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INFLUENCE OF REDUCED PLANTAR SENSATION AND MUSCLE WEAKNESS ON PLANTAR PRESSURE DISTRIBUTION

Berrak YİĞİT, PT

NEUROLOGICAL REHABILITATION
MASTER OF SCIENCES THESIS

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Abbreviations

BMI : Body mass index

Kg : Kilogram

m : Meter

^{0}**C** : Degree Celsius

MTHs: Metatarsal heads

SUMMARY

Influence of Reduced Plantar Sensation and Muscle Weakness on Plantar Pressure Distribution

Berrak YİĞİT, PT

Purpose: To determine the isolated contribution of reduced plantar sensation and of muscle weakness by simulating them on healthy subjects was aimed.

Material and Methods: The study group consisted of 18 subjects (20 - 44 years old). Subjects walked in 4 different conditions (condition 1 was normal condition walking in normal gait pattern, condition 2 was reduced muscle strength walking by wearing waistcoat contains 40% of subject's body weight, condition 3 was reduced plantar sensation walking after 12 minutes ice immersion approach and condition 4 was combination of conditions 2 and 3) with their preferred walking speed. Plantar surface of the foot divided into ten different areas (two areas for hindfoot, two areas for midfoot, three areas for MTHs (1, 2, 3-4-5) and three areas for toes (1, 2, 3-4-5).

Results: There was significant main effect of all areas, of the waistcoat condition and of iced condition (F=66.11, F=57.27, F=14.39 prospectively). There was insignificant main effect of combined condition. There were significant interaction effects between the areas and the waistcoat condition (F=5.71) and between areas and the iced condition (F=7.28). Peak pressure on areas 5, 6 and 7 were higher than other areas under iced condition. Peak pressure on areas 3 and 6 were higher than other areas under waistcoat condition.

Conclusion: Reduced plantar sensation had more effect on plantar pressure than muscle weakness whereas both of them had no effect on healthy subjects. This result may project to diabetic patients with reduced plantar sensation and desensitization training should be considered to manage that problem.

Key words: Reduced plantar sensation, muscle weakness, diabetic foot

Preface

Diabetes is a common condition affecting patients in the developed and developing countries. Complications affecting the lower limb are among the most common manifestations of diabetes, and those precipitated by neuropathy include ulceration, infection, and even amputation. Diabetic foot ulceration is a significant cause of morbidity. In which case, it can lead to prolonged hospital stays, which is evidenced by the fact that approximately 20% of hospitalizations related to diabetes involve diabetic foot ulceration.[1] Some observations show that foot ulceration occurs in 15% of diabetic patients and those patients are 15–46 times more likely to have an amputation than non-diabetic patients.[2] In addition, about one in five hospitalizations among diabetic patients are directly related to foot ulceration. The costs of treating get a higher level because of ulcerations.[3] Therefore, identification of the risk factors of foot ulceration can help prevent ulceration and decrease the prevalence numbers.

The major reason why ulceration is a big problem is the factors that contribute to their development have a larger variety[4]. The factors which cause ulceration are existence of neuropathy[5], peripheral vascular diseases[6], generalized limitations of joint mobility, overweight[7], structural foot deformities[7,8] and soft tissue damages[1,9]. Because the person can no longer notice when his/her feet become injured, people with diabetic neuropathy are more likely to develop foot problems such as skin lesions and ulcers that may become infected[10,11].

Diabetic neuropathy is one of many complications associated with diabetes, with approximately 60 percent of diabetics having some form of nerve damage[2]. It is a progressive disease that can cause loss of sensation, as well as pain and weakness, in the feet and sometimes in the hands[2,12,13]. Diabetic neuropathy may affect both afferent and efferent pathways of the lower extremity[1,5,14]. Motor nerves (efferent pathways) send impulses from the brain and spinal cord to all of the muscles in the body. This allows people to do activities like walking or moving the fingers to pick something up. Motor nerve damage can lead to muscle weakness, atrophy[15], difficulty walking or moving the arms, cramps and spasms. Sensory nerves send messages from the muscles back to the spinal cord and the brain (afferent

pathways). Special sensors in the skin and deep inside the body help people identify the shape of an object, the temperature of an object or if it's standing still or in motion. Sensory nerve damage often results in tingling, numbness, pain, and extreme sensitivity to touch.[13,16] After these nerve damages (with severe neuropathy) gait ataxia may develop[15]. Neuropathy causes problems in feet by disrupting nerves, both reducing sensation of pain and causing problems with the way of walk. Such problems can damage feet in the different ways; reduced sensation prevents recognizing foot which has been injured; failed muscular control causes walk in an abnormal pattern. By changing the pattern and distorting the position of the feet, neuropathy can increase pressure-related injuries, such as calluses or blisters, and deformities which modify the gait.[17]

The loss of protective sensation stops the patient from being warned that the skin is being injured and may result in skin loss, blisters and ulcers. The loss of protective sensation causes to anhidrosis and dry fissured skin also contributes foot deformity, which leads to abnormal pressure distribution in the foot when standing or walking.[18] Therefore patients with diabetic neuropathy have different walking patterns. These changes on gait may lead to a differentiation of plantar pressures distribution.[1,5]

Previous studies showed that patients with diabetic peripheral neuropathy have obvious muscle weakness[19,20,21]. Motor neuropathy leads to atrophic changes in the foot muscles which result in foot deformity and reduced joint mobility[6]. This atrophy occurs on intrinsic muscles of the foot, thus upsetting the delicate balance between flexors and extensors of the toes. This failed balance results in foot deformities such as hammer toes, claw toes, prominent metatarsal heads, and pes cavus. Unfortunately, structural deformities are common sites of abnormally high pressure, and repetitive pressure which causes to tissue breakdown.[2,11,22] The changes in plantar pressure distribution are probably related to diabetic foot ulceration. Therefore, identifying the changes in plantar pressure distribution is the most important step in reducing the rate of foot ulceration.[6]

Especially, to identify problem based rehabilitation approach; reduced plantar sensation and muscle weakness should have an isolated observation. However, diabetic patients have

not only these factors but also some more complications.[23,24] Then simulation studies on healthy subjects must be conducted.

Therefore, the aim of this study was to determine the isolated contribution of reduced plantar sensation under the foot and of muscle weakness. To do so, these factors are separately simulated in healthy subjects. To simulate lost sensibility, plantar sensation will be reduced by submerging feet in ice water and to simulate muscle weakness, subjects will wear a weight waist. Our hypothesis was that reduced plantar sensation, muscle weakness, and also both of them result in a substantially modified plantar pressure distribution.

Introduction

Diabetes Mellitus

Definition

Diabetes mellitus is a metabolic disorder which also is an incurable disease characterized by high levels of blood sugar[25]. It can be caused by little insulin (a hormone produced by the pancreas to regulate blood sugar), resistance to insulin, or both[26]. Except acute complications, through long-term effects of metabolic anomalies and damages on eyes, kidneys, nerves and blood vessels, diabetes may be named as a syndrome[25].

