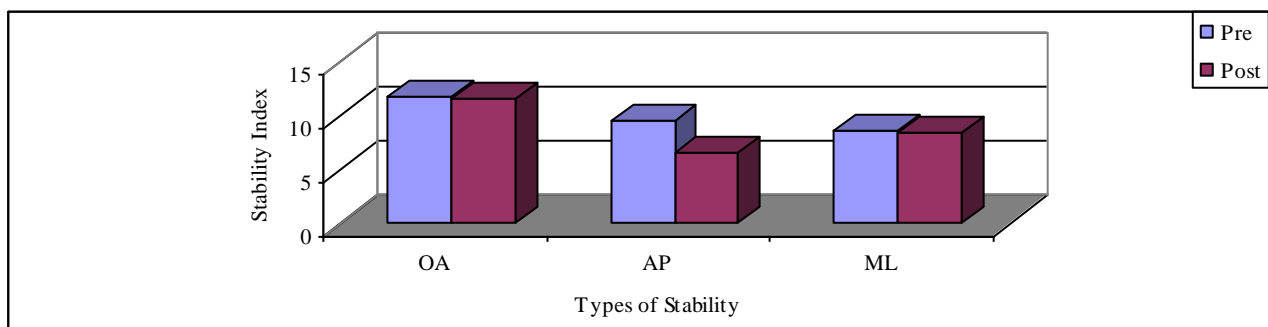


Fig. (2): Stability indices for the study group at stability level six.

Overall stability: The mean values of OA index at stability level six for the study group pre and post treatment were 11.73± 1.39 and 11.58 ± 1.32 respectively.

Anteroposterior stability: The mean values of AP index for the study group pre and post

treatment were 9.6 ± 1.2 and 9.52 ± 1.22 respectively.

Mediolateral stability: The mean value of ML index for the study group pre and post treatment were 8.6 ± 1.1 and 8.47 ± 1.07 respectively.

Table (4): Comparison between stability indices for the study group pre treatment and the control group at stability level eight.

| Stability Index (SI) | | X±SD | t value | Sign. |
|----------------------|---------------|------------|---------|--------|
| Overall | Study group | 11.4± 1.44 | 0.00 | P<0.05 |
| | Control group | 3.35±1.12 | | |
| Anteroposterior | Study group | 9.39±1.25 | 0.00 | P<0.05 |
| | Control group | 2.82±1.11 | | |
| Mediolateral | Study group | 8.39± 1.14 | 0.00 | P<0.05 |
| | Control group | 2.14 ±0.73 | | |

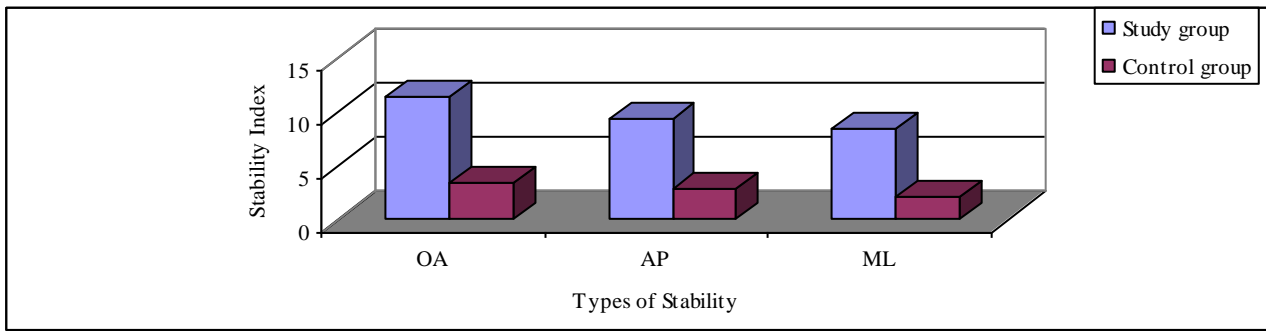


Fig. (3): Stability indices for the study group pre treatment and the control group at stability level eight.

