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Weight-Bearing Foot Reconstruction with Free Latissimus Dorsi Muscle Flap: Long-Term Clinical Results and Histological Changes

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ABSTRACT

This prospective study included two approaches; a clinical as well as histological study. The clinical study evaluated the long-term clinical and functional results after transfer of free latissimus dorsi muscle flap to the weight-bearing portions of the foot. The histological study investigated the light and electron microscopic changes that occurred in these muscles 9-11 months after transfer with a trial to find a correlation between the clinical state and the histological changes. Eleven posttraumatic patients underwent reconstruction of weight-bearing portions of the foot with 11 skin-grafted free latissimus dorsi muscle flaps. All flaps survived. Length of follow-up averaged 25 months. Time before full weight-bearing averaged 4.3 months. Debulking procedures were done for all flaps starting 9 months after the primary operation. Small superficial ulceration developed in 6 cases (54.5%) that healed spontaneously with conservative measures. Chronic ulcers developed in 2 flaps (18.1%) that necessitated surgical excision along with debulking of the flaps. All ulcers developed within the first 6 months after ambulation. All patients needed custom-made footwear. Gate alteration occurred in 8 patients (72.7%). Deep pressure sensation was perceived in all flaps. Histological study revealed a marked decrease in number and diameter of muscle fibers. The muscle fibers were greatly replaced by fat cells and fibrous tissue that divide the muscle into compartments. Great increase in the number and diameter of blood vessels was noted. In most remaining muscle fibers, myofibrils could not be identified; instead, they were replaced by myofibrillar fragments, free myofilaments, lipid droplets and lysosomes denoting obvious degenerative changes. In authors' opinion, these changes may transform the flap after long period into a tissue similar to some extent to the fibrofatty subcutaneous tissue of the sole. We conclude that skin grafted free latissimus dorsi muscle flap with adequate tailoring and tension over the defect is an ideal method for weight-bearing foot reconstruction. Patient selection, extensive education about foot care and frequent follow-up visits are essential to maintain healthy, intact flap and reconstructed foot.

INTRODUCTION

Treatment of soft tissue defects of the sole of the foot has been always a challenging problem to patients and surgeons. The capacity of sole skin to withstand pressure and shearing strains is related to its unique structure, which is characterized by a dense fibrofatty connective tissue layer stabilized by multidirectional fibrous septa that acts as a shock-absorbing system (Fig. 1) **[1]**. These characteristics introduce nuances to soft-tissue reconstruction dissimilar to those in other parts of the lower extremity. Early reports reviewed the results of skin grafts, local tissue flaps and cross-leg flaps **[2-5]**. For its unique characters, it has been postulated that the best way to reconstruct sole skin defects is to use like tissue in small sized defects **[4,6]**. However, sizable defects are not easily reconstructed by traditional methods. Only microvascular free flaps can bring sufficient well-perfused tissue to this difficult area. Controversy has arisen concerning the long-term stability of free flaps in reconstruction of the weight bearing portions of the foot. Some are convinced that sensible free fasciocutaneous flaps are essential **[7]** while others found no difference between sensory fasciocutaneous flaps and skin grafted free muscle flaps **[8,9]** concerning the long-term stability. Histological changes in the transferred free muscle flaps have attracted very few authors for study **[10]**. In this prospective study, we present our clinical experience with 11 free latissimus dorsi muscle flaps for reconstruction of the weight-bearing portions of

the foot. Long-term functional results as well as histological changes in the flap after its transfer to the sole of the foot using both the light and electron microscopy are presented.

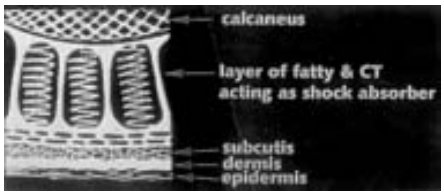

Fig. (1): Sketch of heel soft tissue structure **[1]**.

MATERIAL AND METHODS

Clinical study:

Between December 1997 and January, 2001, 11 free latissimus dorsi (LD) muscle flaps were performed for 11 male patients for the purpose of reconstruction of the weight-bearing areas of the foot in the Department of Plastic and Reconstructive Surgery, Zagazig University, Zagazig, Egypt (Table 1). Patients' ages ranged between 9-41 years (mean 24.2 years). In all patients, trauma was the cause following traffic accidents. The mean delay between the injury and flap transfer was 32 days (range 12-90 days).

Table (1): Site of the reconstructed defects.

Defect site	Number	%
Fore foot	4	36.4
Heel	7	63.6
Total	11	100

Surgical procedures:

After debridement of the recipient site, preparation of the recipient vessels was done. In all patients, posterior tibial vessels were the recipient vessels. Free LD muscle flap was then harvested with an overlying skin paddle for postoperative monitoring. The remaining area of the muscle was covered with a thick partial thickness skin graft. The thoracodorsal artery was anastomosed to the posterior tibial artery by end to side technique in 8 patients and by end to end technique in 3 patients. All thoracodorsal veins were anastomosed to the posterior tibial veins by end to end technique. Nerve suture was not performed. All operations were performed by a single surgeon (the first author). While microsurgical anastomosis was done, the assistant was closing the donor site. The donor site was closed primarily in all cases with insertion of suction drains. Skin grafting of the muscle was done immediately in 3 patients and delayed for 2 days in the remaining patients. The operative time ranged from 6 to 8 hours (mean 6.6 hours). Blood transfusion in the form of one or two units of blood was needed for all patients.

