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Running-related injury prevention through innate impact-moderating behavior

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ABSTRACT

ROBBINS, S. E., G. J. GOUW, and A. M. HANNA. Running-related injury prevention through innate impact-moderating behavior. *Med. Sci. Sports Exerc.*, Vol. 21, No. 2, pp. 130–139, 1989. The purpose of these experiments was to test the Robbins and Hanna hypothesis, which relates differences in discomfort from localized deformation at certain positions on the plantar surface to protective behavior (intrinsic foot shock absorption). A penetrometer was used to quantify the relations between localized load and pain and between load and depth of deformation. The magnitude of load required to elicit pain varied significantly ($P < 0.005$) in relation to position on the plantar surface. With a load of 9 kg and a 10 mm spherical end on the penetrometer, 6% of the sample reported pain at the heelpad, 32% at the distal first digit, and 66% at the first metatarsal-phalangeal joint. This pattern was predicted by the Robbins and Hanna thesis. Two deformation patterns were observed which were best explained by deformation constraint by tight trabecular tethering of the epithelial membrane at the heelpad and distal first digit and unrestricted deformation due to loose trabecular tethering of the epithelial membrane at the first metatarsal-phalangeal joint. These data provide insight into how, when barefoot, the plantar surface resists perforation yet provides protection to local bony structures. These data further support the notion that plantar sensory feedback plays a central role in safe and effective locomotion.

BIOMECHANICS, CUTANEOUS SENSATION, FOOT, GLABROUS SKIN, INJURY PREVENTION, LOCOMOTION, PAIN, RUNNING

The plantar and palmar surfaces are similar histologically, their outer layers consisting of glabrous epithelium covering a specialized adipose tissue. Both have a dense network of fibrous trabeculae which traverse the adipose layer and limit horizontal (and perhaps vertical) movement of the epithelium. Both are highly sensible, possessing a high density of mechanoreceptors and nociceptors including Meissner corpuscles, which are found only in the digits of higher primates (25). Both surfaces are capable of adapting through hyperkeratinization to resist wear.

Despite the above similarities, their primary function is considered different by most: the palmar surface is appreciated in terms of its sensory processes (active touch and tool manipulation), whereas the plantar surface is regarded for its resistance to mechanical forces associated with weightbearing and locomotion. Accordingly, the sensory neurophysiology of the palmar surface has been carefully examined, whereas plantar surface sensibility has only recently received attention (28).

The sensibilities of the two glabrous surfaces are similar, but there are differences. With regard to the perception of plantar events, a recent report found the psychophysical function relating load applied to the plantar surface (0–170 kg) to the perceived magnitude of this load to be linear, with a slope of unity (28). This is comparable to the relation reported for the perceived amplitude of discrete loads applied to the palmar surface (14,15). Sensory thresholds obtained form the plantar surface are about half those of the palmar surface (13,35,36). Some of this divergence may be accounted for by differences in stratum corneum thickness due to hyperkeratinization with use, and perhaps variation in receptor density.

The relation between surface mechanical stimulation and behavior distinguishes the two glabrous surfaces. In conscious, upright humans, one can elicit stereotypic behavioral responses to even light mechanical stimulation of the plantar surface (7,27). Other than the grasp reflex of the infant, nothing similar is evident on stimulation of the palmar surface.

Certain observations suggest that behavior induced by plantar tactile sensations are important to humans. It is common knowledge that individuals have difficulty in maintaining equilibrium and walking when the plantar surface is anesthetized by cold, and gymnasts balance more effectively when barefoot. Moreover, sensations arising from the plantar surface must additionally provide protection against local damage when unshod, and, even when shod, they defend against pressure ulceration (9).

Studies relating plantar tactile stimulation to behavior were influential in framing Sherrington's view of nervous system integration (30). While his notion about

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"chains of reflexes" is no longer considered as important to humans as he suggested, specific reflexes, in particular the plantar tactile flexion reflex, are generally considered to be important to locomotion, although the magnitude of their influence is impossible to quantify with existing methods (19).

The relation between plantar sensibility and behavior has taken on new importance due to a hypothesis which asserts that footwear use results in attenuation of plantar sensation (a reduction of impact-moderating behavior). This is thought to result in impulsive loading, which causes both acute and chronic injuries following the neuropathic medical model, termed "pseudo-neuropathic" by its authors (26). Since the application of this thesis through natural means (promotion of barefoot activity) and through devices (footwear modifications) may have implications in the prevention of common disorders (e.g., stress fractures, osteoarthritis of weightbearing articulations, etc.), more detailed examination of the plantar surface seems warranted.

The analysis of impact attenuation during locomotion is facilitated by examining independently the moderation of the elements that define impact: temporal load control and load magnitude control (1,20,27) (Fig. 1).

Examples of temporal load control are intrinsic foot shock absorption and hip flexion (26,27). Both are considered as avoidance of noxious plantar sensation. Similarly, each places an element that can yield repeatedly without failure (skeletal muscle undergoing eccentric contraction) in series with the vertical skeletal axis. Both are thought to be diminished with footwear use compared to barefoot on natural surfaces, due to the regular interior of footwear (27).

Intrinsic foot shock absorption is a consequence of voluntary or reflexive (i.e., part of the plantar tractile flexion reflex) plantar load redistribution to regions of lower sensibility to noxious stimuli, by activation of digital plantar flexors—essentially a change in forefoot load from the metatarsal-phalangeal joints (the preferred forefoot loading location in shod populations) to the distal digits (thought to be the preferred forefoot site in the unshod) (5). This makes the previously rigid arches capable of yielding during loading. Central to this theory is the as yet unsubstantiated assumption of a lower threshold to pain caused by local deformations at the metatarsal-phalangeal joints compared to the distal digits.

As an example of load magnitude control, plantar surface discomfort may induce the individual to diminish vertical movement (and other dynamic factors that could moderate impact) during locomotion. The magnitude of hip flexion from plantar surface-weightbearing surface interaction has been proposed as a measure of load magnitude control (27).