Overall stability: Mean values of OA index pre treatment for study group and control group were 11.4 ± 1.44 and 3.35 ± 1.12 respectively showed high significant differences.

Anteroposterior Stability: Mean values of AP stability pre treatment for the study group and the control group were 9.39 ± 1.25 and

2.82 ± 1.11 respectively showed high significant differences.

Mediolateral Stability: Mean values of ML stability pre treatment for the study group and the control group were 8.39 ± 1.14 and 2.14 ± 0.73 respectively, showed high significant differences.

Table (5): Comparison between stability indices for the study group pre treatment and the control group at stability level six.

| Stability Index (SI) | | X±SD | t value | Sign. |
|----------------------|---------------|------------------|---------|--------|
| Overall | Study group | 11.73 ± 1.39 | 0.00 | P<0.05 |
| | Control group | 3.57 ± 1.17 | | |
| Anteroposterior | Study group | 9.6 ± 1.23 | 0.00 | P<0.05 |
| | Control group | 2.9 ± 1.15 | | |
| Mediolateral | Study group | 8.6 ± 1.1 | 0.00 | P<0.05 |
| | Control group | 2.4 ± 0.71 | | |

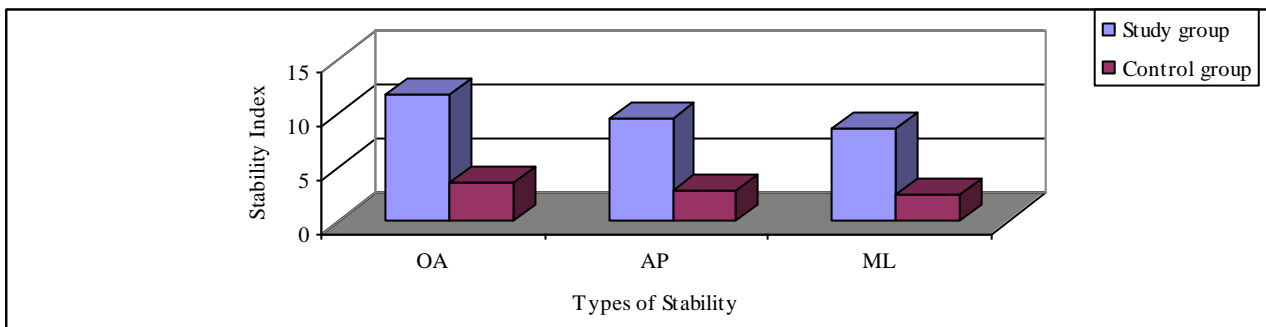


Fig. (4): Stability indices for the group two pre treatment and the normal control group at stability level six.

Overall Stability: The mean values of overall stability index pre treatment for the study group and the control group were 11.73 ± 1.39 and 3.57 ± 1.17 respectively showed high significant differences.

Anteroposterior Stability: The mean values of anteroposterior stability index pre treatment for the study group and the control group were

9.6 ± 1.23 and 2.9 ± 1.15 respectively, showed high significant differences.

Mediolateral Stability: The mean values of mediolateral pre treatment for the study group and the control group were 8.6 ± 1.1 and 2.4 ± 0.71 respectively, showed high significant differences.

Table (6): Comparison between stability indices for the study group post treatment and the control group at stability level eight.

| Stability Index (SI) | | X±SD | t value | Sign. |
|----------------------|---------------|-------------|---------|--------|
| Overall | Study group | 11.3 ± 1.44 | 0.00 | P<0.05 |
| | Control group | 3.35±1.12 | | |
| Anteroposterior | Study group | 9.28 ± 1.2 | 0.00 | P<0.05 |
| | Control group | 2.82±1.11 | | |
| Mediolateral | Study group | 8.29 ± 1.1 | 0.00 | P<0.05 |
| | Control group | 2.14 ±0.7 | | |