Classification

There are 4 basic types of diabetes mellitus[27]:

I- Type I Diabetes: This type is characterized by the lack of insulin production. It can be named as insulin-dependent or childhood onset.

a- Caused by immune system

b- Idiopathic

II- Type II Diabetes: This type is result from the body's ineffective use of insulin. It can be named as non-insulin-dependent or adult onset.

III- Other Specific Types

a- Genetic defects on β-cell function

b- Genetic defects on insulin activity

c- Pancreas diseases

d- Endocrinopathies

e- Caused by drugs and chemical materials

f- Infections

g- Rare forms cased by immune system

IV- Gestational Diabetes: This is hyperglycemia which is diagnosed during pregnancy.

Impaired Glucose Tolerance and Impaired Fasting Glycaemia are intermediate conditions in the transition between normality and diabetes. People with impaired glucose tolerance or impaired fasting glycaemia are at high risk of progressing to type II diabetes, although this is not inevitable.

Epidemiology

Prevalence of diabetes mellitus is increasing depending on socio-economic factors, changes in physical activity level and obesity. In the twenty years ahead, according to World Health Organization estimating scores, number of diabetic people will be approximately 200 million more. For all over the world, prevalence of diabetes mellitus was 2.8% in 2000 whereas in 2030 it will be 4.4%.[28] Naturally, diabetes mellitus complications will also increase[29]. The study of Turkey Diabetes Epidemiology Group in 2002 showed that prevalence of DM was 7.2%; impaired glucose tolerance was 6.7%[30]. In 2000 426.000 people in the Netherlands were diagnosed with diabetes and in 2030 these figures may increase to 720.000. In 2003 the diabetes prevalence in the Netherlands was 600.000 with an incidence of 4.5 per 1000 inhabitants[31].

Complications

Diabetes has acute and chronic complications[12,25,26]. These are as follow:

Acute Complications*:*
1- Diabetic cetoacidose
2- Hyperosmolar nonketotic coma
3- Hypoglycemia

Chronic Complications*:*
1- Diabetic microangiopathy
    a- Diabetic retinopathy
    b- Diabetic nephropathy
2- Diabetic macroangiopathy (atherosclerosis)

a- Coronary artery disease

b- Cerebro-vascular disease

c- Peripheral artery disease

3- Diabetic Neuropathy

a- Symmetric peripheral polyneuropathy

b- Autonomic neuropathy

c- Asymmetric mononeuropathy

d- Radiculopathy

e- Diabetic amyotrophy

4- Infections

5- Diabetic foot

6- Dermopathies

7- Depression

In our simulation study, we used diabetes mellitus as a model and we focused on diabetic neuropathy and diabetic foot ulcers. Therefore, from chronic complications, diabetic neuropathy and diabetic foot ulcers are explained.

Diabetic Neuropathy

Diabetic Neuropathy occurs in 25% of people with diabetes mellitus ten years after they are diagnosed and 50% of patients who have had the disease for twenty years[20,32]. Different types of diabetic neuropathy have different causes. Researchers are studying the effect of glucose on nerves to find out exactly how prolonged exposure to high glucose causes neuropathy. However there are not known exactly yet. Common idea is that nerve damage is composed of a combination of factors:[2]

- Metabolic factors, such as high blood glucose
- Long duration of diabetes, possibly low levels of insulin
- Abnormal blood fat levels
- Neurovascular factors, leading to damage to the blood vessels that carry oxygen and nutrients to the nerves
- Autoimmune factors that cause inflammation in nerves

- Mechanical injury to nerves, such as carpal tunnel syndrome, inherited traits that increase susceptibility to nerve disease
- Lifestyle factors such as smoking or alcohol use.

Diabetic neuropathy can be classified as peripheral, autonomic, proximal, and focal. Each affects different parts of the body in 4 different ways:

1. *Peripheral neuropathy* can be seen frequently bilaterally on lower extremities. Stocking-glove sensory loss is the main symptom. Symptoms include numbness, insensitivity, a tingling, burning, or prickling sensation, sharp pains or cramps, extreme sensitivity to touch, even a light touch, loss of balance and coordination. Loss of sensation may cause neuropathic foot ulcers, loss of proprioseption may cause gait disorders and also especially may cause Charcot deformity. These symptoms are often worse at night.[2,20,25,32] Mostly pain disappears within six months or one year[12].

2. *Autonomic neuropathy* results in changes in digestion, bowel and bladder function, sexual response, and perspiration. It can also affect the nerves that serve the heart and control blood pressure.[2]

3. *Proximal neuropathy*, sometimes called lumbosacral plexus neuropathy, femoral neuropathy, or diabetic amyotrophy, starts with pain in the thighs, hips, buttocks, or legs. In type II diabetes, it can be seen more often. Main effect of proximal neuropathy is weakness in the legs.[13]

4. *Focal neuropathy* results in the sudden weakness of one nerve, or a group of nerves, causing muscle weakness or pain. Any part of the body may be affected. Main symptom is pain. Pain can occur on the different parts of the body such as in the front of a thigh, in the chest, stomach or inside the foot.[2,13,33]

Loss of Sensation

Diabetic people have significant deficits in tactile sensitivity, vibration sense, lower-limb proprioception, kinesthesia and muscular function[20,32,34]. The loss of sensation associated

with diabetic peripheral neuropathy results in impaired balance, altered gait patterns, and increased risk of falling[34,35]. People with diabetic peripheral neuropathy display greater postural sway when standing[35] and many gait studies have reported characteristic changes in walking patterns associated with diabetic peripheral neuropathy, including decreased power generation at the ankle, decreased knee joint flexion, and decreased ground reaction forces[32,34,35].

Muscles and cutaneous receptors transport sensory input to the central nervous system. Central nervous system uses this input to produce motor patterns for posture and gait. The receptors are also important for feedback to understand information about loading, joint kinematics and pressure distribution on the plantar surface of the foot.[6,7,13,32]

The loss of protective sensation results in repetitive trauma from an area of high pressure[6,7]. Pressure can not be noticed; therefore blisters and sores may appear on insensible areas of the foot. If foot injuries are not treated properly, the infection may be contaminated to the bone, and then an amputation may be needed. Some researchers reported that half of all such amputations are preventable if minor problems treated in time.[2,7,33,36]

Muscle Weakness

Motor nerves damage, dysfunctions and atrophy are the results of the motor component of the neuropathy[5,20,37,38]. Muscle weakness is related to neurogenic atrophy which caused by axonal degeneration of the motor fibers[39].

Motor neuropathy leads to weakness and atrophy[6] in the intrinsic muscles of the foot, this resulted in disrupting the delicate balance between flexors and extensors of the toes. Thus weakness leads to the development of hammer toes, claw toes, prominent metatarsal heads, and the collapse of the midfoot and pes cavus. These structural deformities cause abnormal high and repetitive pressure, and this could result in tissue breakdown.[2,11,22]

Diabetic Foot Ulcers

Foot ulceration is a major complication of diabetes[6,7,8,40]. There are many risk factors which is related to diabetic foot ulceration, including peripheral neuropathy[6,11,40,41,42,43,44],

peripheral vascular disease, other microvascular complications, previous ulceration, infections[45], dermal lesions[6,11,41,42,43], biomechanical dysfunctions, high plantar pressures[7,44,46], limited joint mobility[9], elderly patient, patient who lives alone[6,11,41,42,43]. Peripheral neuropathy is the most important component of reasons for foot ulceration, as well as foot deformities and trauma[1,6,9,11,41,42,43,44].