The thesis of intrinsic foot shock absorption is supported by a variety of reports. A relation has been reported between barefoot activity and raising of the main longitudinal arch, presumably by increased intrinsic foot muscle tone (26). The subjects with the greatest reduction in arch span performed barefoot activity outdoors. This suggests that surface irregularities causing local deformations on the plantar surface contributed to intrinsic foot muscular activation. It would appear likely that sensory thresholds for pain (an initiator of avoidance) from deformation are lower at the metatarsal-phalangeal joints, since it is only the region that is spared by digital plantar flexion from intrinsic foot muscle activation. This conclusion can be appreciated by examining displays from plantar load mapping devices, since voluntary digital flexion while bearing weight results in both the redistribution of load to the digits and a shift in load laterally due to the rising medial longitudinal arch as predicted by the Robbins and Hanna thesis (Fig. 2).

Similar conclusions are reached by reviewing investigations related to diabetic neuropathy. Diabetic neuropathy causes both sensory and motor deficits, although the sensory loss is more widely discussed because it is more easily quantified (9,10,12). Harrison and Faris investigated the motor consequences of these neuropathies and found that inexcitable foot muscles are often present in the ulcerated foot of diabetics, which causes increased load at the metatarsal-phalangeal joints (11). The redistribution of load from the digits to the metatarsal-phalangeal joints has been documented in diabetics with plantar ulcerations (33).

Recently, athletic footwear has been shown to attenuate the perceived magnitude of loads applied to the plantar surface (28). If the metatarsal-phalangeal joints have a relatively low threshold to mechanical transients, a protective adaptation that is suggested by the previous reports, one would suspect signs of abuse of this articulation in traditionally shod populations where plantar sensations are attenuated by footwear (in a manner similar to diabetics with neuropathy). This conclusion is suggested by reports which indicate a lower incidence of osteoarthritis at the metatarsal-phalangeal joints and a higher incidence at the distal interphalangeal joints in unshod compared to shod populations (3,31,32).

The hypothesis of impact moderation during locomotion through hip flexion is supported by data which relate the magnitude of hip flexion to the magnitude of plantar surface loading and to surface irregularity (27).

While the above arguments suggest differences in plantar pain sensitivity from localized deformation at certain sites, it would be unwise to accept the Robbins and Hanna thesis without direct confirmation of this. Accordingly, the purpose of this investigation is to quantify the load required to elicit pain from localized deformation at three plantar locations and to test directly the predictability of the Robbins and Hanna hypothesis of intrinsic foot shock absorption; a severe test (attempted refutation through extension of theory

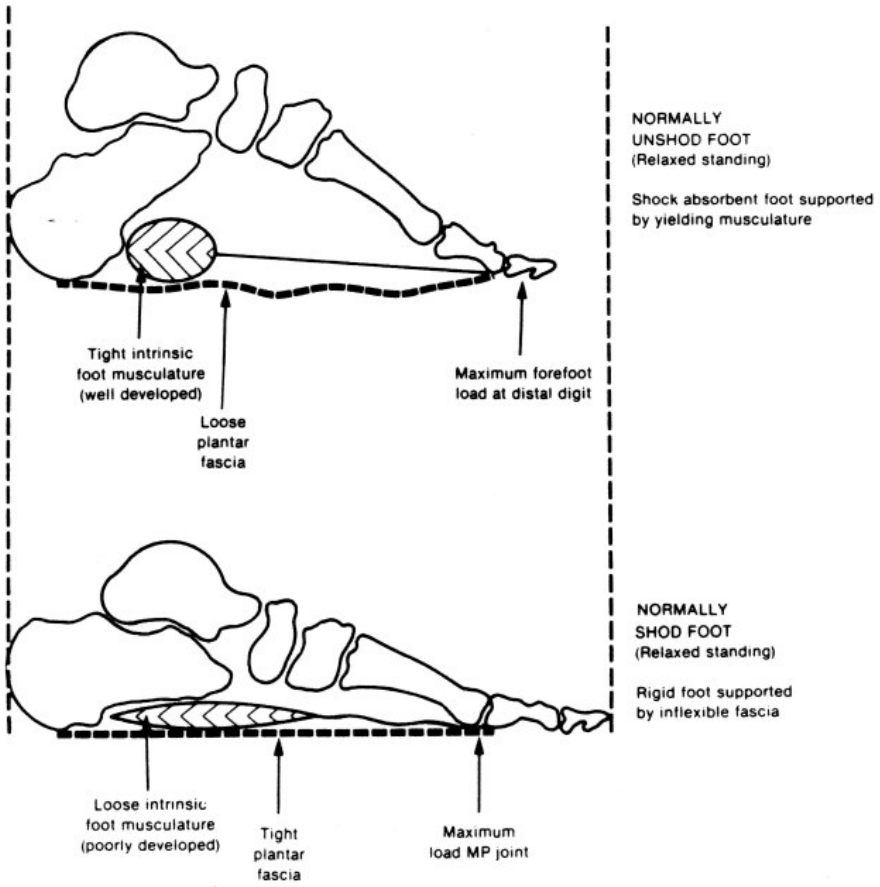

Figure 1—Diagrammatic representation of the Robbins and Hanna hypothesis, which relates plantar sensory-mediated avoidance of the metatarsal-phalangeal joints due to low pain threshold at that site to enhanced intrinsic foot shock absorption, via eccentric contraction of digital plantar flexors producing medial arch deflection.

explainability) by Popper's criteria (24). These thresholds will be related to anatomic differences and deformation patterns.

METHODS

Equipment development. An existing device, a penetrometer (Clockhouse Engineering Limited), consists of a spring-loaded shaft and housing (Fig. 3). A scale (0–9 kg) is provided on the housing for reading the load applied to the shaft. A partially hollow collar (33 mm OD 25 mm ID) was added to the shaft so that it could slide with a minimum of resistance. The device was designed for use in the horizontal position. Removable steel balls were made to attach to the end of the shaft by bonding a tubular sleeve to the ball. This device showed repeatability of ±0.1 kg for load, of ±0.5 mm for deformation, and accuracy of ±2%.

With these modifications, depth of deformation could be measured by obtaining the distance from the most distal aspect of the ball to the rim of the collar. For purposes of these experiments, a 10 mm and a 23 mm spherical end were used, the 10 mm end being the smallest size that subjects would tolerate at full load (9 kg) and the 23 mm end the largest the collar could accommodate. It should be noted that, due to the contour of the plantar surface, the 23 mm end was appropriate for use only at the heelpad.