Postoperative care and follow-up:

Postoperative heparinisation was done for all patients for 5 days. Hospital stay ranged between 21 and 30 days. Elastic stocks were worn by all patients for 6 months to decrease postoperative oedema. Limited partial foot bearing was started 8 weeks after the operation and increased progressively to full weight bearing in 3-7 months (average 4.3 months). In the course of their postoperative rehabilitation, patients were extensively educated about foot and flap care. Length of follow-up averaged 25 months (range 14-48 months). They were followed up every 3-6 weeks during the first postoperative year and then every 3-6 months in the subsequent period. Follow-up included documenting foot pain, presence of ulceration, bulk of the flap, need for change of the footwear or special footwear, work status and estimated time of standing during an average day. Weight-bearing status was tested by making a footprint. The patient was asked to stand by both feet on a layer of gauze impregnated with black ink then to stand on a typing paper on a tile floor **[11]**. The pattern of ink left on the paper accurately reflected the portions of the foot that contacted the floor during standing. The patient was asked about the ambulatory distance before developing pain or discomfort, stair climbing, ability to run and use of ancillary support. Sensory evaluation was done regarding light touch, pain, deep pressure, temperature and static two-point discrimination. Side to side mobility of the flap over underlying tissue was tested by hooking a 100-gm weight to the center of the flap and measuring the yield of the flap in a vertical position. Gait was analyzed by asking the patient to walk for 50 meters and report any

change in the gait. Then, according to the patient satisfaction about his gait, a scale of normal, satisfactory or poor was put.

Histological study:

During harvesting of each LD muscle flap in the primary operation, a muscle biopsy of approximately 5x15x15-mm size was taken with the scalpel and used as control. During the debulking procedure 9-11 months after the primary operation, another biopsy of similar size was taken from the center of the flap. Each biopsy was divided into 2 parts to be processed for light and electron microscopy. For light microscopic study, the specimens were fixed in 10% buffered formalin solution, then, they were dehydrated in ascending grades of alcohol, cleared in xylene, embedded in paraffin, cut into 5 um thick sections and stained with haemtoxylin and eosin, PAS and trichrome stains. For electron microscopy, the specimens were fixed by emersion in 4% glutaraldehyde in 0.1 M phosphate buffer (ph 7.4) at 4°c for 24 hours. The samples were then washed in the buffer and post-fixed in 1% osmium tetroxide for 2 hours. After washing in the buffer, the specimens were dehydrated through a series of graded alcoholic solutions, infiltrated and embedded in epoxy resin. Semi-thin (1 um) and ultrathin (40-60 nm) sections were cut with ultramicrotome using glass knives. The semi-thin sections were stained with toludine blue and examined with light microscope. The ultrathin sections were stained with uranyl acetate and lead citrate and examined with JEOL JEM-100sx transmission electron microscope.

RESULTS

Clinical study:

Some clinical results are exhibited in Figures (2-7).

Flap survival and early postoperative complications:

All flaps survived and healed uneventfully. There were no complications stemming from the microvascular anastomosis. Seroma of the donor site occurred in 1 patient that was treated by reinsertion of a suction drain.

Bulkiness:

All flaps were bulky early in the course of follow-up (Fig. 2b). The bulk of flaps decreased progressively and became static at 6-9 months (average 7.8 months) postoperatively (Fig. 2c). All flaps needed debulking procedures to have good contours (Figs. 2d, 3c, 4b, 5b&c, 6b & 7b).

Side to side mobility of the flap:

Mobility of the flaps as defined above i.e. in terms of tangential shifting was decreased from 2.2-3.5 cm (mean 2.9 cm) to 1.1-1.9 (mean 1.5 cm) after debulking of the flap.

Ulceration:

Categories of ulceration (Table 2) included none, occasional (those seen early after reconstruction and self-resolving) (Fig. 2b) and chronic (those seen throughout the patients postoperative course and necessitating surgical excision) (Fig. 3b). All ulcers started to develop within the first 6 months after ambulation. All patients with early ulceration were reevaluated for protective footwear. In the 2 patients with chronic ulceration, they were evaluated to determine the cause of ulceration. In both of them, debulking of the flap along with ulcer excision was performed. Debridement of the 5th metatarsal head in one of them was needed to prevent recurrence of ulceration.

Table (2): Ulceration of the transferred flaps.

Type of ulcer	Number	%
Non	3	27.4
Occasional	6	54.5
Chronic	2	18.1
Total	11	100

Weight-bearing status:

Full weight bearing on the flap was recovered in all patients in 3-7 months (average 4.3 months). Before this date, all patients used ancillary crutches to avoid full weight bearing on the flap. One patient with associated metatarsal fracture developed pain on the start of ambulation. After further 2 months of non-weight-bearing, he was able to resume partial weight bearing and full weight bearing was resumed 7 months after the operation. When ulceration occurred, rest in bed and limited ambulation was needed till complete healing of the ulcer, then, the patient resumed ambulation. Special custom-made shoes with air-filled spaces in the pressure-areas were used by all patients. After debulking of the flap, the change of footwear

was mandatory for all patients. Regular slightly wider custom-made shoes were worn by all patients 4-6 months (average 5.2 months) after debulking. The first patient in this series has worn regular custom-made shoes for 35 months after the debulking operation without ulceration.

Footprint weight bearing study:

All patients showed abnormality in the weight bearing patterns comparing to the normal foot (Fig. 8).