High plantar pressure and sensory deficit is mainly responsible for ulceration[10,11]. Neuropathy and foot tissue under continuous stress also speed up development of foot ulcers. Continuous pressure results in callus formation. Callus formation increases foot pressure at least 30%. Callus formation and foot ulcers can be seen mostly under metatarsal heads and under hallux.[8,47]

High foot pressure is an important risk factor for foot complications. Foot pressures were higher cause of neuropathy, deformity, callus, and previous foot ulceration or amputation.[48]

Plantar Pressure

The plantar surface of the foot is exposed to ground reaction forces in case of weight loading, like walking, standing. The pressure (or the stress) is the same as normalized force (force/area). The ground reaction forces measure is as much as the weight of the body if the person is standing on foot. Each foot receives half of the body weight evenly distributed over the plantar surface. The body weight mainly applies a vertical force and a small horizontal one.[49,50]

The pressure is focused over the heel and very few affect the forefoot. When a person walks, the situation changes; now, the stress is mainly on the foot rather than the heel. One of the reasons is that the entire weight is accumulated on a single foot for a certain time. Only 22% of the time when walking, both feet step at the same time.[49,50] Another reason is about rollover of the foot which is a component of the stance phase of gait[51]. In normal gait, firstly, foot revolves around the heel, then revolves around ankle joint, metatarsal head and finally around the hallux. As mentioned above, different foot parts contact with the ground during

stance phase at different durations. Therefore, during anteriorly progression of ground reaction forces, support surface also differs in size and location. Heel makes contact at the first approximately 64% of the stance phase. The forefoot and toes make contact at the last 59% of the stance phase. If such is the case, heel and forefoot are in contact together during the middle 23 % of the stance phase.[49] The last reason is that ground reaction forces diversify in magnitude. There is a double- hump curve; first peak represents landing on the heel and second peak represents pushing off with forefoot at the end of stance phase. These peaks are approximately 1.2 time's body weight with the preferred walking speed.[49,50]

Normally, heel and forefoot get higher peak pressure than midfoot. During normal gait pattern, the highest peak pressure occurs on the medial metatarsophalangeal area and on the hallux.[7] It shows that areas which have higher pressure may change depending on body weight and walking speed. Normal gait pattern may also have biomechanical alteration because of the morphological nature, physical activity, age and the presence of some disease such as loss of sensation and muscle weakness caused by diabetic peripheral neuropathy.[19]

Influences of Loss of Sensation and Muscle Weakness on Plantar Pressure

Gait can be assumed as a result of a harmonic correlation between neural and muscular coordinated action which need skeletal function. Failure in this harmonic relationship may results in changes in the locomotor pattern.[20] Petrofsky et al. reported that diabetic patients are at risk for falling even if they do not have any loss of sensation. However they have muscle weakness which breaks down the harmony of gait.[37] This may also explain why there was a change in pressure distribution in the feet in diabetic patients, even in the lack of apparent sensory impairment[37]. Motor dysfunctions such as muscle weakness which results in deficits in foot joint mobility and orthopedic deformities significantly changed peak plantar pressure.[19]

Reduced plantar sensation leads to a modified gait which is marked with more attentive contact to the ground[24]. Eils et al. showed that reduced plantar sensation effects all over the lower limb during gait[24]. Somatosensory impairment studies reported that plantar sensation has a great influence on gait and posture[23,24] and also high pressure distribution[52]. The

cutaneous receptors get information from muscles, tendons, ligaments and joint capsules. The somatosensory system also gets some information from the same structures. It is well known that this information is useful to control balance and gait.[13,24] Reduced plantar sensation modified rollover pattern of the foot. This modification leads to alteration in plantar pressure and gait.[23]

Material and Methods

Subjects

The study was started with 20 healthy subjects (mean age 25.2±6.5 years old) but completed with 18 subjects. During test procedure, two of the subjects was dropped out due to ice intolerance. Diabetic patients have not only muscle weakness and loss of sensation but also some more complications.[23,24] Then, to identify separate effects of muscle weakness and loss of sensation on plantar pressure must be conducted on healthy subjects.

The subjects were included if their age over 18 years, normal walking pattern; visual inspection showed no obvious abnormalities, normal foot pressure distribution; evaluated using visual inspection of foot pressures with the help of a pressure plate.The subjects were excluded if they had neuro-musculoskeletal and orthopedics disorders, pes planus, cardiopulmonary diseases and body mass index (BMI) > 25.

Procedures

The study was made at Maastricht University, Maastricht-The Netherlands, in Health Sciences Faculty, Movement Science Department Laboratory. It was approved by the Local Ethics Committee of Maastricht University Hospital, Maastricht, The Netherlands (See appendix A for report in Dutch and appendix B for report in English). The subjects were instructed about the experimental protocol and their questions were answered and they signed an informed consent form (See appendix C).

Equipment

Testing procedure was performed with the commercially available EMED-AT system (EMED AT-2, type 1377/2, 50 Hz, Novel GmBh; Munich, Germany). The sensor platform accommodates 2736 capacitance transducers with data acquisition at a rate of 50 Hz (figure 1).

Figure 1: Pressure platform

Patients were asked to walk across a wooden walkway measured 12 m long. The plantar pressure platform was placed in the middle of the walkway (figure 2).