Locations tested. Locations tested and the means of their localization can be obtained from Figure 3. Of the three sites, only the metatarsal-phalangeal joint is an articulation. Considerable differences in deformation depth were obtained in relation to the degree of flexion and extension at this joint. To provide meaningful results, the joint was in the neutral position while taking measurements; i.e., the digit was positioned so as to be a continuation of the first metatarsal.

Histological techniques. Tissues taken at postmortem were fixed, embedded, sectioned, and stained with hematoxylin and eosin, following standard pathological practices.

Statistical procedures. Sample means were compared using two-tailed t tests. Chi-square was used for pain threshold data due to the limited degrees of freedom.

Experiment 1. A sample of 100 subjects (58 males, age range 16–64, mean 37; 42 females, age range 14–64, mean 33) from a symptom-free academic and industrial population were tested using the penetrometer with a 10 mm spherical end. The penetrometer was repeatedly pressed into the plantar surface of the right foot while maintaining contact between the plantar skin and the collar of the penetrometer until the desired reading on the penetrometer scale was obtained. Five readings were taken at each of the three sites (Fig. 3) using an ascending scale at increments of 1.8 kg, to a maximum of 9 kg. This was a dynamic test insofar as

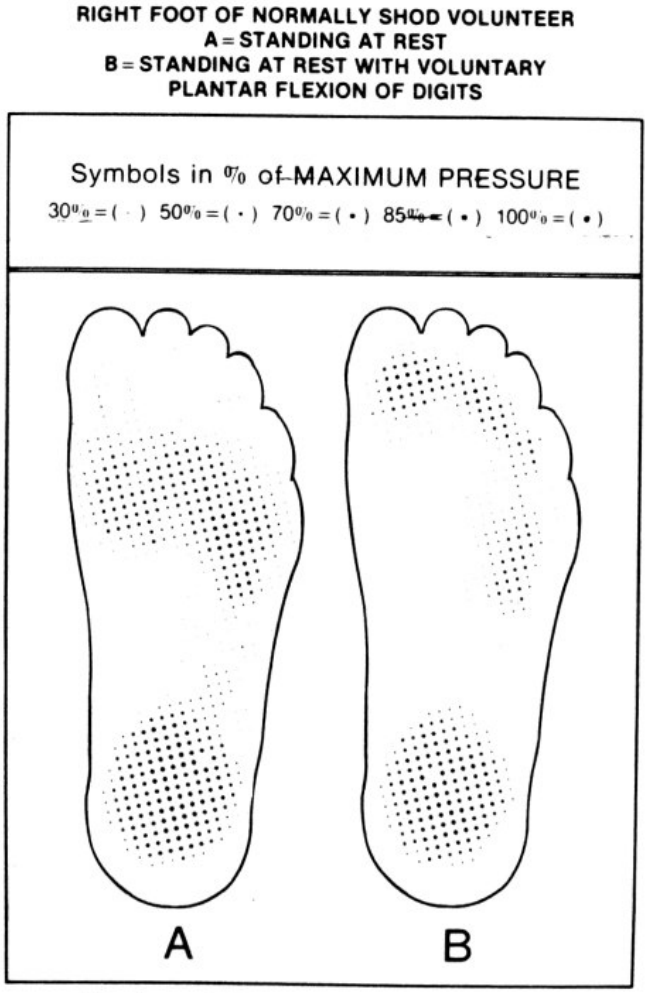

Figure 2—Computer-generated display from a force platform showing how voluntary digital flexion results in redistribution of load from the metatarsal-phalangeal joints to the distal digits and a shift in load laterally due to the rising medial longitudinal arch (courtesy of Midi-Capteurs, Toulouse, France, and Apocom Canada Ltd.).

load was applied for approximately 1 s so as to be similar to the duration of loading during walking and distance running. The interval between readings was 10 s. After the application of load, the depth of deformation was measured, and the subject was asked whether he or she experienced pain.

Experiment 2. A sample of 17 subjects, from a population of symptom-free male recreational runners, were examined at the heelpad site using a 10 mm and a 23 mm end on the penetrometer. The series of loads was ascending in order, using the method discussed above. The depth of deformation was recorded for each load level.

Experiment 3. The load-deformation relation was measured on ten fresh cadavers at the heelpad, by the above methods, prior to and following severing of the plantar skin outside the perimeter of the collar of the penetrometer.

Experiment 4. Adipose layer thickness was measured at the three sites (Fig. 3) on five female and five male

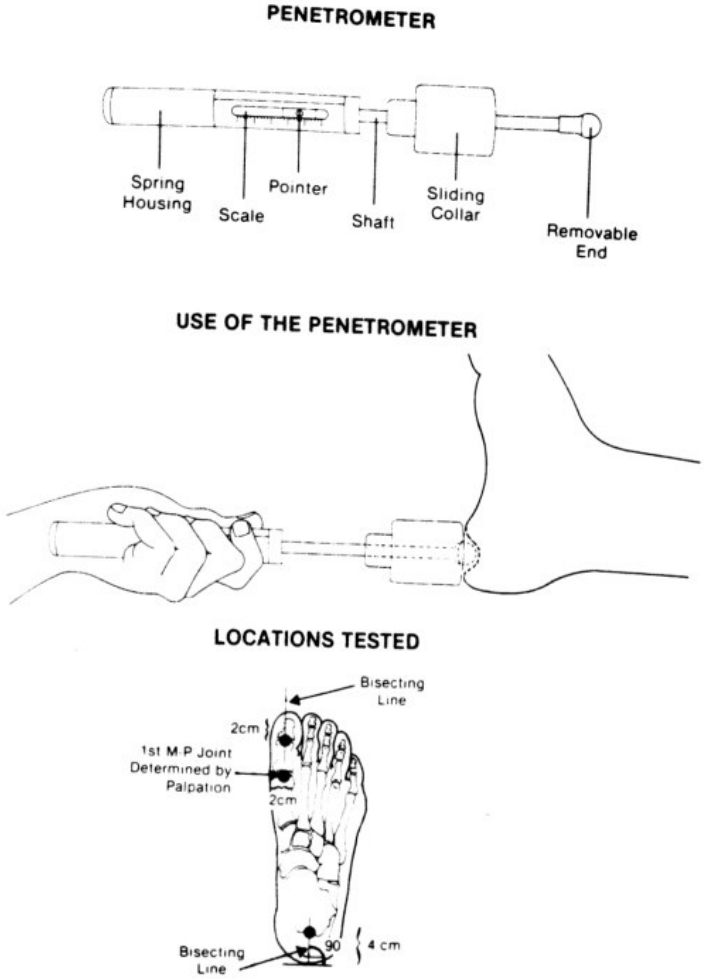

Figure 3—The sliding collar and removable spherical ends are modifications for purposes of these experiments of a device which was originally designed for use in geotechnical studies. MPJ readings were taken with the digit in the neutral position, i.e., the digit being an extension of the first metatarsal.