Walking and work status:

All patients were able to walk, stand and climb stairs without rest for 1.8-4 hours (average 2.2 hours) without pain or discomfort in their last visit. Ability to run was limited in all patients. Four of our patients were pupils and there was no limitation of usual activity in the school in any of them. Jumping and running were refrained by them for fear of injuring the flap. Three patients were drivers and they returned normally to their work without significant complaint. Two patients were governmental employers and changes their jobs from jobs needing street activity to that with office activity. The last 2 patients were farmers who developed chronic ulcers in their flaps. After extensive education regarding walking, care of the flap and stress on wearing slightly wider shoes during routine agricultural work, they were able to return to work after longer periods than other patients.

Gait analysis (Table 3):

Acceptable gait means slight change from normal without obvious limping. Poor gait means obvious limping. It occurred in one patient who had amputated foot on the other side.

Table (3): Gait analysis in reconstructed patients.

Gait	Number	%
Normal	3	27.3
Acceptable	7	63.6
Poor	1	09.1
Total	11	100

Subsequent procedures (Table 4):

Debulking was performed for all flaps 9-11 months after the primary operation. Good debulking by re-elevation of the flap and recontouring with adequate tension and excision of the excess tissue was done in all patients without subsequent complications. The vascular pedicle was not sacrificed in any of our flaps.

Table (4): Subsequent secondary procedures.

Procedure	Number	%
Debulking of the flap alone	9	82
Debulking + excision chronic ulcer	1	9
Debulking + excision of chronic ulcer + debridement of the 5th metatarsal head	1	9

Evaluation of sensation:

All flaps had diminished sensation in comparison to the contralateral normal foot. Only one patient (9%) recognized light touch 23 months after the debulking procedure. Temperature and pinprick sensations were not perceived in any of our patients. Static two-point discrimination could not be identified by any patient. However, protective deep sensation could be felt by all patients.

Histological study:

Light microscopy:

Light microscopic (LM) examination of cross sections (CS) of control LD muscle at the time of harvesting of the flap showed a homogenous arrangement of muscle fibers closely applied to each other (Fig. 9). The longitudinally sectioned (LS) fibers clearly showed cross striations consisting of alternating darkly and lightly stained bands (Fig. 10).

LM examination of the cross sections of LD muscle flap 9-11 months after transfer revealed marked reduction of muscle fibers number and diameter (Fig. 11). The cross striations were not obvious in most remaining longitudinally sectioned muscle fibers (Fig. 12). The muscle fibers were greatly replaced by a large number of fat cells, fibrous tissue and numerous blood vessels. The remaining muscle fibers were arranged as scattered groups of fibers or single widely spaced fibers. The fibers became variable in diameter and degree of staining, some were pale and others were darkly stained (Fig. 11). The fat cells were arranged in groups separating muscle fibers. Although most fat cells were clear, some of them contained pinkly stained material similar to the staining of the muscle fibers (Fig. 13). The blood vessels were more numerous in the transferred muscle specimens in comparison to the normal specimens.

Both large and small vessels were frequently seen. There was increase in collagen deposition in the transferred muscle specimens. The collagen was condensed as thick septa separating groups of muscle fibers and fat cells. It was also condensed around all blood vessels especially the larger ones (Fig. 14).

Electron microscopy:

Electron microscopic (EM) examination of control LD muscle at the time of harvesting showed regularly arranged myofibrils filling the sarcoplasm of all muscle fibers except the regions of myonuclei. The sarcoplasm showed variability in electron density (lucent in some fibers and denser in others). The intrefiber spaces were very narrow (Fig. 15a). Few blood capillaries were observed in widened parts of interfiber spaces in close contact with adjacent muscle fibers. They had thin walls and relatively wide lumina (Fig. 16a). Neuromuscular junctions were frequently seen. The axon terminals had electron lucent cytoplasm containing mitochondria and synaptic vesicles. The axon terminals occupied sarcolemmal invaginations (Fig. 16a). Each myofibril consisted of very narrow I-bands alternating with wider and denser A-bands. The center of each A-band had a lucent H-zone traversed at its middle by a dense M-line. Each I-band was traversed by a very electron dense Z-line. The part in-between two consecutive Z-lines is the sarcomere. The myofibrils consisted of regular sarcomeres and were separated from each other by narrow intermyofibrillar spaces (Fig. 17a).

EM examination of the transferred muscles revealed that most remaining muscle fibers exhibited abnormal configuration. Moreover, the fibers, which appeared nearly normal at low magnification, showed myofibrillar changes when examined at higher magnification. While the normally appearing muscle fibers were filled with myofibrils, no myofibrils could be identified in the abnormal fibers (Fig. 15b). The abnormal muscle fibers had electron dense sarcoplasm, irregularly scattered fragments of myofibrils, free myofilaments, lipid droplets, lysosomes, vacuoles and numerous euchromatic myonuclei with prominent nucleoli (Fig. 18). Each myofibrillar fragment consisted of a Z-line and I-band with portions of adjacent A-bands (Fig. 19). The remaining normally appearing fibers had electron lucent sarcoplasm and numerous myofibrils. Most of these myofibrils appeared abnormal at high magnification and had irregular Z-lines, wide intramyofibrillar spaces and distorted myofilaments. Moreover, the observed non-distorted myofibrils had wider I-bands and longer sarcomeres than those of the control muscle fibers (Fig. 17b). The widened interfiber spaces contained large fat cells, fibroblasts, excessive amount of collagen fibers and blood capillaries (Figs. 15b, 16b, 20 & 21). The fat cells were distended with moderately electron dense lipid leaving only a narrow cytoplasmic rim adjacent to the cell membrane (Fig. 15b). In accordance with the marked LM vascular changes, EM examination revealed prominent changes on the level of blood capillaries. They became larger in diameter but their lumina were narrower than the capillaries of control specimens. Their walls consisted of abnormally thick endothelial cells presenting irregular luminal surfaces and connected to each other with tight junctions. Each capillary was surrounded with a prominent basement membrane, which split to enclose the pericytes. Fibroblasts and collagen fibers were seen adjacent to the capillaries outside the basement membrane (Fig. 16b). In contrast to the control specimens, neuromuscular junctions could not be identified in the transferred muscles.