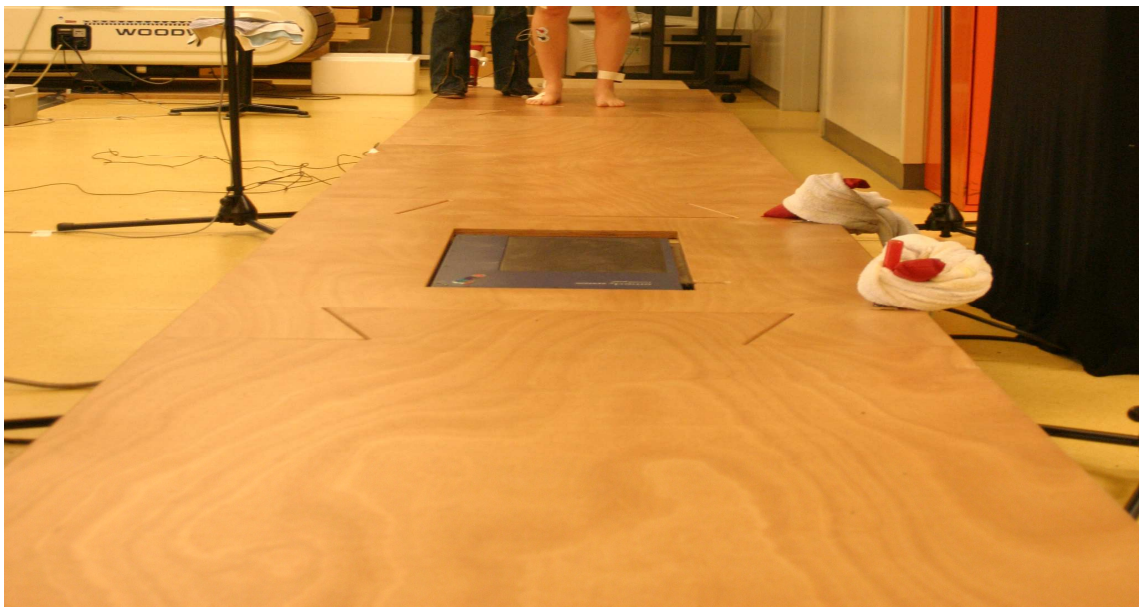


Figure 2: Wooden walkway

The walking velocity was measured by two infrared sensor poles placed two meters apart. The first sensor and a digital timer were positioned in front of the pressure platform. The second

infrared sensor was positioned behind the platform and stopped the timer if it was interrupted. The sensors were set at shoulder level to prevent movements of arms or legs that may trigger the timer too early as the subject passes through them.

The temperature of the plantar area was measured at the heel, the forefoot and the hallux with a K type thermocouple (Testo 901). When the sensor of this device touched the skin, temperature was immediately measured (figure 3).

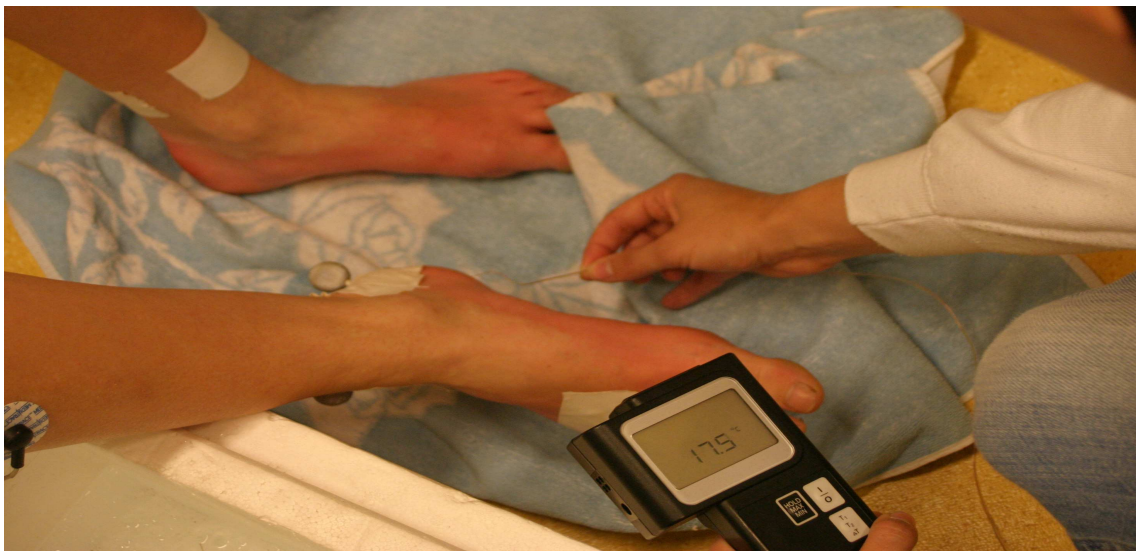


Figure 3: Temperature measurement

Pre-testing Procedure

Body weight, body height and age were measured and recorded on a data form (See appendix D). After this process, the subjects were asked to stand on the pressure plate with their right foot to measure if he/she has pes planus or not.

The preferred walking velocity was determined during this pre-testing period after several trials that were done to familiarize with the equipment and to find the right starting point. This precaution was important to eliminate purposely targeting the plantar pressure platform. This velocity equaled their preferred walking velocity. A small range of variance was allowed (±5%) to make it practicable for the subjects to perform the tests. The subjects

walked five times at their normal preferred walking velocity to increase the reliability of measurements and to calculate an average value for walking velocity.

Testing Procedure

Normal Condition

All the results were compared with the normal walking pattern. Thus, the subjects firstly walked without iced feet and without wearing waistcoat in a normal walking pattern. Every walking condition was done five times.

Reduced Plantar Sensation

The plantar sensation was reduced by iced immersion technique. Foot immerged to the level of the medial malleolus in ice water (0-1˚C) for twelve minutes (figure 4). The subject sat on a chair with feet on a metal grid that was placed in a polystyrene container. The feet was placed on a grid that was surrounded by iced water so plantar surface could be cool efficiently.

The feet dried off at the end of twelve minutes. The temperature of the plantar area was measured at the heel, the forefoot and the hallux. Then the subject walked over the walkway. The measurement was successful when the velocity was within the determined range and if the foot was placed just centre of the pressure platform. The subject had to walk without targeting the platform to avoid changing the gait pattern. He/she had to pass trough it simultaneously.
After two measurements the participant was placed his/her feet back into the ice water for one minute to sustain the effect of the cooling.