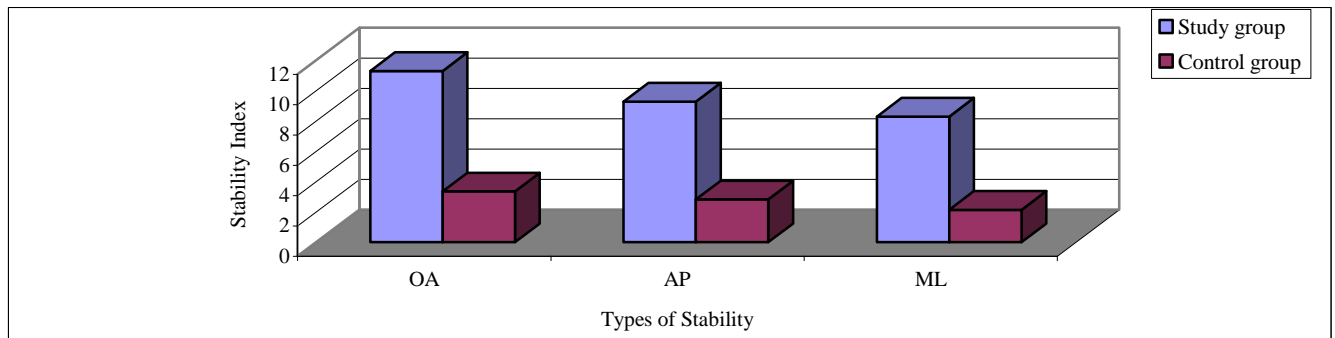


Fig. (5): Stability indices for the study group post treatment and the normal control group at stability level eight.

Overall Stability: The mean values of overall stability index post treatment for the study and control groups at stability level eight were 11.3 ± 1.44 and 3.3 ± 1.12 respectively, showed high significant differences.

Anteroposterior Stability: The mean values of the anteroposterior stability index post treatment for the study and control groups at stability level eight were 9.28±1.2 and

2.81±1.11 respectively, showed high significant differences.

Mediolateral Stability: The mean values of the mediolateral stability index post treatment for the study and the control groups at stability level eight were 8.29±1.1 and 2.1±0.73 respectively, showed high significant differences.

Table (7): Comparison between stability indices for the study group post treatment and the control group at stability level six.

| Stability Index (SI) | | X±SD | t value | Sign. |
|----------------------|---------------|--------------|---------|--------|
| Overall | Study group | 11.58 ± 1.32 | 0.00 | P<0.05 |
| | Control group | 3.57±1.17 | | |
| Anteroposterior | Study group | 9.52±1.22 | 0.00 | P<0.05 |
| | Control group | 2.96±1.15 | | |
| Mediolateral | Study group | 8.47±1.07 | 0.00 | P<0.05 |
| | Control group | 2.42±0.68 | | |

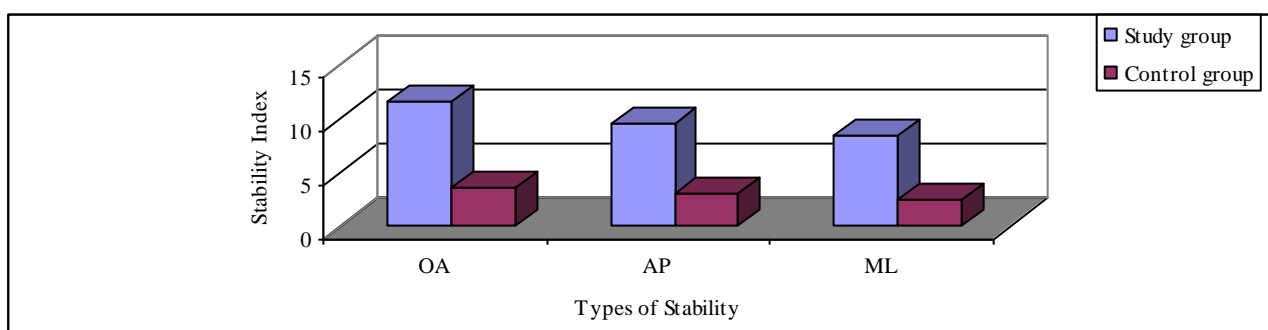


Fig. (6): Stability indices for the study group post treatment and the control group at stability level six.