DISCUSSION

In recent years, free tissue transfer either in the form of free muscle or fasciocutaneous flaps have become the corner stone in reconstruction of the weight-bearing areas of the foot, especially in gross posttraumatic defects **[1,9,12]**.

Like other reported series **[13-16]**, in this work, free latissimus dorsi muscle flap has been preferred for reconstruction of the weight-bearing areas of the foot for its known favorable anatomical features. It is large in size, so it can be tailored easily according to the size of the defect. In most people, the LD muscle can be harvested without loss of strength of upper extremity. The vascular pedicle is long and possesses vessels of compatible size to the posterior tibial vessels, which allows microsurgical anastomosis outside the zone of injury without the need for vein grafts **[17]**.

The high survival rate (100%) and the low rate of acute postoperative complication in the present study (donor site seroma in 1 patient) is comparable to previous reports **[12,14,15,16]**. The

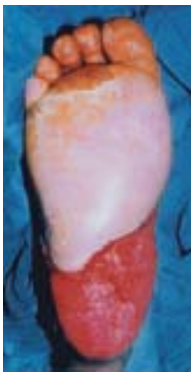

Fig. (2-A): Preoperative plantar view of a soft tissue defect of the heel.

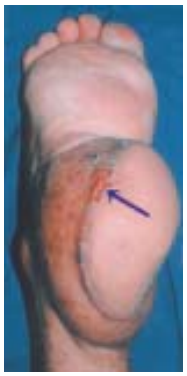

Fig. (2-B): View 10 weeks after transfer of free LD muscle flap with a small occasional ulcer (arrow) and marked bulkiness.

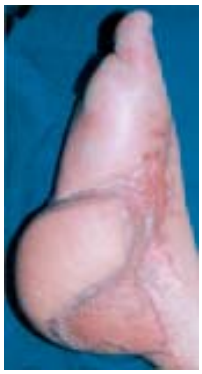

Fig. (2-C): View 9 months after the operation before the debulking procedure.

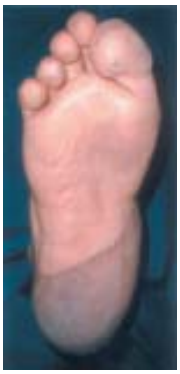

Fig. (2-D): Stable well contoured flap 35 months after debulking.

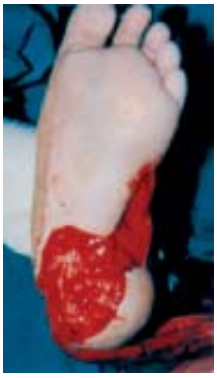

Fig. (3-A)

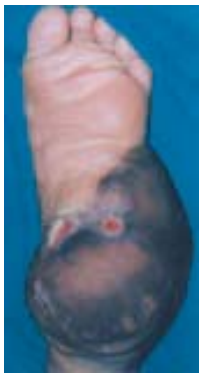

Fig. (3-B)

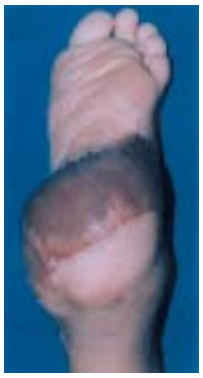

Fig. (3-C)

Fig. (3-A): Intraoperative view of a debrided defect of the heel with the free-transferred latissimus dorsi muscle flap before insetting.
(B): View 9 months postoperatively with the presence of bulkiness and small chronic ulcers.
(C): View 26 months after excision of the ulcers along with flap debulking with good contour and without recurrence of ulceration.

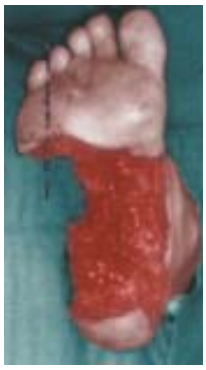

Fig. (4-A)

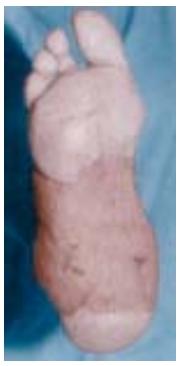

Fig. (4-B)

Fig. (4-A): Intraoperative view of a debrided defect of the sole. The little toe became completely ischaemic at the end of the operation that necessitated its amputation.
(B): Postoperative view 23 months after the debulking procedure with excellent contour and without ulceration.

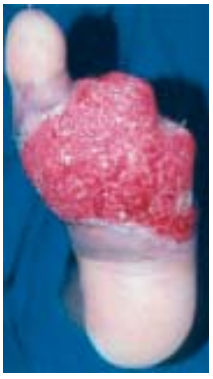

Fig. (5-A)

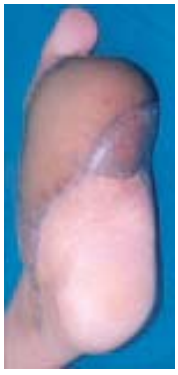

Fig. (5-B)

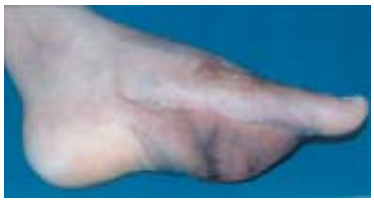

Fig. (5-C)

Fig. (5-A): Preoperative view of a defect of the forefoot.
(B&C): Postoperative views 5 months after the debulking procedure with good contour.

