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Abnormal viscoelastic behaviour of passive ankle joint movement in diabetic patients: an early or a late complication?

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Abstract *Aims/hypothesis:* The goal of the present study was to compare the range of motion and both the viscous and elastic components of passive ankle joint movement in short- and long-term diabetic patients with that of a control population. *Methods:* Thirty-four diabetic patients and 16 control subjects entered into the study. Patients with a history of over 15 years of diabetes were considered as a long-term diabetic group. In order to quantify the passive ankle joint movement, a device was designed to measure the dorsi- and plantar-flexion angle and the net moment at the ankle. Elastic behaviour was examined as the separate slope of regression lines (stiffness) of plantar and dorsal components in the loading moment–angle curve. It was also examined as the slope of the regression line in the final 10% of each component. Hysteresis, a characteristic of viscoelastic materials that indicates loss of energy during unloading, was corrected for range of motion and used to examine viscous behaviour of the ankle joint. *Results:* Total and plantar ranges of motion were significantly lower in long-term diabetic patients than in short-term diabetic and control groups ($p<0.05$). Plantar-flexion stiffness was significantly lower in short-term diabetic patients than in control subjects and long-term diabetic groups ($p<0.05$). Corrected hysteresis was significantly higher in long-term diabetic than in short-term diabetic and control ($p<0.05$) groups in the dorsal range of motion. *Conclusions/interpretation:* This study shows that both decreased plantar and total ankle joint ranges of motion, and increased viscous component of

passive ankle joint movement are among the late complications of diabetes.

Keywords Ankle · Diabetes · Hysteresis · Moment · Passive movement · Stiffness · Viscoelastic

Abbreviations ROM: range of motion

Introduction

Elastic materials deform instantaneously when they are subjected to externally applied loads and resume their original shapes instantly when the applied loads are removed. Therefore, the loading and unloading paths for an elastic material coincide, indicating no loss of energy. A different group of materials, including most biological soft tissues, is called viscoelastic. These exhibit gradual deformation and recovery when subjected to loading and unloading. Thus, an area between loading and unloading curves (hysteresis) forms, demonstrating loss of energy during unloading [1].

Several studies have demonstrated altered elastic behaviour of soft tissues in diabetes. One recent study on knee ligaments showed an abnormal viscous, rather than elastic, component of tissue response in response to dynamic loading [2], while another study reported no alteration in the elastic behaviour of passive ankle joint movement in diabetic patients [3].

It is known that the majority of diabetic foot ulcers have, in the large part, biomechanical causes. Altered gait patterns of diabetic patients may contribute to the development of an abnormally high pressure under the foot and most ulcers occur at the sites of high plantar pressure [4]. However, data on the biomechanical characteristics of ankle joint movement are scarce, except for on limited joint movement, which is a common abnormality [5].

Total moment at a joint may be divided into two components: (1) active, from the reduction of active forces in individual muscle crossing a joint into an equivalent resultant force and moment about some specified point; and (2) passive, from deformation of passive tissues such as

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tendons, ligaments, skin and inactive muscles. It is known that under conditions of decreased active tension, such as ankle dorsal and plantar flexor weakness and atrophy resulting from diabetic neuropathy, passive tension may make a greater contribution to the total tension than that found in healthy individuals [6]. So in the absence of appropriate active tension, it is possible that a considerable portion of the ankle moment during gait may come from passive structures.

The goal of the present study was to compare range of motion (ROM) and both viscous and elastic components of ankle joint moment in diabetic patients with those in a control population. In addition, we examined the probable association between these changes and the duration of diabetes.

Subjects, materials and methods

Subjects Diabetic patients were invited to participate in our study. Patients were excluded if they had any other neurological, endocrine, cardiac, pulmonary or musculoskeletal diseases or diabetic foot ulcer. Patients with a history of over 15 years of diabetes were classified as long-term diabetic. Subjects gave informed consent to their participation in the study. This study was approved by the Ethics Committee of Tehran University of Medical Sciences.