fresh cadavers. All were between 25 and 50 yr of age at their death. The soft tissues were incised with a scalpel to the underlying bone and measured to the nearest 0.5 mm. To obtain adipose thickness rather than soft tissue thickness, 2.5 mm (the estimated thickness of periosteum and epidermis microscopically) were subtracted from the measured value.

RESULTS

Pain sensitivity (Fig. 4). Pain threshold varied in relation to location on the plantar surface, with the heelpad highest, distal first digit intermediate, and first metatarsal-phalangeal joint lowest. At 9 kg, chi-square (2) = 79.95, $P < 0.005$.

Adipose tissue thickness. The mean adipose thicknesses were (in mm) HP 13.75, DFD 7.25, and MPJ 5.25, with significant differences ($P < 0.05$) between the heelpad and the other two locations. There were no significant differences between men and women, except at the distal first digit (male 9, female 5.5; $P < 0.05$).

Load-depth of deformation (Figs. 5, 6, and 7).

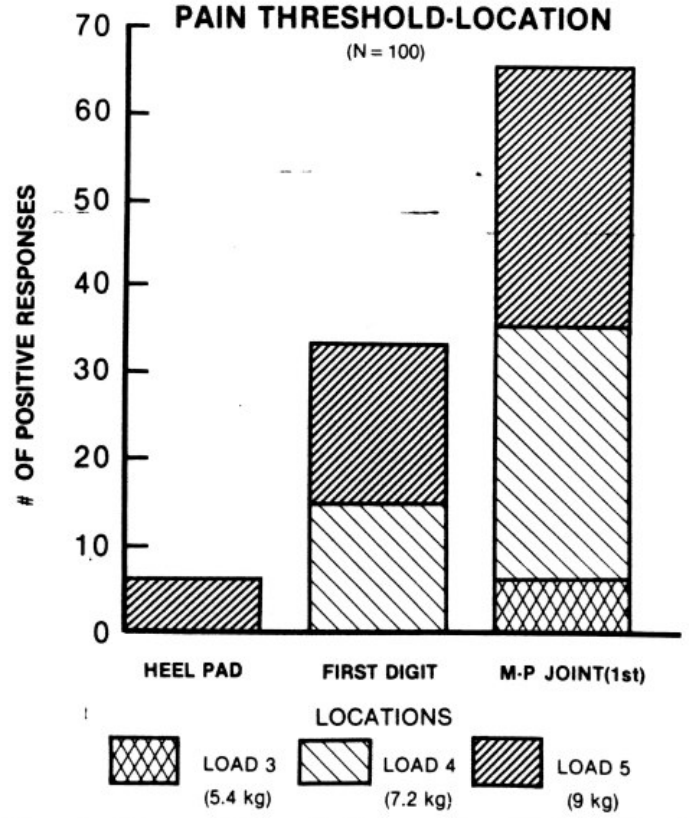

Figure 4—Pain sensitivity results from experiment 1: the number of subjects experiencing pain at specific loads utilizing a penetrometer with a 10 mm spherical end.

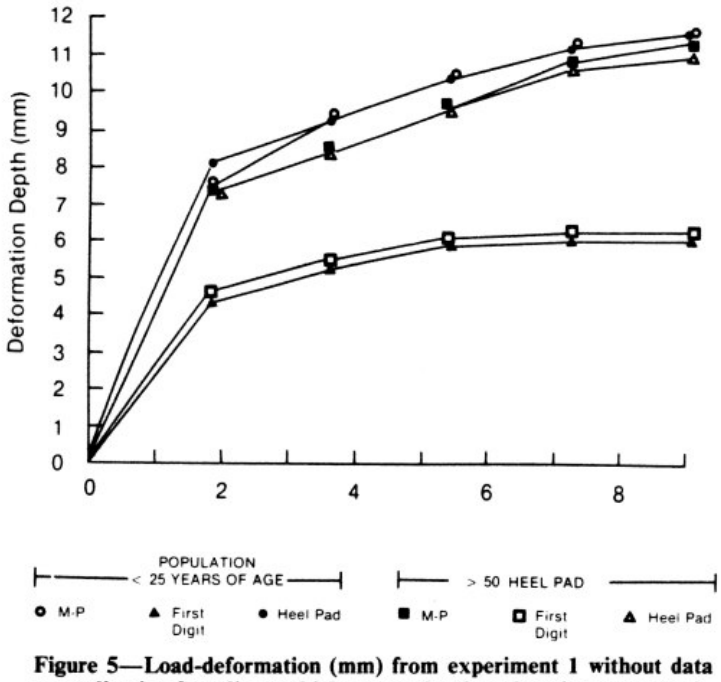

Figure 5—Load-deformation (mm) from experiment 1 without data normalization for adipose thickness at the three locations examined.

There was no significant difference in the mean depth of deformation (in mm) at the heelpad and first metatarsal-phalangeal joint location (both asymptotic at 12), both being significantly different from the distal first digit location (asymptotic at 6; $P < 0.05$). There were no significant differences between the sex groups; for example, a load of 9 kg, mean and standard error at different sites were:

males: $N = 58$; HP 11.47 ± 1.95; MPJ 11.18 ± 1.80; DFD 6.10 ± 1.02;
females: $N = 42$; HP 11.57 ± 2.57; MPJ 11.52 ± 2.32; DFD 6.10 ± 1.16.