Fig. (6-A): Plantar view of a defect in the heel.

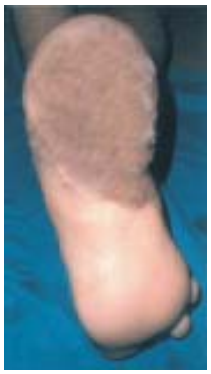

Fig. (6-B): View 11 months after the debulking procedure with a nice contour.

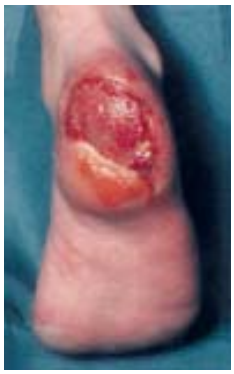

Fig. (7-A): Plantar view of a defect in the heel.

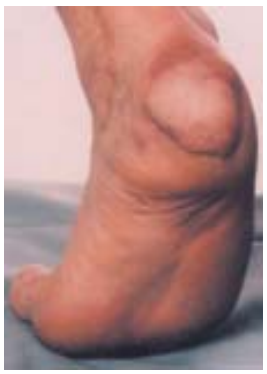

Fig. (7-B): View 15 months after the debulking procedure with a good contour.

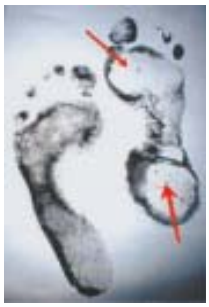

Fig. (8-A)

Fig. (8-B)

Fig. (8-C)*

Fig. (8-A,B&C): Footprints of 3 cases showing abnormal long-term weight-bearing pattern of the reconstructed feet (arrows). c* Amputated other foot.

LM microphotographs

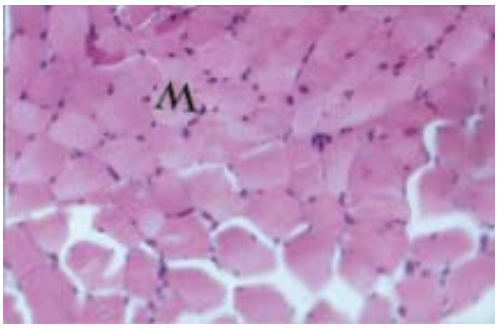

Fig. (9): CS of control LD showing homogenous arrangement of muscle fibers. (M) H&E X 200.

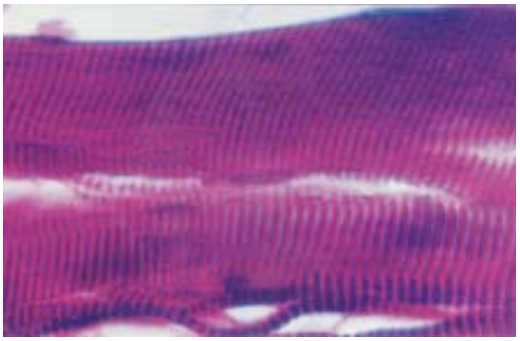

Fig. (10): LS of control LD muscle fibers showing normal cross striations. H&E X 600.

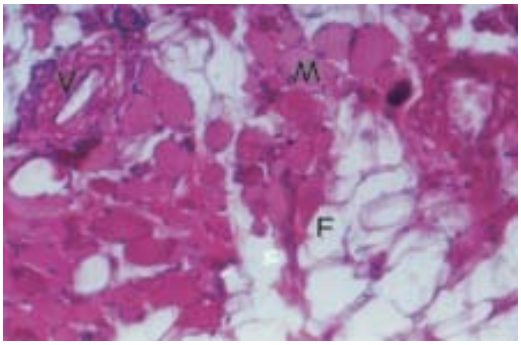

Fig. (11): CS of remaining transferred muscle fibers (M). The fibers are widely separated from each other and variable in size and staining. Fat cells (F), blood vessels (v). (PAS) X 200.

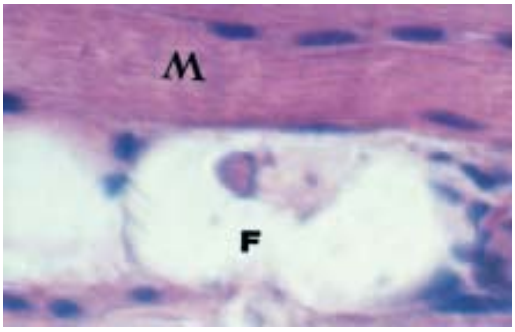

Fig. (12): LS of transferred muscle fibers (M) showing loss of cross striations. Fat cell (F). (H&E) X 600.

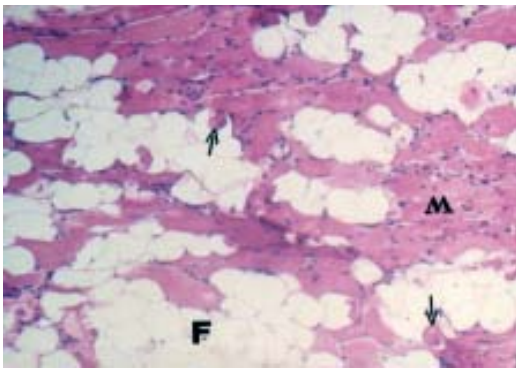

Fig. (13): Transferred LD flap showing groups of fat cells (F) and muscle fibers (M). Some fat cells contain pink material (arrows). (H&E) X 200.