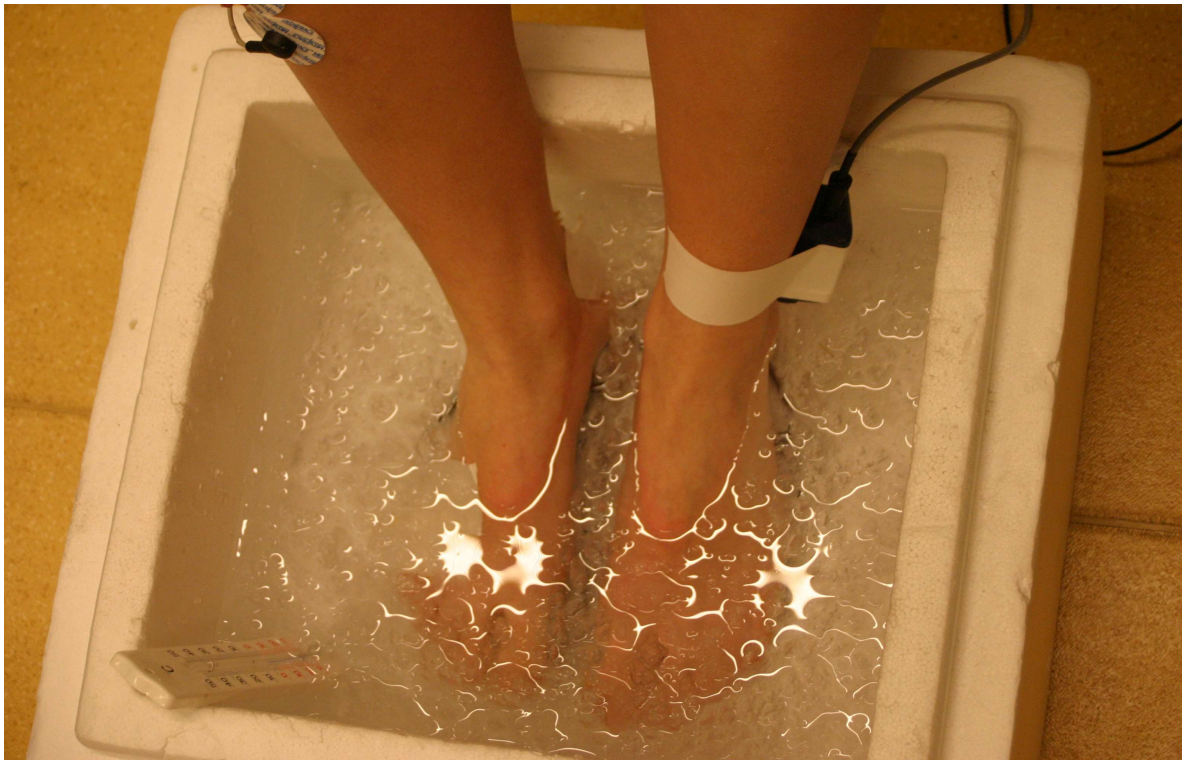


Figure 4: Ice immersion

Reduced Muscle Strength

Muscle weakness was simulated by wearing a waistcoat containing 40% of the body weight (figure 3.2.5.1). The waistcoat can increase the body weight, without seriously disturbing the participant's centre of mass, because the added weight is placed very close to the body.

Then, the subject walked through the walkway. The measurement was successful when the velocity was within the determined range and if the foot was placed just centre of the pressure platform. The subject had to walk without targeting the platform to avoid changing the gait pattern. He/she had to pass trough it simultaneously.

Figure 3.2.5.1: Waistcoat back

Combination of Factors

The third walking condition was a combination of the conditions mentioned above.

Data Analysis

Measurements of total force, peak pressure and the area of contact were displayed on the monitor immediately after the walk, together with a color-coded pressure pattern. This information was recorded and analyzed by the computer.

Peak pressure the primary indicator for studying the risks of skin break down[8,19] which was the main parameter for this study.

Data was analyzed by commercial software Novel Database Pro, version 11.38. Parameters were determined in 10 different areas after using the PRC mask for subdividing the foot: two areas for the heel and the midfoot, three areas for the forefoot, and one each for the hallux, second toe, and lateral toes. Parameters for every area were calculated in novel projects with novel automask.

Statistical Analysis

Individual means of five repeated trials was calculated. For statistical analysis, SPSS 10.0 for Windows (Standard Version, 1989-1999) statistics program was used and significance level was accepted if $p < 0.05$. A series of 1-way repeated-measures analyses of variance (ANOVAs) was used to evaluate within subjects effects of different conditions (iced, waistcoat and combined) and normal walking. After a significant main effect for conditions was determined, a series of pairwise comparisons was performed to assess for differences between the four groups. The repeated-measures independent factors used were the ten different areas, waistcoat condition and iced condition.

Results

Subjects

Two of the subjects could not participate because of the ice intoleration. Subject's characteristics can be seen on table 8.1.1.

Table 1: Subject's Characteristics

Gender (n)		Age (years)		Body Weight (kg)		Body Height (m)		BMI (kg/m^2)	
Male	Female	Mean	SD	Mean	SD	Mean	SD	Mean	SD
6	12	25,72	6,52	71,44	11,77	1,8	0,1	22,03	2,5

SD: Standard deviation

The mean of preferred walking velocity was 1.4±0.1 second.

Immerging the feet in the ice water for twelve minutes reduced the skin temperature approximately 12^0C under heel, hallux and forefoot (table 8.1.2). Table 8.1.2 represents the mean temperature of the skin during both the iced and combined conditions.

Table 2: Mean Temperatures for ice and combined conditions.

	Heel	Hallux	Forefoot
Mean (^{0}C)	13,01	11,71	12,28
SD	1,7	1,7	1,3

SD: Standard deviation

Peak Pressure

For this section, area representations are as follow:

Area 1 …… medial hindfoot

Area 2 …… lateral hindfoot

Area 3 …… medial midfoot

Area 4 …… lateral midfoot

Area 5 …… first metatarsal head

Area 6 …… second metatarsal head

Area 7 …… 3-4-5 metatarsal heads

Area 8 …… hallux

Area 9 …… second toe

Area 10 …….. toes 3-4-5

For this section condition representations are as follow:

Condition 1 …….. normal walking

Condition 2 …….. walking with waistcoat

Condition 3 ……. iced condition

Condition 4 ……. combination of ice and waistcoat condition

Table 3: Mean peak pressure values of medial hindfoot for all conditions.

Area 1	Mean (kPa)	SD	n
Condition 1	487.55	109.28	18
Condition 2	412.53	70.65	18
Condition 3	449.69	99.34	18
Condition 4	382.56	108.97	18

SD: Standard deviation

n: number of subjects

Table 4: Mean peak pressure values of lateral hindfoot for all conditions.

Area 2	Mean (kPa)	SD	n
Condition 1	466.30	90.75	18
Condition 2	405.64	75.18	18
Condition 3	455.44	88.12	18
Condition 4	352.80	61.36	18

Table 5: Mean peak pressure values of medial midfoot for all conditions

Area 3	Mean (kPa)	SD	n
Condition 1	92.11	40.43	18
Condition 2	93.02	35.27	18
Condition 3	76.46	27.47	18
Condition 4	85.82	28.94	18

Table 6: Mean peak pressure values of lateral midfoot for all conditions.

Area 4	Mean (kPa)	SD	n
Condition 1	105.61	30.49	18
Condition 2	93.49	22.13	18
Condition 3	91.00	23.96	18
Condition 4	88.26	24.42	18

Table 7: Mean peak pressure values of the first metatarsal head for all conditions.

Area 5	Mean (kPa)	SD	n
Condition 1	310.56	126.51	18
Condition 2	278.84	90.66	18
Condition 3	341.07	99.01	18
Condition 4	339.29	160.07	18

Table 8: Mean peak pressure values of the second metatarsal head for all conditions.

Area 6	Mean (kPa)	SD	n
Condition 1	370.90	84.31	18
Condition 2	395.76	79.49	18
Condition 3	395.07	89.31	18
Condition 4	389.62	94.83	18

Table 9: Mean peak pressure values of 3-4-5 metatarsal heads for all conditions.

Area 7	Mean (kPa)	SD	n
Condition 1	332.11	63.26	18
Condition 2	283.02	65.83	18
Condition 3	338.61	81.25	18
Condition 4	315.37	87.33	18

Table 10: Mean peak pressure values of hallux for all conditions.