Equipment and procedures As a subject's ankle is moved passively by an examiner the movement will be opposed; this phenomenon is clinically denoted as resistance and is a result of stretching of internal passive structures. In order to quantify this phenomenon in the sagittal plane, a device (Fig. 1a) was designed to measure dorsi- and plantar-flexion angle and net passive moment at the ankle. Subjects were asked to lie supine and one of their legs was secured with a Velcro strap. The device was designed in such a way that their knee joints were positioned in 40° flexion, the most relaxed state of calf muscles [7]. The subject's foot was firmly fixed to a movable footplate and the lateral malleolus, considered as the axis of dorsi- and plantar-flexion of ankle, was aligned with the axis of rotation of the footplate. Subjects were instructed to remain as relaxed as possible during the test. A high-precision potentiometer measured the angular position of the footplate. The moment required to rotate the footplate was measured using a 250 N load cell (FORT 250; World Precision Instruments, Aston, UK). Data from the potentiometer and load cell were acquired at 50 Hz in real time. The footplate was rotated manually with an angular speed of about 0.2 rad/s, which is close to the speed of ankle rotation during everyday tasks [8]. The joint was exercised for three cycles. Mean values of the next three cycles were recorded and used for analysis.

Statistical analysis Four stiffness parameters and hysteresis were used to evaluate elastic and viscous ankle joint behaviour respectively (Fig. 1b). Data are expressed as means \pm SEM. Statistical analysis was performed using one-way

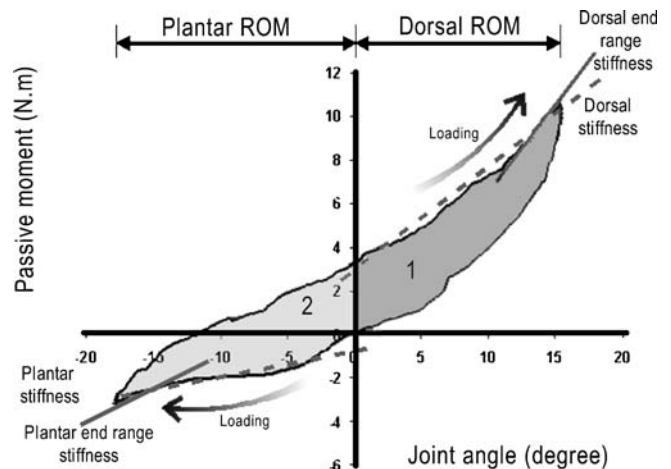
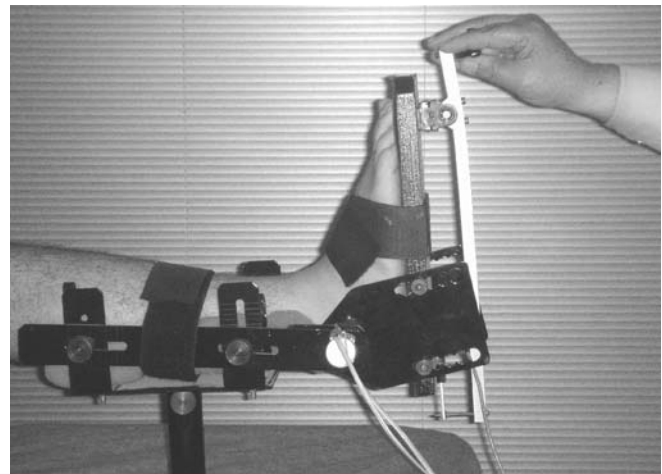


Fig. 1 a The device designed to measure passive moment-angle curve. b Passive moment-angle curve of a long-term diabetic patient. Stiffness parameters and hysteresis, which were used to evaluate elastic and viscous behaviour of the ankle joint, are demonstrated. Plantar- and dorsal-flexion stiffness were calculated as the slope of regression line of the plantar and dorsal components in the loading moment-angle curve (dashed lines). End-range plantar- and dorsal-flexion stiffness were calculated from the slope of regression line in the final 10% moment of the same curve (solid lines). Note that the loading moment-angle curve is the upper curve for dorsal-flexion and lower curve for plantar-flexion as shown by arrows. Hysteresis was calculated as the area between the loading and unloading curves (area 1 for dorsal, 2 for plantar) and normalised as its proportion to dorsal and plantar ranges of motion

ANOVA followed by Tukey's HSD as post hoc test. A value of $p < 0.05$ was considered significant.