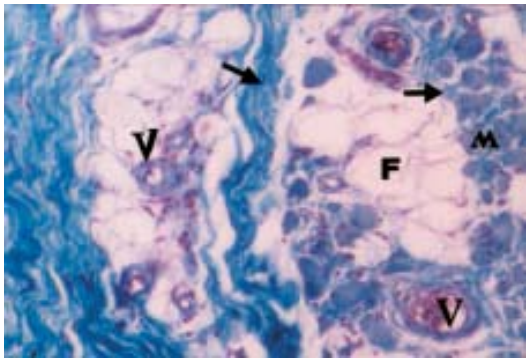

Fig. (14): Transferred LD muscle showing great reduction of muscle fibers (M). Fibrous tissue (arrows) is seen in-between fat cells groups (F) and around blood vessels (V). (Trichrome) X 200.

EM microphotographs

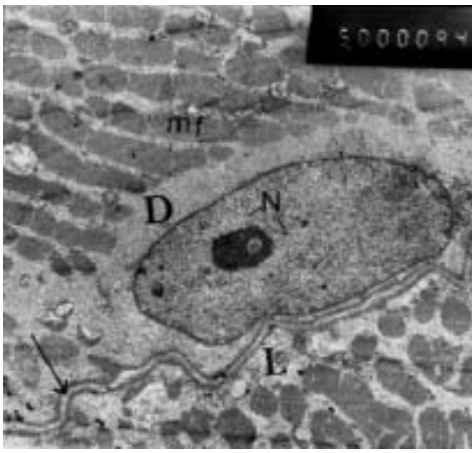

Fig. (15-A): Two adjacent control LD muscle fibers showing narrow interfiber space (arrow) and variable electron density of the sarcoplasm [lucent (L), dense (D)]. Nucleus (N), myofibrils (mf) X 5,000.

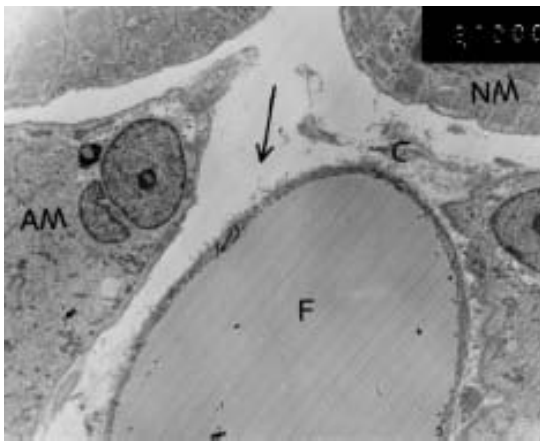

Fig. (15-B): LD 9-11 months after transfer showing normally appearing (NM) and abnormal (AM) muscle fibers. A fat cell (F) and collagen fibers (C) are seen in the widened interfiber space X 2000.

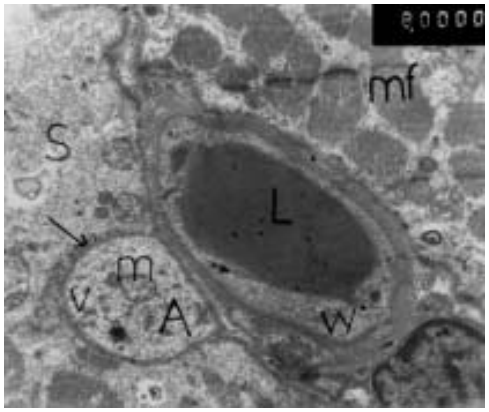

Fig. (16-A): Control LD muscle fibers. A capillary with thin wall (W) and wide lumen (L) is seen in the interfiber space. An axon terminal (A) containing mitochondria (m) and synaptic vesicles (v) is enclosed within sarcolemmal invagination (arrow) myofibrils (mf), sarcoplasm (S) X 8,000.

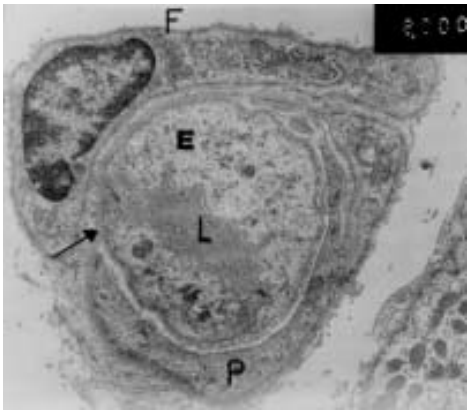

Fig. (16-B): Transferred LD muscle showing a blood capillary with narrow irregular lumen (L). Its wall consists of abnormally thick endothelial cells (E) connected together with tight junctions (t). The capillary basement membrane divides (arrow) to enclose pericytes (P). A fibroblast (F) is seen outside the basement membrane X 8,000.

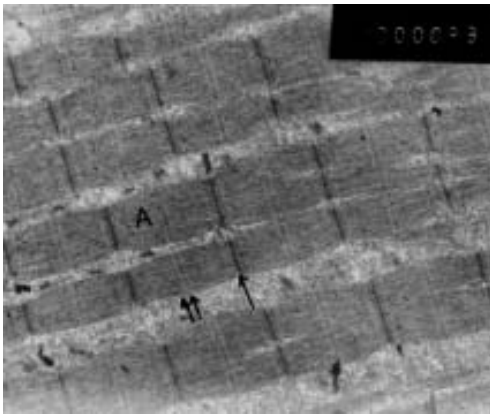

Fig. (17,a): LS of myofibrils of control LD muscle fibers. The myofibrils consist of I-bands (I) with very dense Z-lines (arrow) alternating with A-bands (A) with lucent H-zones containing dense M-lines (double arrow) X 10,000.