Area 8	Mean (kPa)	SD	n
Condition 1	392.61	152.72	18
Condition 2	363.60	113.23	18
Condition 3	379.89	180.40	18
Condition 4	338.95	146.46	18

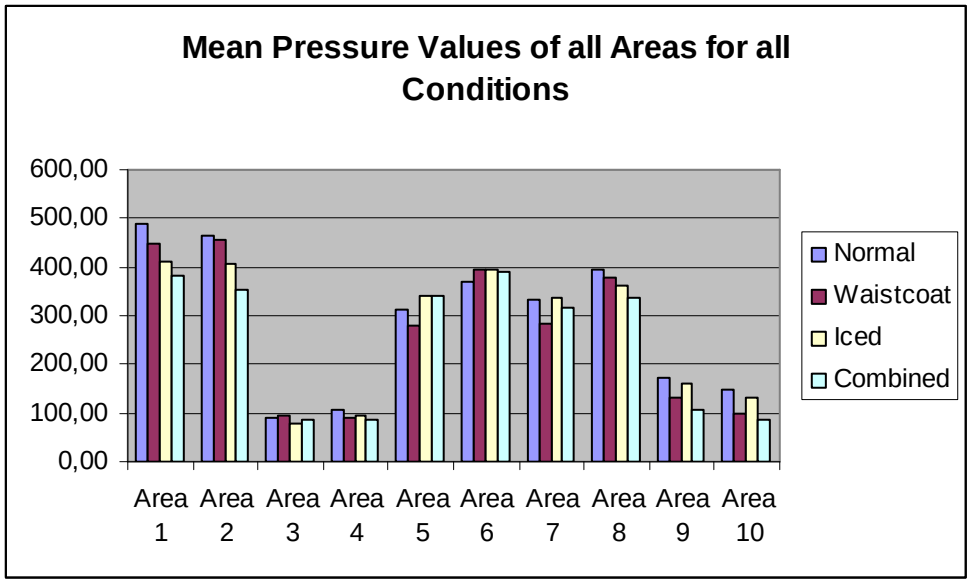
Table 11: Mean peak pressure values of the second toe for all conditions.

Area 9	Mean (kPa)	SD	n
Condition 1	173.00	64.40	18
Condition 2	159.95	65.75	18
Condition 3	132.67	50.53	18
Condition 4	106.46	37.38	18

Table 12: Mean peak pressure values of toes 3-4-5 for all conditions.

Area 10	Mean (kPa)	SD	n
Condition 1	147.81	52.56	18
Condition 2	131.77	25.08	18
Condition 3	98.78	40.50	18
Condition 4	86.78	36.43	18

The tables cited above are descriptive tables for mean peak pressure about all areas for all conditions. According to table 5 and table 8, peak pressure on area 3 and 6 were higher under waistcoat condition than other areas. Table 7, table 8 and table 9 showed that peak pressure on area 5, 6, 7 were higher under iced condition than other areas. Table 7 and table 8 indicated that peak pressure on area 5 and 6 were higher under combined condition than other areas. The graph which cited below showed a brief summary of mean pressure values of all areas under all conditions.



Graph: Mean peak pressure values of all areas for four different conditions

Effects and Interactions of Conditions

There was a significant main effect of all areas (F=66.11). There were also significant main effects of the waistcoat condition (F=57.27) and of iced condition (F=14.39. There were significant interaction effects between the areas and the waistcoat condition (F=5.71) and between areas and the iced condition (F=7.28). There were not significant interactions between waistcoat and iced conditions ($p>0.05$) and between areas, waistcoat and iced conditions ($p>0.05$). This indicates that waistcoat and iced conditions have different effects apart from the area.

Table 13 indicates interaction between 10 areas and waistcoat condition. Under column waist, 1 means no added mass and 2 means added mass (for areas see section 8.2, page 20). This table showed that area 3 and 6 had higher peak pressures than other areas.

Table 14 represents interaction between 10 areas and iced condition. Under column ice, 1 means no iced and 2 means iced (for areas see section 8.2, page 20).This table pointed out that peak pressures were higher under area 5, 6 and area 7 than other areas.

Table 13: Interaction between waistcoat condition and areas

				95% Confidence Interval	
AREA	WAIST	Mean (kPa)	Std. Error	Lower Bound	Upper Bound
1	1	468,62	22,84	420,43	516,81
	2	397,55	18,97	357,53	437,56
2	1	460,87	19,37	420,01	501,73
	2	379,22	14,46	348,71	409,73
3	1	84,29	7,34	68,80	99,77
	2	89,42	7,34	73,93	104,91
4	1	98,31	5,85	85,97	110,64
	2	90,88	4,98	80,38	101,37
5	1	325,82	24,98	273,11	378,53
	2	309,06	27,25	251,57	366,55
6	1	382,99	19,81	341,19	424,78
	2	392,69	18,40	353,87	431,50
7	1	335,36	16,20	301,19	369,53
	2	299,19	17,09	263,13	335,25
8	1	386,25	37,65	306,81	465,68
	2	351,27	28,73	290,66	411,89
9	1	152,83	12,63	126,19	179,48
	2	133,21	10,05	112,01	154,40
10	1	123,29	9,97	102,25	144,34
	2	109,27	6,29	96,01	122,54

Table 14: Interaction between iced condition and areas

				95% Confidence Interval	
AREA	ICE	Mean (kPa)	Std. Error	Lower Bound	Upper Bound
1	1	450,04	20,36	407,08	493,00
	2	416,12	21,79	370,16	462,09
2	1	435,97	17,37	399,33	472,61
	2	404,12	16,60	369,10	439,14
3	1	92,56	8,62	74,37	110,76
	2	81,14	6,30	67,86	94,43
4	1	99,55	5,78	87,35	111,75
	2	89,63	5,47	78,08	101,18
5	1	294,70	23,82	244,44	344,96
	2	340,18	28,57	279,90	400,46
6	1	383,33	16,83	347,82	418,84
	2	392,34	20,77	348,52	436,17
7	1	307,56	13,25	279,61	335,52
	2	326,99	18,76	287,42	366,56
8	1	378,10	28,23	318,55	437,66
	2	359,42	35,60	284,32	434,52
9	1	166,48	13,78	137,40	195,55
	2	119,56	9,54	99,44	139,69
10	1	139,79	8,30	122,27	157,31
	2	92,78	8,76	74,30	111,25

Discussion

The goal of this study is to isolate individual factors which result from diabetic neuropathy so as to determine the influences of these factors on plantar pressure. It was supposed that muscle weakness and loss of sensation affect plantar pressure. This case can result in increasing foot ulceration risks. It is hypothesized that each of these factors and/or both of them would generate a modified plantar pressure under different areas.

A simulation method is used to isolate and manipulate muscle weakness and reduced plantar sensation. The advantage of this method is that the investigation of one factor can be isolated at a time; something that can not be seen in diabetic neuropathic patients. Unfortunately, this method which is very complex has also a disadvantage which is important during making comments for results. Therefore many environmental factors which influenced the results occurred during the experiment such as unfamiliar walking patterns for subjects and uncomfortable feeling of ice.