Overall Stability: The mean values of overall stability post treatment for the study control groups at stability level six were 11.58 ± 1.32 and 3.56 ± 1.17 respectively, showed high significant differences.

Anteroposterior Stability: The mean values of the anteroposterior post treatment for the study and control groups at stability level six were 9.52 ± 1.2 and 2.59 ± 1.18 respectively, showed high significant differences.

Mediolateral Stability: The mean values of the mediolateral stability index post treatment for the study and control groups were 8.47 ± 1.07 and 2.42 ± 0.68 respectively, showed high significant differences.

DISCUSSION

No literature exposed to the effect of balance training program on the type one uncontrolled diabetes with neuropathy. Also, the effect of the type one uncontrolled diabetes with neuropathy on balance and postural control, in addition to the response to training program, so from this point the need of our study has been derived and established.

No Significant difference was reported when comparing the pre treatment mean values of all measured balance variables of the study group and the control group.

The pre treatment mean values of overall, anteroposterior and mediolateral stability indices of the dynamic balance test at the stability level eight, showed a significant increase in the study group in relation to the control group. This indicates that patient in the study group had a significant balance problem.

The elevated stability indices of the dynamic balance test at both stability levels eight and sixth in the pre treatment results of the study group could be attributed to muscles weakness especially the foot and ankle. In addition to limited joint mobility and sensory problem in the form of reduced somatosensation especially in the diabetic neuropathy patients and the difficulty in adapting sensory information to changing environment demand which might affect their abilities to maintain stability at different levels of unsteady surface⁴.

The significant disturbed standing balance seen in the type one uncontrolled

diabetes with neuropathy of the present study which was reported by elevated stability indices values might result from impaired sensation from cutaneous receptors in the planter aspect of the foot as a result of diabetic neuropathy. As this impairment was manifested by lack of rapid postural adjustments that are essential for dynamically stability standing. This come in agreement with Nichols, 2001⁸ who reported that, sensory problem can disrupt postural control by affecting the subject ability to adapt sensory inputs to changes in task and the environmental demands and also by preventing the development of accurate internal models of the body for the postural control.

Deficits in the standing postural control of the type one uncontrolled diabetes with neuropathy could be attributed also to severe muscle weakness specially of the foot and ankle muscles. As diabetes affect muscle strength and decrease power required to produce joint stability and adequate reactions⁴.

The findings of the current study could be confirmed by the study of Paolo, 2001¹¹ who examined postural responses in patients with somatosensory deficits due to peripheral neuropathy and showed significant delay in muscle response latencies in response to platform perturbations and in ability to modulate response amplitudes in relation to stimulus size. This electromyography study was done on the trunk (Paraspinal muscles) and leg muscles (gastrocnemius and hamstring).

The result of the collected data of this study before starting treatment in the study group showed that the mean values of the stability indices at stability level sixth was higher than stability level eight which indicated that balance was severely disrupted on stability level four. This could be attributed to inability of diabetic patients to activate distal muscles (ankle synergy) quickly enough to recover stability at the maximum disturbance produced at level four due to timing problem.

In general, diabetes affects muscle strength or the amount of force the muscle produce. The lower extremity strength can be reduced by as much as 40% between age 30

and 80 years⁹. Endurance which is the capacity of the muscle to contract continuously at sub maximal level decrease by diabetes and aging process which lead to smaller size of muscles and this reduction in muscle mass is greater in the lower extremity than the upper extremity. The muscle cell die and they are replaced by connective tissue and fat¹⁰.