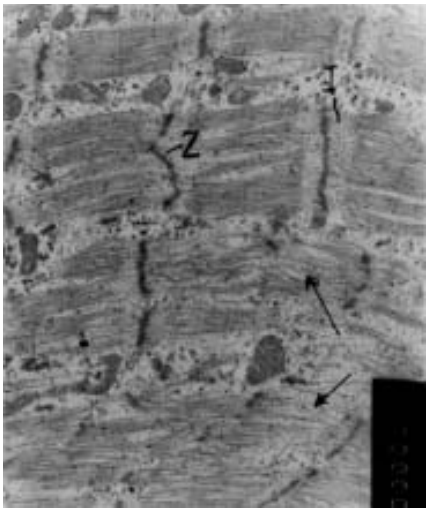

Fig. (17,b): A part of normally appearing transferred LD muscle fiber showing myofibrils with wide I-bands (I), irregular Z-lines (Z) and distorted myofilaments (arrow) X 10,000.

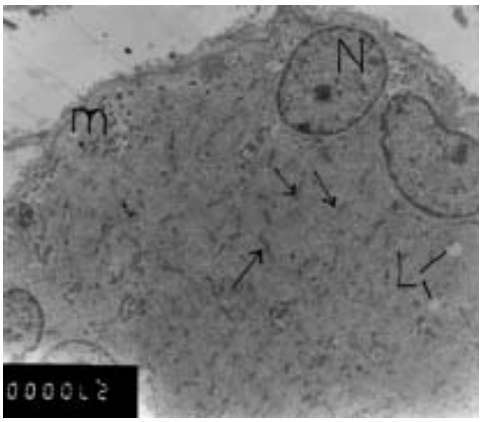

Fig. (18): A portion of an abnormal muscle fiber. Numerous scattered fragments of myofibrils (arrows) mitochondria (m), lipid droplets (L) and multiple nuclei (N) are seen X 2,700.

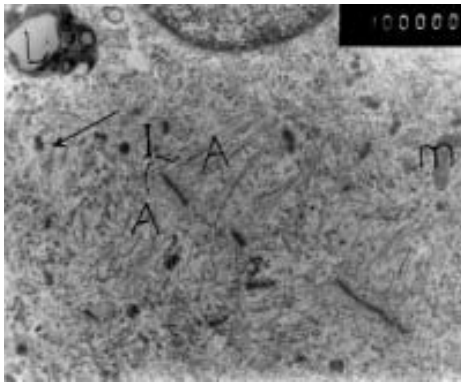

Fig. (19): A higher magnification of Fig. (15,b). Each myofibrillar fragment consists of an I-band and adjacent parts of A-bands (A). Lysosomes (L), free myofilaments (arrow) and mitochondria (m) are also seen X 10,000.

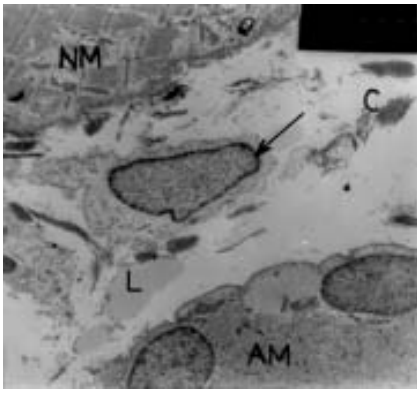

Fig. (20): Transferred LD muscle showing a fibroblast (arrow), collagen fibers (C) and lipid droplets (L) within a widened interfiber space between a normally appearing (NM) and abnormal (AM) muscle fibers. X 2,700.

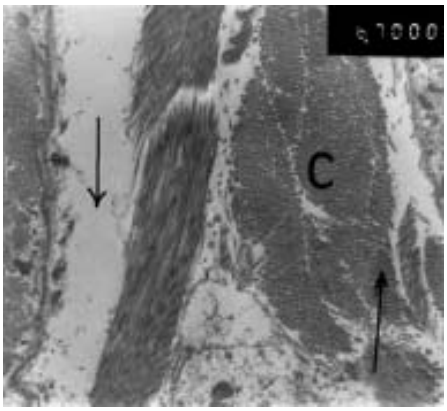

Fig. (21): An electron micrograph showing excessive collagen fibers (C) within a widened interfiber space (arrow) X 6,700.

high success rate reflects the high reliability of free tissue transfer in reconstruction of this extremely difficult area. In the view of this high success rate and the limited size of the reliable local flaps **[4,5,6]**, it can be said that this modality of treatment is the first choice in reconstruction of sizable defects of the pressure areas of the foot.

The oedema in transferred microvascular muscle flaps is common but poorly studied problem. Also, the extent of shrinkage of transferred non-innervated muscle flap is unclear. The amount of swelling and the rate of atrophy are unknown **[18]**. In our study, the flap reached its maximal bulkiness in the first month, then decreased gradually to become static after a mean of 7.8 months. Previous reports radiologically measured the free-transferred muscle thickness and found that the muscle swells a mean of 2.4 times their initial thickness in the first 2 weeks after surgery then subsides gradually to attain its initial thickness in 6-9 months with no further changes for at least 23 months **[19,20]**. The mechanism of oedema formation in transferred free muscle flaps is still unclear. Experimental studies have shown that after flap elevation, blood flow is reduced for less than 1 hour, probably because of vasospasm caused by surgical trauma, then stopped completely in the ischaemia period before flap reanimation by microvascular anastomosis. Subsequently, there is a long-lasting increase of blood flow, attaining its maximum in 48 hours. Vascular resistance decreases and arterial inflow and venous pressure increased. Denervated muscle flaps do not contract and venous pressure is still increased. This phenomenon lasts more than 8 weeks after surgery with increasing oedema **[21,22]**. Salmi et al. **[20]**, using colored duplex ultrasonography, found increased blood flow and decreased vascular resistance in the transferred free muscle flaps for a period up to 6 months. The blood flow and pressure changes are expected to be more in the muscle flaps transferred to the sole of the foot due to the dependant position of the foot with higher venous pressure. So, oedama and swelling in these flaps are expected to be more. For these reasons, compressive elastic stocks are recommended to decrease the swelling in the early few months after healing of the flap.