Similar measurements taken from fresh cadavers, which were used in experiments 3 and 4, were within one standard deviation of the means obtained from experiment 1, which confirms the validity of postmortem testing when investigating patterns of plantar surface deformation.

When the depth of deformation was expressed as percentage of total adipose tissue thickness (as taken from experiment 4), the mean deformation at maximum load (9 kg) was 84% and 89.5% at the heelpad and distal first digit, respectively, both differing significantly ($P < 0.05$) from the mean first metatarsal-phalangeal joint deformation, which was 221% of the measured adipose thickness (Fig. 6).

Age-related differences. There were no significant differences in deformation depth (in mm) between different age groups; for example, at a load of 9 kg, the mean and standard error at different sites were:

>50 yr: $N = 21$; HP 10.98 ± 2.82; MPJ 11.33 ± 1.90; DFD 6.26 ± 0.87;
<25 yr: $N = 30$; HP 11.56 ± 9.07; MPJ 11.65 ± 2.18; DFD 6.07 ± 1.13.

Histological differences (Fig. 8). At the three locations, by microscopic examination, visual differences were noted in adipose thickness, trabecular thickness, and trabecular density. There was increased thickness of fibrous trabeculae at the heelpad compared to the other two positions. There was a higher density of trabecular tissue relative to adipose tissue at the heelpad compared to the other locations.

Experiment 3. There was no change in load-deformation relations following severing of the plantar skin around the circumference of the penetrometer collar.

DISCUSSION

Pain threshold data support the Robbins and Hanna hypothesis, which relates differences in discomfort from localized deformation of the plantar surface to the initiation of intrinsic foot shock absorption. When barefoot on natural surfaces (naturally deposited ground), sensitivity differences can be seen to induce behavior that results in the transfer of forefoot load from the metatarsal-phalangeal joints to distal digits, also changing arch support from unyielding plantar fascia and ligament onto yielding musculature. Conversely, the lack of similar adaptations in shod populations is explained by the regular interior of modern footwear,

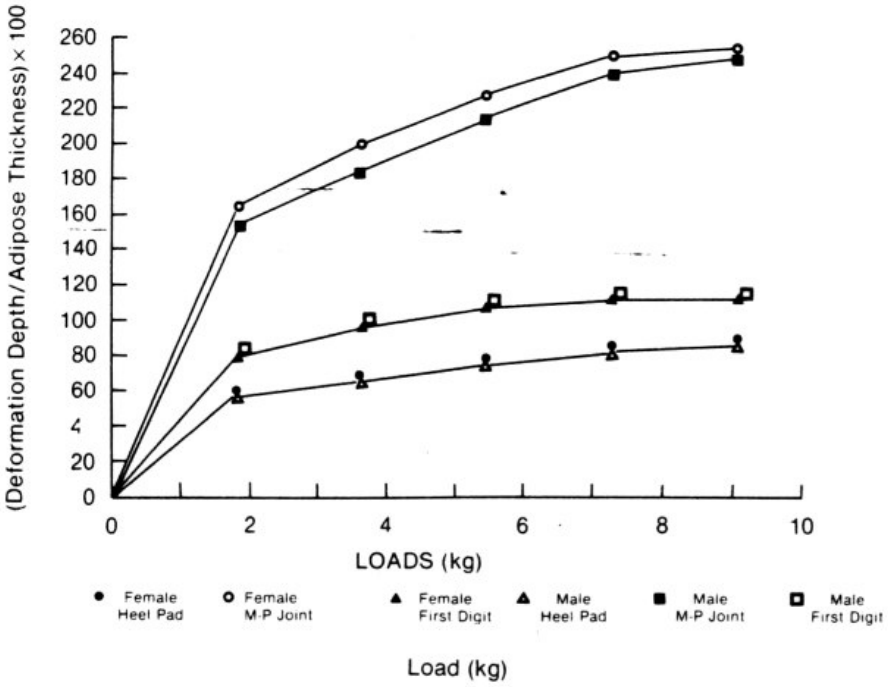

Figure 6—Load-deformation from experiment 1 following normalization for differences in adipose thickness at the three testing sites on the plantar surface.

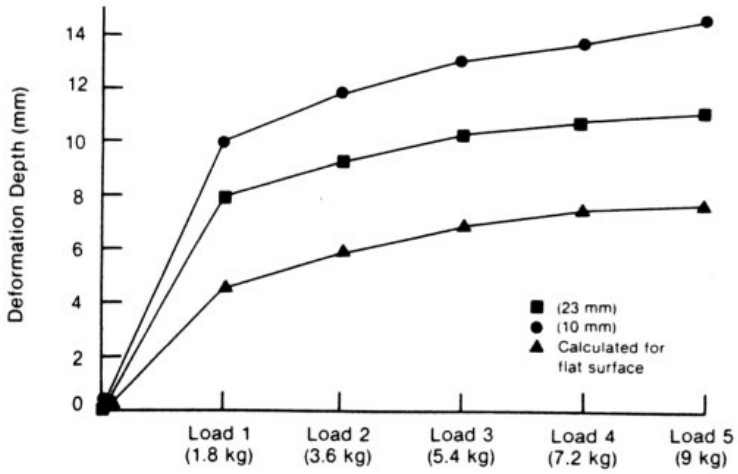

Figure 7—Load-percentage of maximum deformation from experiment 2. This indicates that the heelpad loses it shock absorbency at a load of less than 9 kg independent of the size of the deforming object.

which diminishes sensory feedback on weightbearing. This may result by direct trauma in osteoarthritis of the metatarsal-phalangeal joints in shod populations and, indirectly (via augmented impact), in running-related injuries and perhaps osteoarthritis of the hip articulation.

Although intrinsic foot shock absorption may be voluntary avoidance, its stereotypic character—its association with withdrawal through hip flexion and loading of the contralateral extremity—suggests that the flexion reflex to plantar tactile stimulation contributes to this response (16,26,27,29). If this reflex is utilized during locomotion, one might expect evidence of knee and ankle flexion, if the testing system allowed its measurement. The tactile flexion reflex may provide impact moderation rather than solely local protection as has been previously suggested (7,16,29).

