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Plantar Pressure Measurements and Geometric Analysis of Patients With and Without Morton's Neuroma

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Abstract

Background: The purpose of this research was to see if there were any differences in peak pressure, contact time, pressure-time integrals, and geometric variables such as forefoot width, foot length, coefficient of spreading, and arch index between subjects with Morton's neuroma (MN) and control subjects.

Methods: Dynamic peak plantar pressure, contact time, pressure-time integral, and geometric data were extracted using the EMED-X platform in 52 subjects with MN and 31 control subjects. Differences in peak pressure, contact time, pressure-time integral, and geometric data between participants with and those without MN were determined using independent-samples *t* tests. There were no significant differences in age, weight, height, and body mass index between patients with MN and control subjects.

Results: There were no significant differences in the peak pressures of all masked areas and pressure-time integrals under metatarsal 2 to 4 heads between patients with MN and control subjects. In addition, no significant differences were observed between patients with MN and control subjects in geometric measurements of forefoot length, width, coefficient of spreading, foot progression angle, and arch index.

Conclusion: No relationship was found in this study between peak pressure, contact time, and pressure-time integral under the metatarsal heads, forefoot width, foot length, coefficient of spreading, and foot progression angle in a symptomatic MN group compared with a control group. The need to perform osteotomies to treat MN not associated with other lesser metatarsal phalangeal joint pathologies is questionable.

Level of Evidence: Level III, Case-Control Study

Keywords: Morton's neuroma, pressure, width, metatarsalgia

Morton's neuroma (MN) is a common cause of metatarsalgia affecting the second and third intermetatarsal spaces. It is more prevalent in women than men, with the hospital admission rate for MN treatment highest in women aged 50 to 55 years.[31] Specific symptoms of MN include shooting pain, numbness, and/or tingling in the second, third, and fourth digits, burning sensation, cramping, and a feeling of "walking on a lump in the ball of the foot."[1] This is aggravated by walking in high-heeled shoes with a narrow toe box. MN is clinically diagnosed by Mulder's click, which has 98% sensitivity in diagnosing MN.[35] Magnetic resonance imaging and ultrasound are equally effective in diagnosing MN,[7] but a recent systematic review suggested that ultrasound can more accurately diagnose MN than can magnetic resonance imaging, with 90% sensitivity and 88% specificity.[42]

Although there is a debate over the exact etiology of MN, chronic trauma is a theory implicated by many authors.[1,3,6,17,20] Abnormal metatarsal parabola,[33] biomechanical factors such as pronation[1,17] and or supination,[10,36] and equinus deformity[4,37] have been variously proposed to increase pressure in the forefoot and cause repeated trauma to plantar intermetatarsal nerves.[26] Kim et al,[26] in an anatomic study, refuted the entrapment theory proposed by Gauthier[14] and postulated that the condition was caused by pinching of the common digital nerve by the adjacent

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metatarsal heads and metatarsophalangeal joints during walking. MN is a form of metatarsalgia, and it has been hypothesized to result from repeated loading of the metatarsal heads.[22] On the basis of the aforementioned theories, the increased pressure in the forefoot and under the metatarsal heads may traumatize the common digital nerve, leading to fibrosis and pathologic changes.

MN is often managed conservatively with the use of metatarsal pads,[17,19,27] which are thought to lead to pain reduction by decreasing pressure on the nerve by widening of the forefoot.[27] Recently, Bauer et al[6] and Catani et al[10] advocated percutaneous metatarsal osteotomy to decompress the affected nerve as an operative treatment for MN. They theorized that symptoms of MN can be managed by addressing the "hyperpressure" of the affected metatarsal head. However, they did not investigate plantar pressure measurements before and after performing metatarsal osteotomy to confirm a change in pressure. Surprisingly, no case-control studies reported in the literature have investigated forefoot pressure in individuals with MN compared with an asymptomatic control group.