Muscle weakness was simulated with the waistcoat containing 40% of the subject's body weight. Muscle weakness can be described as an imbalance between the load on the muscle and the load capacity of the muscle. Muscle weakness can be simulated by changing the relation between load and load capacity. In diabetic neuropathic patients, load capacity is normally decreased because of the reduced muscle mass. The load was changed for the subjects of this study. It is supposed that increasing the load/load capacity ratio by increasing the load will have a similar effect on gait as increasing this ratio by decreasing the load capacity; this is the case which diabetic neuropathic patients have. Previous studies found out that neuropathic patients have loss of muscle strength in a range from 15% to 40%.[38,39,53] Reduced load capacity occurs at the presence of muscle weakness. Increasing the load with 40% also changes the load capacity and may therefore simulate muscle weakness in healthy people at maximum load level.

It is assumed that the subjects had normal muscle strength (100%) and with the loaded waistcoat it was 140%. The load increased and the load/load capacity ratio is therefore 1.4. In neuropathic patients this ratio is estimated to be between 1.2 and 1.7 according to previous studies[38,39,53]. Thus weight should be adequate to simulate muscle weakness. This load was tolerable for subjects.

BMI was also increased 40% by adding 40% body weight. Patients with type II diabetes are generally overweight. To be overweight is a slow process and an adaptation period is developing during this time. Muscles have to be stronger to be able to carry this added load. However, in neuropathic patients, the strength of the leg muscles is reduced instead of been strong. Thus gait pattern changed to adapt this new weight. The subjects in this study became overweight in a few seconds. This results in a different and unfamiliar gait pattern. Furthermore, in diabetic neuropathic patients, reduction of leg muscles strength differ by the type of neuropathy (distal or proximal neuropathy may occur). Therefore, it is supposed that strength of all leg muscles is reduced more equally in this study. This case generated a difference between the subjects of this study and the diabetic patients.

Another difference could be appeared between the subjects and diabetic patients but it was eliminated. Some research studies on diabetic gait showed that walking velocity is different for diabetic patients and controls. Diabetic patients have lower walking velocity[32,37,54,55]. To eliminate this situation, the subjects walked during all the different conditions at their constant velocity.

There are many simulation studies which were reduced plantar sensation in different ways[23,24,56,52,57]. One of the methods to reduce plantar sensation is the use of an anaesthetizing crème (EMLA) that can be applied to the plantar area[56]. However this is a very expensive and time wasting method. Therefore in our simulation, we used ice immersion approach. Other studies used ice immerging approach which varies from some points[23,24,52,57]. In the study of Eils et al., subjects placed one foot without a grid in ice water so that only the plantar part of the foot was submerged[23]. Without submerging all part of the foot and with one foot, it was changed the walking pattern in a different way and it was not a direct simulation of a diabetic neuropathic foot. In another study, Eils et al. used the same procedure with a grid. However,

they again used one foot[24]. Nurse et al. did not use submerging. They used shaved ice which was sealed inside a plastic bag and both feet placed on the surface of the bag[52]. Taylor et al. used ice water with 2^0C and proceed for 30 minutes[57]. In this study, a combination of important points from the previous simulation studies used to reduce plantar sensation. We used directly submerging both feet in ice water with a grid. It is therefore assumed that the plantar sensitivity was mainly reduced during our experiment.

The cooling method of this study was 12 min ice immerging approach which affects the mechanoreceptors in the plantar surface of the foot. According to Nurse et al., pressure sensation is difficult to alter through skin temperature, but when the temperature decreases ten degrees Celsius the sensation is severely limited[52]. A study executed by Eils et al. showed that no additional loss of sensitivity occurred after ten minutes of cooling in water of 0^0C[23]. This can be explained by the 'Hunting reaction', which states that cooling leads to a vasodilatation reaction. The dilatation results in a small rise in temperature, which can cause the stop in the reduction of pressure sensitivity[23]. It is obviously noticed that the mean temperature is above ten degrees Celsius that were stated by Nurse et al.[52] This may mean that the sensitivity was not reduced enough. However, the mean skin temperature did not differ more than 1-2^0C mentioned by Nurse et al.[52] Also, the subjects walked in a strange and unfamiliar pattern after immersion.

Although influence of the loss of sensation on plantar pressure is well described in the literature[23,24,52,56,57], influence of muscle strength on plantar pressure could not be seen. According to the relevant literature, this study may be the first muscle weakness simulation to find out the influence on plantar pressure. The main result from muscle weakness simulation was that under medial midfoot and second metatarsal head pressure was higher than normal walking condition.

In reality, muscle weakness does not a direct effect on plantar pressure but in directly it has a great effect on plantar pressure. Firstly, it is changing walking pattern. Reduced muscle strength at the ankle joint is the main cause that leads to the changes in neuropathic walking pattern[5]. There is a loss of neuromuscular foot control. In addition decreased balance between plantar and dorsal flexors and between invertors and evertors mostly failed the rise of forefoot

both in the sagittal and frontal plane.[5] This case results in the foot deformity. Foot deformities are important contributory risk factors and predictive of foot ulceration, possibly by predisposing the skin to high pressure at the site of the foot deformity[4,11,18,58,59]. Van Schie et al. reported that toe deformities have been suggested to be related to muscle weakness in the toe flexors[11]. Similarly to Van Schie study, Cavanagh et al. showed that claw toe deformity has been associated with high metatarsal head pressures based on the flexors and extensors weakness[22]. These structural changes lead to increased supinatory moments in neuropathic feet with an increased pressure under 2nd, 4th and 5th metatarsal heads. Such structural alterations could contribute to ulceration in the neuropathic or neuroischaemic foot where the contact plantar surface may be even more reduced as a result of additional neurological or vascular pathology. This could explain that, in such patients, the area under metatarsal heads is the most frequently affected part of the sole by plantar ulcers.[46,60] Our subjects did not have any deformity as they are healthy. However, our findings showed the similar results with the neuropathic subjects. Under second metatarsal head, high pressure occurred. Then it may be though that muscle weakness simulation was mainly similar with real conditions. Medial midfoot also had high pressure due to intrinsic muscle weakness. However, this case needs more investigations.

In the present study, the result after reduced plantar sensation was that under all metatarsal heads, peak pressures were higher than other areas. Other areas had lower pressure than normal condition.

This finding about metatarsal heads supports results of other studies that have used an ice immersion approach[24,52]. It is well documented that cold application leads to a decrease of nerve conduction velocity and also influences skin receptors[23,24,52]. Therefore, in the present study, there is low peak pressure under the heel and the toes. The heel has a role in the initial ground contact while the hallux has a role in the push-off phase. It appears that subjects made a more carefully touch down and push-off to protect the foot in order to reduced feedback. Furthermore, it has to take account that chronic loss of sensation, as in diabetic neuropathy, may produce a compensatory mechanism which used inputs from other sensory systems such as visual or vestibular systems.[2,23,24,34,37,55,56] Thus an acute loss of sensation which has done in our experiment may be compensated by increasing pressure at the metatarsal heads and

decreasing pressure at the periphery[52]. However, our subjects who walked in normal condition have areas of relatively high pressure under some metatarsal heads. Those with normal sensation do not ulcerate because their sensory feedback prevents durable immoderate loading.[36]

On the other hand our findings about high pressure under metatarsal heads and reduced pressure under heel and toes are in accordance with recently performed plantar pressure distribution measurements[24,52]. Parallel to Nurse et al. and Eils et al. studies, our results showed significantly high peak pressure under second metatarsal head and reduced peak pressure under the heel, toes and the forefoot at ground contact and push-off after ice immersion of the foot. Our findings are also parallel to studies which have neuropathic patients as a subject. They reported that one of the most common sites of ulceration in diabetic neuropathy patients is at the metatarsal heads, an area that is frequently shown to have higher pressures[1,8,40,61,62,63] and a reduction of peak pressure under the heel and the toes was also reported in neuropathic patients in comparison to normal patients[23,24].