Results

Patients' characteristics There was no significant difference between patients and control subjects in age, sex, height, weight and body mass index (Table 1).

Range of motion Total and plantar, but not dorsal, ROM were significantly lower in long-term diabetic patients than in short-term diabetic and control groups ($p < 0.05$, Table 1).

Table 1 Characteristics and comparison of viscoelastic indices in patients and control subjects

	Control	Long-term diabetes	Short-term diabetes
Age	50.5±2.2	55.9±1.8	49.3±2.2
Men/women	7/9	8/12	6/8
Height (cm)	168±2.4	165.8±1.5	165.4±3
Weight (kg)	75.4±4.2	74.8±2.5	70±2.8
BMI (kg/m ²)	26.5±1.2	27.1±0.7	25.6±0.9
Duration of diabetes (years)	–	19.4±1.7 ^a	7.4±1.5
Total range of motion (degree)	48.5±1.2	40.9±1.1 ^b	47.4±1.5
Plantar-flexion range of motion (degree)	35.6±0.4	29.0±0.6 ^b	33.1±0.9
Dorsal-flexion range of motion (degree)	12.9±0.4	11.9±0.5	14.3±0.8
Plantar-flexion stiffness (N m/degree)	1.41±0.10	1.30±0.12	0.96±0.13 ^c
Dorsal-flexion stiffness (N m/degree)	3.54±0.24	3.71±0.2	3.01±0.37
End-range plantar-flexion stiffness (N m/degree)	6.24±0.71	4.19±0.39	4.60±0.69
End-range dorsal-flexion stiffness (N m/degree)	7.89±0.82	7.40±1.04	8.07±1.01
Plantar normalised hysteresis (N m)	1.61±0.15	1.74±0.14	1.46±0.15
Dorsal normalised hysteresis (N m)	1.04±0.16	1.65±0.15 ^b	1.01±0.13

Values are means±SEM

^a $p<0.001$ compared to short-term diabetes group

^b $p<0.05$ compared to controls and short-term diabetes group

^c $p<0.05$ compared to controls and long-term diabetes group

Viscoelastic response Plantar-flexion stiffness was significantly lower in short-term than in long-term diabetic and control subjects ($p<0.05$). However, we did not find any statistically significant difference in other stiffness indices among the groups (Table 1). Although there was no significant difference in corrected hysteresis for the plantar component among the study groups (Table 1), corrected hysteresis was significantly higher in long-term diabetic patients than in short-term diabetic patients and control subjects ($p<0.05$) in the dorsal component.

Discussion

In our study, ankle ROM was significantly reduced in long-term diabetic patients, and this was mainly attributable to limited plantar-flexion. However, short-term diabetes was not associated with limited ankle joint mobility. Therefore, it seems that, in contrast to the limited mobility of small hand joints, reduced ankle ROM is a late complication of diabetes.

Total moment at a joint may be divided into active and passive components. In the absence of appropriate active tension and decreased ROM, the contribution of passive components to the joint movement will increase [3]. It is well known that the strength of calf muscles [6] and ROM [9] are reduced in diabetic patients. This suggests that a greater proportion of the ankle moment during gait may come from passive structures. Therefore we decided to examine passive ankle joint movement in diabetic patients.