Other factors such as "wideness" or "splay foot" have been suggested as contributing to the etiology of MN.[15,24,40] The normal contour of the forefoot and the presence of the transverse arch across the metatarsals is an important mechanism by which shock absorption within the forefoot can occur during gait.[15,23,38] Pathologic conditions, such as "splay foot," "anterior flat foot," and "collapsed metatarsal arch," may increase the pressure in the forefoot and cause metatarsalgia.[38] Additionally, "splay foot" may also produce compressive forces of the forefoot when wearing shoes, leading to irritation of the affected nerve. On the contrary, narrowness of the forefoot, which can lead to closer proximity of the metatarsal heads impinging on the nerve, has been mentioned as an etiology of MN.[34] Park et al[34] did not find any significant differences in forefoot width, intermetatarsal angle, and metatarsal distance between radiographs of subjects with MN (n = 84) and age- and sex-matched control subjects (n = 84). However, their study was based on weight-bearing radiographs that mimic midstance and not during propulsion, when the forefoot is dynamically under greatest stress. Although forefoot width is often cited as a contributing cause of MN, no studies in the literature have examined this factor dynamically.

Dynamic barefoot pressure data can be collected using the EMED-X capacitance transducer matrix platform (Novel, Munich, Germany), which has been shown to be a reliable tool for measuring plantar forefoot pressures and foot geometry.[2,16] The purpose of this research was to first test the hypothesis that forefoot peak pressure, contact time, and/or pressure-time integrals would be greater under the affected metatarsal heads in patients diagnosed with MN compared with control subjects. We also investigated if there were any significant differences in geometric measurements, such as forefoot width, foot length, coefficient of spreading, foot progression angle, and arch index, in patients diagnosed with MN compared with control subjects.

Table 1. Demographic Description of Patients With Morton's Neuroma and Control Subjects.[a]

Variable	Patients With Morton's Neuroma (n = 52)	Control Subjects (n = 31)	*P*
Age, y	52 ± 14	49 ± 10	.28
Weight, kg	76 ± 19	71 ± 16	.22
Height, m	1.67 ± 0.07	1.64 ± 0.10	.22
Body mass index, kg/m^2	27 ± 6	26 ± 4	.31
Sex (female:male)	39:13	19:12	
Feet studied	61	62	
Foot (right:left)	30:31	31:31	

[a] Data are expressed as mean ± SD or as numbers.

Methods

Approval was obtained from the University of Western Australia human research ethics committee for this study (approval RA/4/1/2543). Eighty-three participants consisting of 52 subjects with MN and 31 control subjects were recruited from the University of Western Australia Podiatry Clinic from 2012 to 2014. Control subjects consisted of 12 men and 19 women, and the MN group consisted of 13 men and 39 women, who were all from the University of Western Australia staff. The demographic information of the participants is provided in Table 1. Patients with MN and control subjects were recruited using a university circular e-mail advertisement. All subjects were given a patient information sheet, and written consent was obtained to participate in the study. Inclusion criteria for subjects with MN were a minimum 6-month history of neuroma symptoms and a clinically demonstrated painful Mulder's click with ultrasound confirmation of MN. All subjects with MN were treated conservatively prior to the study and were pain free on the data collection day. Ultrasound diagnosis of MN was made by an experienced musculoskeletal radiologist and assessed on both transverse and longitudinal axes as an abnormal ovoid hypoechoic thickening corresponding to the location of maximum tenderness.[11] Each subject with MN was clinically examined by the corresponding author as well as an experienced musculoskeletal radiologist to rule out any other source of pain such as capsulitis and lesser metatarsophalangeal joint instability such as plantar plate pathology. The inclusion criterion for control subjects was a negative history of MN or neuroma-like pain in the forefoot. Exclusion criteria for both neuroma and control groups were any history of surgery to the lower extremity;

Table 2. Interspaces Affected in Subjects With Morton's Neuroma.