In conclusion, muscle weakness simulation results in high pressure under second metatarsal head and medial midfoot, and reduced plantar sensation simulation results in high pressure under all metatarsal heads. These results indicate that reduced plantar sensation has more influence on plantar pressure than muscle weakness on healthy subjects. On the other hand, reduced plantar sensation incidence is also higher in diabetic subjects. That is why further investigations may be done on diabetic patients with reduced plantar sensation. Furthermore desensitization training may be considered for management of diabetic foot.

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11. Appendix

Appendix A: The Ethical Committee Report (in Dutch)

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Aan Dr. H.H.C.M. Savelberg
Faculteit Gezondheidswetenschappen
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UNS 50 kamer 2230

afdeling MED.ETHISCHE COMMISSIE
uw kenmerk MECsecretariaat@ctcm.azm.nl
ons kenmerk 043 - 387 6009
doorkiesnummer MEC 06-3-024.4/pl
datum 24 mei 2006

Geachte dr. Savelberg,

Hierbij bericht ik u dat de Medisch Ethische Commissie op grond van artikel 2.2.a van de WMO op 19 mei 2006 een positief oordeel heeft uitgebracht gelet op het gestelde onder artikel 3 van de WMO met betrekking tot het door u ingediende onderzoeksvoorstel genaamd, **"Onderzoek naar de factoren die leiden tot een diabetisch looppatroon; een simulatie experiment"** (MEC 06-3-024).

De commissie heeft de volgende stukken in haar toetsing betrokken:

- protocol met versiedatum 8 mei 2006;
- proefpersonen- c.q. patiënteninformatie met versiedatum 8 mei 2006 (inclusief de verzekeringsinformatie over de wijze waarop de verzekering ex artikel 7 lid 1 WMO is geregeld);
- toestemmingsformulier met versiedatum 19 mei 2006;
- ABR-formulier met versiedatum 1 mei 2006;
- het curriculum vitae van de onafhankelijke arts;
- de advertentie/wervingstekst met versiedatum 19 mei 2006.

De commissie ontvangt nog graag bericht van de start- en einddatum van genoemde studie. Het positieve oordeel verliest zijn geldigheid, ook als met het uitvoeren van het onderzoek niet is begonnen, binnen een jaar nadat dit besluit is genomen. De commissie ziet graag ieder jaar tijdig, vóór afloop van de geldigheid, informatie tegemoet over de voortgang van de studie onder vermelding van het aantal proefpersonen dat aan het onderzoek heeft deelgenomen.

In een aan deze brief aangehechte bijlage zijn nadere, wettelijke, eisen vermeld waaraan de onderzoeker zal dienen te voldoen of welke door de commissie onder de aandacht worden gebracht.

Met vriendelijke groet,

namens de Medisch Ethische Commissie,

mr. R.C.W. van Gils,
ambtelijk secretaris

dr. C.E.M. de Die-Smulders,
voorzitter

Cc: De heer M.P.W. Lamberti BA, directeur Clinical Trial Center Maastricht
CCMO
UM, BMB/F&BI, verzekeringen MBB 4-6

De volgende leden hebben deelgenomen aan de besluitvorming inzake bovengenoemde studie:

Prof. dr. T. Gorgels, cardioloog (vice-voorzitter)
Dr. J. Offermans, gynaecoloog
Mevr. A. Hilton, verpleegkundige
Dr. P. Portegijs, universitair docent huisartsgeneeskunde
Dr. L. Stolk, ziekenhuisapotheker/klinisch farmacoloog
Prof. dr. M. Prins, methodoloog
Dr. A. v.d. Arend, filosoof
Mevr. H. Tjon-a-Fat, behartiger van de belangen van proefpersonen

Appendix B: The Ethical Committee Report (in English translation by Hans Savelberg)

This is a short translation by Hans Savelberg of the approval letter of the Medical Ethical Committee of AZM, number MEC 06-3-024.4/pl

Dear Dr. Savelberg,

Based on article 2.2.a of the WMO (law on human research), the Medical Ethical Committee has passed a positive judgement with respect to the research proposal that was submitted by you, titled: “onderzoek naar de factoren die leiden tot een diabetisch looppatroon; een simulatie experiment” (an investigation in factors that cause a diabetic gait pattern; a simulation experiment) (MEC 06-3-024).

For her judgement the committee used the following documents:

- research protocol dd may 8, 2006
- information for subjects dd may 8, 2006 (including information with respect to subject insurance)
- informed consent form dd 19 may 2006
- ABR-form, dd May 1, 2006
- Additional information, dd May 1, 2006
- Curriculum vitae of an independent medical doctor
- Recruitment form dd May 19, 2006

The committee likes to be informed about the exact start and end dates of the study. The positive judgement is valid for 1 year. The committee wants to be informed every year about the progress of the study and the number of subjects included.

Sincerely,

List of members of the MEC that contributed to this decision

Appendix C. Informed Consent Form:

INFORMED CONSENT

for participation in the experimental study:

Investigating factors that contribute to a diabetic gait pattern; a simulation

I have been informed about the study by the researchers, and I know what I will have to do during the study. I have been given opportunity to ask questions on the study. Moreover I had time to consider my participation in this study. I can decide at any moment to cancel my participation.

I agree in participating in this study, moreover I agree to use my medical and research data in the way it has been explained to me.

I like not to be / to be **(please, select an option)** informed about the results of the study.

I do not / I want to invited **(please, select an option)** for participation in forthcoming studies.

Name :
Date of birth :

Signature : Date :

As responsible research, I declare that the above mentioned subject has been verbally informed about this study.

Name :
Position :

Signature : Date :

Appendix D: Data Form

DATA FORM

Date: …-…-2006

Subject Name:	
Code:	
Age:	
Body Weight (Kg):	
Body Length (m):	

Preferred walking velocity (time in seconds):					Remarks
1)	2)	3)	4)	5)	
Mean -5%:					
Mean:					
Mean +5%:					

Condition	Run 1 (s)	Run 2 (s)	Run 3 (s)	Run 4 (s)	Run 5 (s)	Remarks:
Normal (1)						
Waistcoat (2)						
Iced (3)						
Combined (4)						

Fill out what measurement failed with an "X" or was correct and saved with a "V"																										
Condition	Run 1						Run 2						Run 3					Run 4					Run 5					Remarks:
Normal																											
waistcoat																											
Iced																											
Combined																											