The behavioral response to noxious plantar stimulation varies in relation to position of stimulation on the plantar surface (16). Stimulation of the heel or plantar surface near the metatarsal-phalangeal joints results in plantar flexion of the digits and flexion at the hip, knee, and ankle. When stimulating the hollow formed by the medial longitudinal arch (a contact area in most footwear but not when barefoot), the response is identical, with the exception of extension of the digits rather than plantar flexion. Stimulation of areas on the plantar surface not normally used for weightbearing when unshod, such as the skin of the medial longitudinal arch, may interfere with intrinsic foot shock absorption by inducing digital dorsiflexion. Plantar flexion of the digits following noxious plantar stimulation has been shown to utilize intrinsic foot musculature rather than long flexors (16).

The nature of pain elicited by deformation by large (>10 mm) objects varied in relation to plantar surface site. At the heelpad or distal digit, subjects reported pain commencing as the penetrometer was withdrawn—about 1 s, which is consistent with the latency of C fibers of the palmar surface (4). When load was sustained at a level that was uncomfortable but not painful, most subjects reported pain within several seconds, which was not associated with a marked withdrawal response. Pain was described as gradually increasing in intensity, never sharply localized, and gradually becoming more diffuse so as to interfere with its localization. Since pain was perceived only after 1 s following stimulation, during locomotion (particularly when running), the duration of weightbearing is too

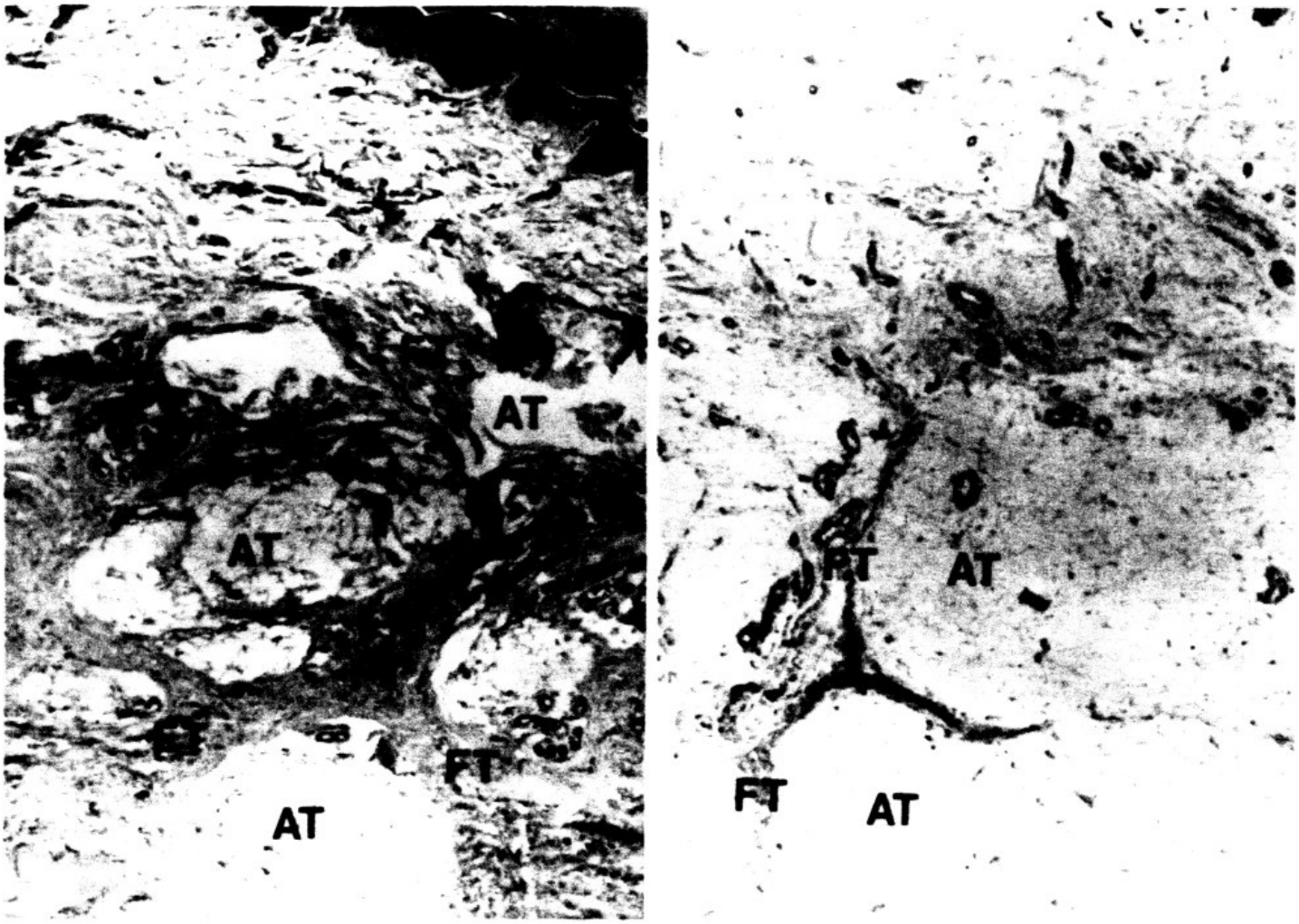

Figure 8—Transverse sections through the human plantar surface. *Left*—heelpad; note the dense fibrous trabeculae (FT) relative to the small cavities containing adipose tissue (AT). *Right*—first metatarsal-phalangeal joint: note the sparse and thin trabeculae and greater amount of adipose tissue. The distal digit anatomy (not shown) resembles the heelpad. Disruptions of the adipose cells and fragmentation of the trabeculae are common artifacts produced from sectioning tissues of this type.

brief to elicit pain at first contact, at least from the range of loads used in this experiment. Perhaps initial contact lowers the pain threshold and produces background discharge (sensitization), a phenomenon known to exist with polymodal nociceptors with C afferent fibers, resulting in a sustained sensation of pain at future contacts (2,4,22). Without this explanation it is difficult to account for plantar pain generation during the cyclic loading in locomotion in its various forms on irregular surfaces. This explanation seems consistent with the pain one experiences when walking on hot or irregular surfaces when barefoot. The gradual onset of pain at the preferred weightbearing sites indicates that this may also protect against ulceration from sustained pressure.