Interspace	Right Foot	Left Foot	Both Feet	Total
2/3	11	4	6	21 (40.4%)
3/4	8	10	1	19 (36.5%)
Both	2	8	2	12 (23.1%)
Total	21 (40.4%)	22 (42.3%)	9 (17.3%)	52 (100%)

any proximal nerve entrapment at the level of the ankle, knee, hip or back; any history of significant trauma to the forefoot area; rigid toe deformities; and an inability to ambulate without pain.

There were no significant differences in age, weight, height, and body mass index between patients with MN and control subjects (Table 1). The numbers of affected interspaces with MN are summarized in Table 2.

Measurements

Dynamic peak plantar pressure, contact time, pressure-time integral, and geometric data were extracted using the EMED-X platform, which was set flush to the floor within a 10-m walkway. The EMED-X platform is composed of 6080 capacitive sensors within a sensing area of 475 × 320 mm (sensor resolution, 4 sensors/cm^2) and had a pressure range of 10 to 1,270 kPa, accuracy of ±5%, and hysteresis < 3%.[32] The sampling frequency was 100 Hz. A 2-step approach at a self-selected speed was used for all trials, which has been demonstrated previously to be as reliable as the midgait approach.[8] Participants stood on the platform barefoot and took 2 steps forward to determine the starting position. At the starting position, subjects were instructed to strike the platform on their second step. Subjects were instructed to use their usual gait while looking straight ahead and to avoid targeting the platform. Subjects practiced walking across the platform until they were comfortable with the procedure. Subjects performed 5 trials with the right and left feet, until a total of 10 successful steps were recorded. A trial was successful when only 1 foot contacted the platform, contact was made on the second step, and participants did not target the platform. Trials not meeting these criteria were excluded.

EMED-generated data were analyzed using Novel Database medical software program version 15.2.3. All measurements were calculated from the maximum pressure picture of the step during gait, which corresponds to the contact or push-off phase of gait. The maximum pressure picture is a color-coded image of the foot that represents the maximum pressure value recorded by each sensor. The foot was divided into 10 regions (masks) using EMED Automask software. Masks for heel, midfoot, first to fifth metatarsal heads, hallux, second toe, and third to fifth toes (Figure 1) were used for analysis. For each mask, peak pressure (kPa) and contact time (ms) were measured. The pressure-time integrals (kPa · ms) under metatarsals 2 to 4 were measured to assess the cumulative effect of both pressure and time on MN formation. For each subject, means of the 5 measurements were used for statistical analyses. Five geometric measurements that included forefoot width (cm), foot length (cm), coefficient of spreading (forefoot width/foot length), foot progression angle (the angle between the axis of the foot and line of progression), and arch index (midfoot area divided by total foot area) were automatically calculated using the software algorithms for each trial.

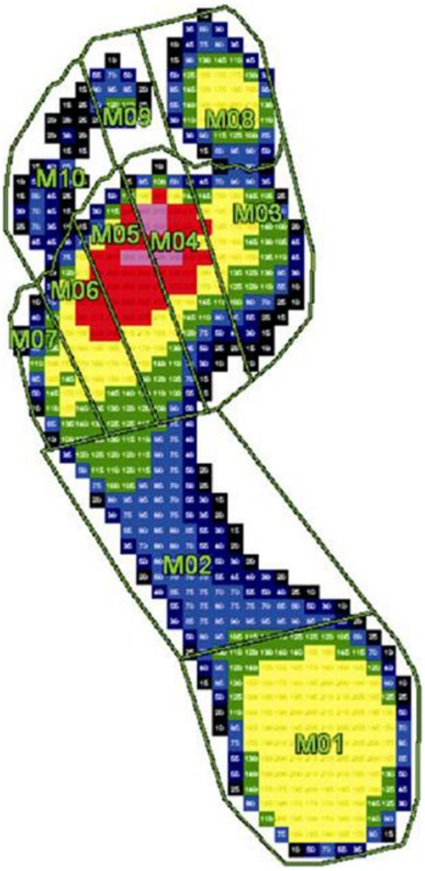

Figure 1. Ten mask areas depicted for peak pressure and pressure-time integral measurements.