So that deficits related to standing balance in the diabetic patients of the present study might be due to reduced sensation, distorted proprioception of the lower limb, decline in the muscle strength of the lower limb specially foot and ankle, decline in the muscle endurance that may affect their ability to maintain balance in addition to limited joint mobility.

In the present study, the type one uncontrolled diabetes with neuropathy were subjected to balance training program on the Biodex Stability System. The stability indices at stability level eight and sixth showed that there were no significantly difference pre and post treatment and this mean that no improvement in balance. The reasons for no improvement in balance, it could be related to that the systems which are responsible for balance control are somatosensory (touch and proprioception), visual and vestibular systems. This somatosensory system plays the biggest role in balance so losses of sense in the planter aspect of the sole of the foot associated with peripheral neuropathies can have a profound effect on balance. A person with sensory loss such as a bilateral leg peripheral neuropathy who does not receive normal sensory input from the sensory receptors in the feet and ankles will attempt to compensate by depending more on visual and vestibular input for balance. If there is significant sensory loss in the feet of the person will be unable to adjust easily to changes in the support surface during tasks such as walking on grass, uneven surfaces and even walking in shoes with soft soles¹⁵.

Another explanation for absence of improvement in balance, it could be related to the uncontrolled blood glucose which interferes with balance. Increased blood glucose increases vascular resistance and thereby reduces blood flow to the nerve. Endoneural hypoxia causes more capillary

damage, which in turn causes further hypoxia. This impairs axonal transport and reduces sodium potassium ATPase activity within the nerve, which reduces nerve conduction velocity. Abnormal thickening of endoneural blood vessel walls has been found in some patients with diabetic neuropathy, similar to the microangiopathic findings in diabetic renal disease, so the end result is no improvement of balance¹³.

Also, the increased blood glucose level increases non enzymatic glycosylation of collagen which cause abnormal cross linking and subsequent stiffness of the soft tissue which lead to limited ankle dorsiflexion and decrease the mobility of the first ray. In addition chronic edema of the lower limb can lead to fibrosis of the soft tissue, which compounds the local stiffness. It has been speculated that contracture of tendon achilles in uncontrolled blood sugar is by product of the glycosylation¹⁸.

The non significant improvement in the balance in the uncontrolled diabetes with neuropathy comes in agreement with Sacks et al. (2002)¹⁴ who found that the loss of balance and coordination was not due to age or diabetes but to nerve damage in the form of neuropathy. They compared the balance of four groups of subjects: 18 normal healthy controls between ages 20 and 40; 12 normal healthy controls between ages 70 and 79; 13 elderly diabetic subjects with no symptoms of neuropathy; and 14 elderly diabetic subjects with symptoms of neuropathy. The results showed that younger subjects had higher scores for all balance tests, while the elderly diabetic subjects with symptomatic neuropathy scored significantly worse on all balance tests. These findings provided strong support for the hypothesis that peripheral neuropathy profoundly affects lower extremity physical function independently of age and diabetes.

The present study is consistent with that of Skeleton D and Beyer, (2003)¹⁶ who found that diabetic peripheral neuropathy lead to problems throughout the body. One of these problem is neuropathy linked with loss of balance. Clinical neuropathy can develop within the first ten years after receiving diabetes diagnosis. About 60% of people with diabetes have some form of neuropathy. The

exact causes of neuropathy are unknown, several factors may contribute to the disorders, including high blood glucose which cause chemical changes in the nerves impairs the nerve ability to transmit signals. It also damage blood vessels that carry oxygen and nutrients to the nerves.