Passive joint moment can be divided into elastic and viscous components. In the present study, plantar-flexion stiffness was significantly lower in short-term diabetic patients than in long-term diabetic and control groups. Salsich et al. have recently reported that, for ankle joint stiffness, there is no difference between diabetic patients and control subjects [3]. In that study, the mean duration of diabetes was about 18 years, close to our long-term diabetic group. However, this study did not evaluate changes in short-term diabetic patients. If we consider several reports that have

shown that soft tissues exposed to a hyperglycaemic environment are stiffer [10], both our study and that of Salsich et al. had unexpected findings. The latter [3], in an effort to find a solution to the contradiction between their study and reports of increased connective tissue stiffness in previous studies, proposed that structures containing collagen within the muscle tendon unit (perimysium, endomysium) contribute to passive stiffness, mostly at the end range of movement, and that within the physiological range of muscle length change, passive stiffness can be attributed to structures within the myofibril (e.g. structural proteins such as titin). In order to examine whether this hypothetical alteration can have any effect on physiological end-range elastic behaviour of the ankle joint, we compared the slope of the final 10% of the plantar and dorsal moment–angle curve, but found no significant difference. However, it should be emphasised that altered biomechanical behaviour of isolated soft tissues is not the only determinant of passive joint movement. In diabetic patients, atrophy of calf muscles has been reported [6], which in early stages of diabetes may reduce ankle joint passive stiffness. But, after long-term exposure to a hyperglycaemic environment, non-enzymatic glycation may cause soft tissue abnormalities, leading to increased joint stiffness compared to the level in control subjects. In the present study we have shown that long-term diabetes is associated with increased viscous, rather than elastic, components of passive ankle joint movement, which is in agreement with a study by Duquette et al. on knee ligaments of spontaneously diabetic rats [2].

Limited ankle joint movement has several clinical manifestations, including reduced shock absorption during the heel strike phase of gait and increased pressure on metatarsal heads as body weight is transferred to the forefoot [9]. It is known that altered gait patterns of diabetic patients may contribute to the development of abnormally high pressure under the foot, and most ulcers occur at the sites of high plantar pressure [5]. Based on the increased role of passive structures in total joint moment in diabetic patients, due to muscle weakness and atrophy [3], it is reasonable to assume that abnormal viscoelastic behaviour of passive ankle joint

movement may contribute to the changes in gait patterns and development of diabetic foot ulcers. However, no study has evaluated this aspect of ankle joint biomechanics in diabetic patients.

In summary, this study shows that decreased plantar and total ankle joint ROM and increased viscous components of passive ankle joint movement are among the late complications of diabetic patients. We also observed a significant reduction in plantar elastic component in short-term, but not long-term, diabetic patients. The possible effects of altered viscoelastic behaviour of passive ankle joint movement on gait patterns, development of high plantar pressure and foot ulcer in diabetic patients need to be evaluated in future.

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References

- Nordin M, Frankel VH (2001) Basic biomechanics of the musculoskeletal system. Lippincott Williams & Wilkins
- Duquette JJ, Grigg P, Hoffman AH (1996) The effect of diabetes on the viscoelastic properties of rat knee ligaments. *J Biomech Eng* 118:557–564
- Salsich GB, Mueller MJ, Sahrman SA (2000) Passive ankle stiffness in subjects with diabetes and peripheral neuropathy versus an age-matched comparison group. *Phys Ther* 80:352–362
- Bowker JH, Pfeifer MA (2001) Levin and O’Neal’s the diabetic foot. Mosby
- Rosenbloom AL, Silverstein JH (1996) Connective tissue and joint disease in diabetes mellitus. *Endocrinol Metab Clin N Am* 25:473–483
- Andersen H, Gadeberg PC, Brock B, Jakobsen J (1997) Muscular atrophy in diabetic neuropathy: a stereological magnetic resonance imaging study. *Diabetologia* 40:1062–1069
- Hicks JH (1953) The mechanics of the foot 1: the joints. *J Anat* 87:345–357
- Esteki A (2002) Passive dynamic flexion moment at the human ankle joint. *Pejouhandeh* 7:15–20
- Simmons RW, Richardson C, Deutsch K (1997) Limited joint mobility of the ankle in diabetic patients with cutaneous sensory deficit. *Diabetes Res Clin Pract* 37:137–143
- Menzel EJ, Reihnsner R (1991) Alterations of biochemical and biomechanical properties of rat tail tendons caused by non-enzymatic glycation and their inhibition by dibasic amino acids arginine and lysine. *Diabetologia* 34:12–16