Statistical Analysis

All data were explored for normality prior to inferential analysis. Differences in peak pressure, contact time, pressure-time integral, and geometric data between participants with and those without MN were determined using independent-samples *t* tests. Retrospective power calculations

Table 3. Peak Pressures (kPa) for 10 Masks and Pressure-Time Integrals (kPa · ms) for Metatarsals 2 to 4.[a]

	MN Right (n = 30)	Control Right (n = 31)	*P* (95% CI)	MN Left (n = 31)	Control Left (n = 31)	*P* (95% CI)
M1PP	342 ± 130	316 ± 73	.25 (−80 to 21)	357 ± 111	327 ± 86	.25 (−81 to 21)
M2PP	170 ± 58	156 ± 45	.46 (−15 to 33)	148 ± 51	157 ± 43	.46 (−15 to 33)
M3PP	298 ± 106	313 ± 127	.32 (−113 to 38)	366 ± 151	328 ± 146	.32 (−113 to 37)
M4PP	477 ± 174	494 ± 161	.95 (−71 to 67)	476 ± 121	474 ± 150	.95 (−71 to 67)
M5PP	461 ± 174	455 ± 151	.96 (−59 to 62)	437 ± 117	439 ± 122	.96 (−59 to 62)
M6PP	306 ± 99	297 ± 93	.12 (−69 to 8)	303 ± 86	272 ± 65	.12 (−69 to 9)
M7PP	227 ± 147	224 ± 124	.87 (−61 to 52)	216 ± 125	211 ± 96	.86 (−61 to 52)
M8PP	435 ± 213	454 ± 209	.73 (−89 to 127)	446 ± 238	448 ± 195	.96 (−108 to 113)
M9PP	206 ± 97	259 ± 360	.44 (−83 to 189)	173 ± 124	199 ± 82	.34 (−28 to 79)
M10PP	178 ± 69	151 ± 56	.10 (−59 to 5)	116 ± 55	152 ± 85	.05 (−.37 to 72)
M4PTI	153 ± 65	154 ± 48	.96 (−28 to 30)	154 ± 40	150 ± 48	.72 (−27 to 19)
M5PTI	152 ± 69	146 ± 46	.71 (−35 to 24)	148 ± 42	143 ± 42	.65 (−26 to 17)
M6PTI	108 ± 36	106 ± 33	.82 (−19 to 15)	109 ± 31	98 ± 26	.10 (−26 to 3)

Abbreviations: CI, confidence interval; M1 (heel); M2 (midfoot); M3 (first metatarsal); M4 (second metatarsal); M5 (third metatarsal); M6 (fourth metatarsal); M7 (fifth metatarsal); M8 (hallux); M9 (second toe); M10 (third, fourth, and fifth toes); MN, Morton's neuroma; PP, peak pressure; PTI, pressure-time integral.

[a] Data are expressed as mean ± SD.

were conducted using Power and Sample Size Calculation version 3.0.43.[13]

Results

There were no significant differences in the peak pressures of all masked areas and pressure-time integrals under metatarsal 2 to 4 heads between patients with MN and control subjects (Table 3). However, contact time on the right heel was reduced significantly in subjects with MN compared with controls. Contact times were not significantly different in all other masked areas (Table 4). In addition, no significant differences were observed between patients with MN and control subjects in geometric measurements of forefoot length, width, coefficient of spreading, foot progression angle, and arch index (Table 4).

With power set to 80%, type I error 5%, and the minimum pooled standard deviation for each variable, retrospective calculations indicated that minimum detectable differences were 21 kPa for peak pressure, 51 ms for contact time, 0.6 cm for foot length and width, 0.015 for coefficient of spreading, and 0.04 for arch index. These minimum detectable differences increased as pooled standard deviations increased.