At the metatarsal-phalangeal joint, subjects reported an abrupt onset of localized pain as soon as the penetrometer met resistance, which is probably caused by pressure on underlying periosteum, tendon, or joint capsule. The pain was frequently associated with immediate withdrawal. The latency suggests that receptors of at least the initial pain are nociceptors with myelinated fibers (A-delta) (10). Because of its rapid onset, pain from surface deformations at this site would probably occur during locomotion at the threshold observed in this experiment. Subjects did not describe this pain as becoming diffuse when load is sustained.

Though no detailed work has been done on the physiology of plantar sensibility in humans, data from the plantar surface of primates indicate an abundance of A-delta and C fibers (the primary afferents of nociceptors) in the deeper epithelial layers of the glabrous skin and, in the case of C fibers, probably in the trabeculae of the subcutaneous layer (10). Data suggest that at the heelpad and distal digits pain receptors are located in continuity with the trabeculae and are stimulated by deformation caused by trabecular tension, since the periosteal origin of pain is unlikely because the penetrometer end never reaches it and surface deformation sensors located in cutaneous layers cannot respond because deformation is asymptotic when pain is perceived. This is also supported by the mechanics of the tethered model (to be mentioned later), which

predicts a rapid increase in trabecular tension after the epithelium is firmly tethered.

As an explanation of the directional characteristics of SAII sensory fields, Johansson has similarly proposed that receptors (thought to be of the Ruffini type) of SAII primary afferent fibers of the palmar surface, which are known to sense depth of surface deformation and shear, may be in continuity with the subcutaneous trabeculae (12).

The load required to reach asymptotic deformation never exceeded 9 kg at the three sites that were examined. Further, at the heelpad (and likely at the other sites though not obtainable by the apparatus that was used), deformation depth was asymptotic at the above load regardless of the diameter of the spherical deforming end on the penetrometer.

The following equation, as obtained from these experiments, relates deformation depth and diameter of a spherical deformer at the heelpad:

$$y^2 = 4ax, \text{ where } a = \frac{d - \sqrt{d^2 - 0.96d - 25.2}}{0.3},$$

x = load applied at heel (kg),
y = depth of deformation of heelpad (mm),
d = diameter of spherical deforming object (mm),
a = constant for each value of d.

Deformation depth at the heelpad is asymptotic, with its maximum depth inversely related to the diameter of the deforming object. The depth of deformation was asymptotic at 15 mm with the 10 mm end, 11 mm with the 23 mm end, and, when considering a flat surface, deformation depth is calculated to be 7.6 mm. This indicates progressive constraint on deformation depth at less than the measured thickness of the heelpad adipose layer. Similar constraint is present at the distal first digit, since deformation depth does not exceed adipose thickness at this site as well. Data reported by Petit and Galifret suggest a similar deformation pattern at the distal digits of the human hand (23). At the first metatarsal-phalangeal joint, the pattern of deformation differs only in that its depth exceeds the measured adipose thickness (Fig. 6).

Either of two models might explain the mechanics of the plantar surface (variability in depth of deformation, asymptotic deformation at similar loads at different sites) while respecting the mechanical properties of the tissues (relatively inelastic epidermis and trabeculae; displaceable adipose tissue) (Fig. 9). By a membrane tension model, displaceable adipose tissue is covered with a flexible and inelastic membrane in varying degrees of tension: relatively tight at the heelpad and distal digits, which limits deformation at less than the measured adipose thickness, and loose at the metatarsal-phalangeal joint, allowing adipose displacement in excess of measured thickness.

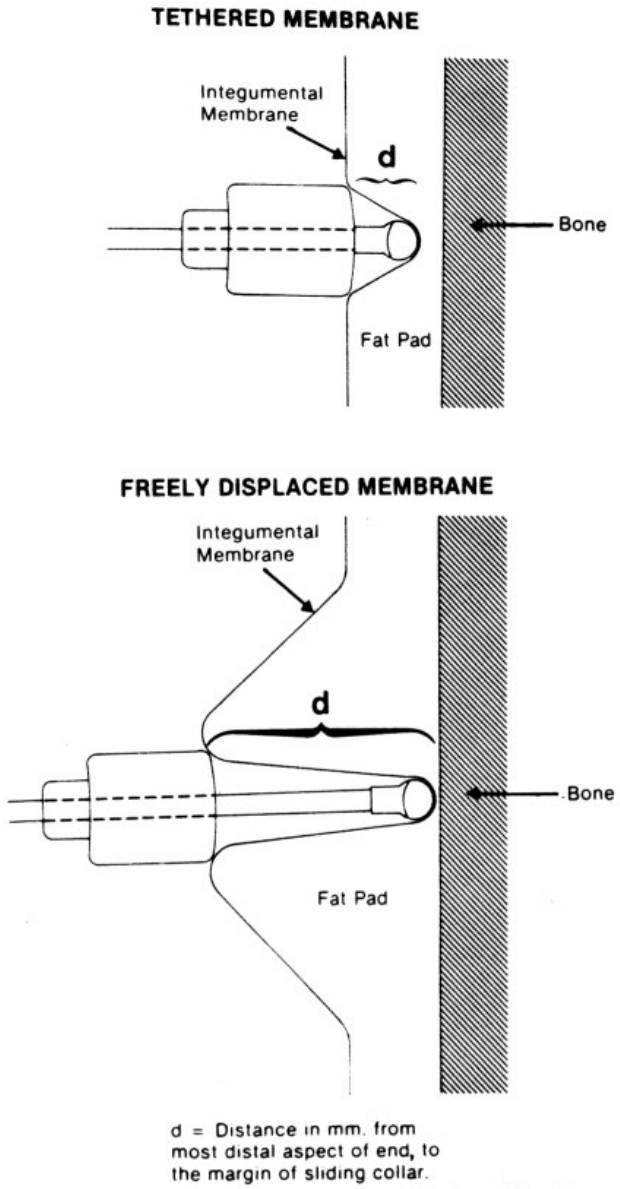

Figure 9—Diagram of the two patterns of deformation that were observed. Experiment 3 suggests that tethering and freely displaceable membranes were due to differences in trabecular properties at the three sites. The tethered membrane explains the mechanics of the plantar surface at heelpad and distal first digit. The freely displaceable membrane model explains the mechanics of the plantar surface at the first metatarsal-phalangeal joint.