The finding of this study concerning no improvement in the balance in the uncontrolled diabetes with neuropathy are consistent with those of Dellon (2004)² who reported that, two metabolic changes occur in the peripheral nerves of patients with diabetes that render the nerve susceptible to chronic compression. The most critical is the increased water content within the nerve as the result of glucose being metabolized into sorbitol, which causes the nerve to have an increased volume. The second metabolic change is a decrease in the slow anterograde component of axoplasmic flow, which transports the lipoproteins necessary to maintain and rebuild the nerve. The peripheral nerves cross areas of anatomic narrowing, such as, the carpal tunnel at the wrist, the cubital tunnel at the elbow, fibular tunnel at the outside of the knee, or the tarsal tunnel at the ankle, which causes external pressure on the nerve, especially when its volume is already increased from the water content.

It can be concluded that performing balance training program can not improves balance in type one uncontrolled diabetes subjects with neuropathy.

REFERENCES

- 1- Aaron, L., Brarason, D. and Roy, F.: Diabetic autonomic neuropathy. *Diabetes Care*, 26 (2): 1553-1579, 2003.
- 2- Dellon, A.: Diabetic Neuropathy: Review of a Surgical Approach to Restore Sensation, Relieve Pain, and Prevent Ulceration and Amputation: *J foot and ankle International*, 25(10) : 749-755, 2004.
- 3- Gardener, T.: Practical implementation of an exercise in elderly diabetic persons. *Aging*, 30(1): 77-89, 2001.
- 4- Gutierrez, E., Helber, M., Dealva, D. and Richardson, J.: Diabetes mellitus. *Clin Biomech* 16(6): 522-528, 2001.
- 5- Hess, J., Woollacott, M. and Shivitz, N.: Aging process. *Clin-Exp-Res*.18(2): 107-115, 2006.
- 6- Khan, K., Ambroset, B., Donalson, M. and Mckay, H.: Physical activity to prevent falls in older people :time to intervene in high risk groups using falls as an outcome. *Br J Sports Med*. 23(4): 144-145, 2001.
- 7- Nakamura, H., Tsuchida, T. and Mano, Y.: The assessment of postural control in the elderly using the displacement of the center of pressure after forward platform translation. *J Electromyogr Kinesiol*, 11: 395-403, 2001.
- 8- Nichols, D.S.: The development of postural control. 4th "ed", Philadelphia, F.A.,Davis Co, 266-288, 2001.
- 9- Opara, E.: Oxidative stress, micronutrients, diabetes mellitus and its complications. *JR Soc Health* , 122(1): 28-34, 2002.
- 10- Osullivan, F.: Strategies to improve motor control and motor learning. *J Electromyogr Kinesiol*, 15: 195-203, 2002.
- 11- Paolo, P.: Autoimmune Diabetes Not requiring Insulin at diagnosis (Latent Auto immune Diabetes of the Adult). *Diabetes Care*, 24: 1460-1467, 2001.
- 12- Rozzi, S., Iephart, S., Sterner, R. and Kuligowski, I.: Balance training for persons with functionally unstable ankles. *JOSPT*, 29(8): 478-486, 1999.
- 13- Rubenstein, L., Kenny, R. and Koval, K.: Guidelines for prevention of falls in older persons. *J AM Geriat SOC* 4(49): 664-672, 2004.
- 14- Sacks, D.S., Bruns, D.E., Goldstein, D.E., Maclaren, N.K., McDonald, G.M. and Parroll, M.A.: Guide lines and recommendations for laboratory analysis and in the diagnosis and management of Diabetes Mellitus. *Clin Chem* 48: 436-472, 2002.
- 15- Shumway-Cook, A. and Wollacott, M.: Postural control in normal human. *Physiother*, 11(4): 32-33, 2001.
- 16- Skeleton, D. and Beyer, N.: Exercise and injury prevention in older people. *J Med Sci Sports*, (13)1: 77-85, 2003.
- 17- Zachary, J.: American Association of clinical Endocrinologists Meeting. *Diabetes Care*, 25(2): 1464-1471, 2002.
- 18- Zheng, Y., Choi, Y., Wong, K. and Chan, S.: Biomechanical assessment of planter foot tissue in diabetic patient using an ultrasound indentation system. *Ultrasound Med. Biol*. 26(5): 451-456, 2000.