Discussion

Our study showed no significant differences in peak pressure and contact time between patients affected with MN and control subjects in 10 masked areas of the foot. In addition, the pressure-time integrals over the second through fourth metatarsal heads in subjects with neuromas were not significantly increased compared with the control group. This is in contrast with any suggestion that peak pressure should increase in the forefoot in the immediate area of an MN. Bauer et al[6] believed that "hyperpressure" under the metatarsals should be addressed for operative management of MN. Bauer et al performed a retrospective study comparing open neurectomy (n = 26) and percutaneous deep transverse metatarsal ligament release with oblique osteotomy at the level of the head of the affected metatarsal on patients diagnosed with MN. They found that the short-term clinical outcomes of the percutaneous osteotomy group were as good as those of the open neurectomy group, with significantly better outcomes in the long term.[6] A number of their subjects had other associated conditions, such as hallux valgus, metatarsophalangeal joint subluxation, and toe deformities. MN can occur simultaneously in the presence of other forefoot conditions. However, it is also important to note that in patients with digital deformities, hallux valgus, systemic arthropathies, and neurological conditions, plantar pressure in the forefoot can increase.[9,12,18,29,39] However, all our subjects had painful MN without any other forefoot problems, which may explain why "hyperpressure" was not significantly different between our MN and control groups. In view of our results, if lesser metatarsal osteotomies are being considered as a treatment of MN, it would seem prudent to undertake preoperative plantar pressure measurements to confirm the need for osseous operative intervention.

Previous studies investigating plantar pressure measurements of patients with generalized forefoot pain have demonstrated increases in peak pressures in the forefoot.[21,28] Keijsers et al,[25] however, attempted to classify forefoot pain

Table 4. Contact Time (ms) for 10 Masked Areas and Geometric Measurements for Patients With Morton's Neuroma and Control Subjects.[a]

	MN Right (n=30)	Control Right (n = 31)	P (95% CI)	MN Left (n = 31)	Control Left (n = 31)	P (95% CI)
M1CT	424 ± 68	462 ± 71	.04 (2 to 74)	445 ± 96	451 ± 79	.79 (−39 to 50)
M2CT	490 ± 85	524 ± 89	.13 (−10 to 79)	502 ± 104	516 ± 94	.59 (−37 to 64)
M3CT	601 ± 74	625 ± 77	.23 (−15 to 62)	632 ± 78	610 ± 97	.35 (−66 to 23)
M4CT	633 ± 72	655 ± 73	.24 (−15 to 59)	653 ± 76	649 ± 85	.86 (−45 to 37)
M5CT	634 ± 87	667 ± 78	.13 (−9 to 75)	664 ± 74	665 ± 86	.96 (−40 to 42)
M6CT	639 ± 74	658 ± 76	.31 (−19 to 58)	656 ± 76	653 ± 88	.87 (−45 to 38)
M7CT	567 ± 134	612 ± 75	.11 (−10 to 101)	603 ± 78	605 ± 88	.92 (−40 to 44)
M8CT	517 ± 113	570 ± 109	.07 (−3 to 110)	563 ± 107	563 ± 121	.99 (−58 to 58)
M9CT	443 ± 119	443 ± 104	.90 (−57 to 57)	426 ± 126	476 ± 97	.08 (−7 to 107)
M10CT	535 ± 111	507 ± 116	.34 (−86 to 30)	502 ± 134	505 ± 132	.93 (−65 to 70)
FL (cm)	25.83 ± 1.54	25.5 ± 1.71	.49 (−1.12 to .55)	25.66 ± 1.49	25.46 ± 1.74	.63 (−1.02 to .62)
FW (cm)	9.99 ± 0.83	9.94 ± 0.74	.82 (−.45 to .35)	10.12 ± 0.76	10.00 ± 0.76	.53 (−.51 to .26)
FPA (deg)	9.85 ± 5.83	10.71 ± 5.89	.56 (−2.13 to 3.87)	8.56 ± 5.74	7.63 ± 6.13	.54 (−3.9 to 2.09)
COS	0.38 ± 0.02	0.39 ± 0.02	.47 (−.01 to .16)	0.39 ± 0.02	0.39 ± 0.02	.67 (−.01 to .01)
Arch index	0.21 ± 0.05	0.22 ± 0.05	.32 (−.01 to .04)	0.20 ± 0.06	0.22 ± 0.05	.11 (.00 to .05)