Following a tethering model, a flexible and inelastic membrane is tethered by fibrous trabeculae which traverse the adipose layer: tightly tethered at the heelpad and distal digits, resulting in deformation of less than the measured adipose thickness, and loosely tethered at the metatarsal-phalangeal joint, allowing deformation in excess of measured adipose thickness through vertical displacement (Fig. 6).

Experiment 3 serves to examine the role of membrane tension in restraining deformation. The tethering model is supported by the lack of change in load-deformation relations after the elimination of membrane tension through severing of the cutaneous layer

around the circumference of the penetrometer collar. This thesis is also supported by histologic examination, since trabeculae are more abundant and massive at the positions where deformation depth does not exceed adipose thickness. Contrary to another report (5), the authors of this paper found, on dissection of fresh cadavers, no evidence of positive pressure on incision of the heelpad, nor did the adipose tissue flow forth freely.

The lack of constraint at the first metatarsal-phalangeal joint has an obvious utility. In contrast to the other locations, this an articulation. Tight tethering would interfere with locomotion by reducing dorsiflexion of the first digit. However, this mobility has come at a cost, since contact with rigid objects when barefoot (e.g., the 10 mm penetrometer end), which would not reach bony structures at the other sites, is capable of causing damage here. This explains the additional protection presumably offered by the low pain threshold, which directs contact away from this poorly protected area. The low incidence of osteoarthritis at the metatarsal-phalangeal joints in unshod populations attests to the effectiveness of this protective mechanism (3,31,32). Conversely, the high incidence of metatarsal-phalangeal joint osteoarthritis in shod populations suggests that this site is inappropriate for weightbearing, for it still deteriorates despite protection from local damage afforded by footwear.

The heelpad is often considered according to viscoelastic theory, although Thompson notes that its deformation pattern is complex and defies easy classification (34). In our experiments, when the penetrometer load was held constant for 30 s, the so-called "creep phase," change in depth of deformation was noted only at loads of 5 kg or less, regardless of the diameter of the penetrometer end (6) (Fig. 7). When considering the magnitude of the influence of heelpad viscoelastic behavior during locomotion in all its forms in humans, since 5 kg is significantly lower than the peak load in locomotion and 30 s significantly longer than the duration of loading in the support phase of human locomotion, we conclude that, during locomotion in humans, the heelpad does not exhibit significant viscoelastic behavior. These results are similar to Thompson, who noted "at extremely low loads [<3 N, calculated from Thompson] the tissue deforms readily. After some moderate preload, however, the tissue becomes progressively stiffer" (34). In the reports that have concluded otherwise, deformation depth is a derived unit (rather than directly measured) by twice integrating accelerometer data. This means that the error inherent in accelerometer data is grossly magnified (5,8,22).

In addition to its role in providing local protection to the calcaneus, several authors have suggested that the heelpad provides significant impact absorption to the human skeleton during locomotion, particularly when running. Cavanagh et al. (5) have called the plantar surface a "biological shock absorber" but used methods that were unable to determine how energy was partitioned between the heel, foot, and leg. Paul et al. reported "significant shock absorber" action of the heelpad, "reducing the peak dynamic force transmitted by 20–28%" (21). However, their investigation was performed on the rabbit hind foot. The rabbit's method of locomotion does not resemble that of humans (humans are bipedal with a vertical skeletal axis during locomotion), and, unlike the foot of humans, the rabbit's hind foot is extremely large and possessing a thick heelpad relative to a low body mass. It seems unwise to apply a model validated on a rabbit hind foot in the hope of understanding how yielding tissues behave during locomotion in man. Our penetrometer data show that the depth of deformation at the heelpad reaches a plateau at a load of less than 9 kg (Fig. 7). At this modest load, the slope of the load-deformation curve becomes zero; thus, the heelpad loses its shock-absorbing properties and can be considered rigid (6). The area underneath the load-deformation curve is energy absorbed (6). This value is 0.3 J by our data and 0.01 J as derived from Thompson's data (34). There will be no appreciable increase in this energy, thus no increase in shock absorption, as the load increases above 9 kg. Based on the above, we conclude that the heelpad does not impart significant shock absorption to the body during locomotion, and particularly during running. These data are consistent with Light et al., who considered the shock wave at heel strike as being produced by the heelpad "bottoming"; thus, it has limited shock absorbing capability, which is easily overwhelmed (18).

Kuhns (17) has suggested that heelpad adipose tissue deteriorates with age. With respect to the relation between load and deformation, no evidence of deterioration was found in our experiments (Fig. 6).

The mechanics of heelpad deformation provide insight into how it protects locally during locomotion when unshod. The heelpad offers little resistance to deformation by fairly long pointed objects (e.g., length up to 15 mm; end diameter < 10 mm; from experiment 2) and would contain such objects within the void created by deformation, without perforation of the epidermis. Most of the load on weightbearing would be sustained by the surrounding heelpad. This protection would have had a significant selective advantage since infections which frequently follow puncture wounds would often be fatal to man prior to the era of antibiotics.

When considering blows to the heelpad from large objects (end diameter > 10 mm), the calcaneus would be protected by deformation constraint, which maintains large objects at a distance from the calcaneus (2.5 mm from the calcaneus for 10 mm-ended objects; 6.15 mm from the calcaneus for a flat surface; from experi-

ment 1) while disturbing the load over a wide area of the calcaneus through tension of trabeculae.

Plantar deformation patterns suggest facile accommodation to irregular weightbearing surfaces. When compared to locomotion with existing footwear, these mechanics of the bare foot may offer improved balance during locomotion. This is compatible with the preference of many gymnasts and dancers for being barefoot to wearing footwear.

It becomes apparent that the sensibility and mechanics of the plantar surface cannot be viewed separately. They interrelate in a way that can be seen as optimizing utility, within the constraints imposed by an evolving morphology and within the limited range of adaptation of biological tissues.

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