Abbreviations: CI, confidence interval; COS, coefficient of spreading; CT, contact time; FL, foot length; FPA, foot progression angle, FW, foot width; M1, heel; M2, midfoot; M3, first metatarsal; M4, second metatarsal; M5, third metatarsal; M6, fourth metatarsal; M7, fifth metatarsal; M8, hallux; M9, second toe; M10, third, fourth, and fifth toes.

[a] Data are expressed as mean ± SD.

on the basis of plantar pressure measurements but did not find any significant differences in peak pressures under the metatarsal heads between subjects with (n = 283) and those without (n = 311) forefoot pain. None of the participants in their study had a confirmed diagnosis of MN, and 207 subjects had other toe deformities, heel pain, and ankle pain, which may have affected plantar forefoot pressures. In contrast to our findings, they found that pressure-time integrals and contact times under metatarsals 2 and 3 were significantly increased in the forefoot pain group in comparison with their asymptomatic group. They proposed that in subjects without foot deformities, the peak plantar pressure under the heads of metatarsals was not as important as other factors such as plantar fat pad thickness and pain tolerance.

Our results showed no significant difference between the arch index of patients and control subjects, which supports our previous findings using the Foot Posture Index to compare foot type with the presence of MN.[30] Although the Foot Posture Index is a static measurement of foot type, we did not see any difference in the arch index measured on the basis of the dynamic plantar pressure measurement system. Therefore, on the basis of our subjects, we can conclude that foot type may not be a sole predisposing factor in the pathogenesis of MN.

Logically, the presence of pain can potentially alter a person's gait pattern.[41] At the time of the data collection, all patients were pain free in order for them to meet the inclusion criteria and participate in the EMED testing. In this manner, we assumed that all subjects with MN did not guard against pain with an unusual foot strike on the platform.

There were no significant differences in width, length, and coefficient of spreading between patients with MN and control subjects. On the basis of previous studies, men have wider feet than women.[5,38] Male subjects constituted 39% of the control subjects and approximately 25% of the subjects with MN. No attempt was made to separate men from women in either group for this part of the data analysis. This unequal distribution may have affected our results, as there were higher percentages of men in the control group. However, a recent study by Park et al[34] would seem to support our findings, showing no significant differences between forefoot width and intermetatarsal distances of patients with MN compared with a control group using weightbearing radiographs. Their study did not evaluate what happens to the width of the forefoot during the push-off phase. In our study, we did not see any significant differences in forefoot width and coefficient of spreading dynamically between patients with and those without MN.

This study had some limitations. It was not possible to confirm the diagnosis of MN by operative histopathology, and it was necessary to rely on clinical examination and ultrasound findings to recruit subjects into the study. However, on the basis of previous literature, clinical examination[17,35] and ultrasound[7,42] are highly reliable in detecting MN. In addition, in future studies it may be beneficial to see a comparison between unaffected and affected feet for patients with unilateral MN for all EMED parameters studied. The gender imbalance may have been less problematic if we had studied unaffected and affected feet with unilateral subjects with MN.

In conclusion, in our sample of patients with MN and control subjects, we did not observe any significant increase in barefoot peak pressure and contact time measurements. We question the need to perform osteotomy to treat MN not associated with other lesser metatarsal phalangeal joint pathologies. Similarly, there were no significant differences in width, length, foot progression angle, arch index, and coefficient of spreading between the 2 groups. The influence, if any, of different styles of footwear on forefoot measurements, including plantar pressures, in subjects with MN is another area of research that needs to be explored.

Declaration of Conflicting Interests

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