All flaps in our series were bulky and needed subsequent debulking procedures to improve

the contour to shoes. The bulkiness was more apparent in the area of the flap that is overlaid by the skin paddle than the skin grafted part but both parts needed debulking procedures. In other reports, debulking was needed only for myocutaneous flaps [1,13,16]. The need for debulking in all flaps of our series may be due to incomplete tension of the flap over the defect with subsequent redundancy that necessitated debulking later on. Inadequate tension was intended because we were too cautions to prevent jeopardizing the blood supply of the distal part of the flap. However, some authors recommended some tension on the flap during insetting to avoid this redundancy and bulkiness [13,15]. The great diminution of side to side mobility of the flap after the debulking procedures in our study supported this suggestion. Noever et al. [1] found that side to side mobility was more with musculcutaneous flaps than with skin grafted muscle flaps, a notice found in our study. So, this simple test may be used as an indicator for the need for debulking of the flap and creation of some tension on it during the debulking. However, this is better to be done after the flap becomes static in size and this is expected to be 9 months after operation. For these reasons, skin grafted free muscle flaps with adequate tension on the flap during insetting may decrease the need for debulking procedures.

The rate of ulceration in our study is in accordance with the previous reports [8,9,12, 13,15,16]. However, Harris et al. [14] reported higher chronic ulceration rate in a study done for children. Development of ulceration early after the start of ambulation may be due to the lack of durability of the transplanted muscle and overlying skin graft which is not acclimatized to the new weight bearing-function. By time, superficial ulcers heal, the grafted skin becomes durable and acclimatized to its new environment. Also, the better patient orientation with his new sole and care of the flap experienced by time adds to the decrease ulceration rate late after surgery. Another incriminated factor in ulcer development is inadequate muscle contouring due to lack of tension in the initial procedure with subsequent bulkiness [8,13]. In our study, no subsequent ulceration was noted after debulking of the flap and establishment of adequate tension on it in the revision procedure. This reinforces the principle that proper flap contouring should be performed at the initial procedure. Irregular bony architecture of the foot has been reported to be a crucial factor in the development of ulceration [13]. One of the chronic ulcers developed in our series was due to irregularity of the 5th metatarsal head that was treated by debridement with no subsequent ulceration. So, addressing any irregularity in the bony architecture of the foot and treatment of it is a central part in foot reconstruction to prevent ulceration. In conjunction with all previous factors, a suitable footwear plays an important role. At the start of ambulation, the weight bearing has to be gradual with the aid of crutches for some time. Special custom-made shoes with air filled spaces in the pressure areas were worn by all our patients. Change of the footwear was needed for all our patients at various stages of postoperative rehabilitation. Even after the patient started to wear usual footwear, we preferred it to be slightly wide and the patient had to be learnt how to care of the flap and his foot to avoid ulceration. This is also recommended by some authors [8,9,13,15]. In the view of the above mentioned factors, we agree with Potparic and Rajacic [9], that ulceration should not be taken as the only criterion for evaluation of the flap durability but all the above mentioned factors have to be put in mind in evaluation of an ulcerated flap transferred to the sole of the foot.

The goal of the surgeon reconstructing the lower extremity is to regain the final balance and interplay of structures that make possible the unique biomechanical functions of the foot which play a central role in both the stance (heel strike, foot flat, midstance and push off) and swing phases of normal ambulation. The elastic adipose tissue of the plantar soft tissue pad dissipates both vertical and transverse shear forces produced during stance phase [13]. By reconstruction of the plantar soft tissue pad, we aim at regaining a reasonable function of the foot as the weight-bearing organ of the body and an important part integrated in the gate. The ultimate purpose is to have a patient with a reasonable normal daily activity in standing, walking, working and climbing stairs without discomfort. The majority of our patients could regain the normal daily activity. Although 2 of our patients changed their jobs, we think that this was only due to the fact that they were governmental employers and the law gives them the right to change their jobs after the occupational accidents with the same income. Gold-

berg et al. [13] reported the decrease of the work hours in the minority of their patients. The only patient in our series who developed pain after the start of ambulation was found to have metatarsal fracture in the 5th toe and the pain completely disappeared after a period of rest and union of the fracture. In previous studies, radiological bony abnormalities were noted in those patients with pain during ambulation [8,13]. This proves the importance of addressing any skeletal injuries to be put in the plan of foot reconstruction. Functional restoration has been studied objectively by some authors [8,11,13]. We used a technique modified from the paint and paper tracing technique of Sommerlad and Mc Grouther [11] to measure the weight distribution and visual gait analysis because it is the only available method in our department. Although abnormal weight bearing distribution was found in all our patients, gait analysis showed either satisfactory or normal gait in most patients. This is due to the fact that all our patients, except one, had no injuries in the subtalar, ankle, knee or hip joints or other bone injuries. The joints and bones of the lower extremity play together the symphony of the gait. Potparic and Rajacic [9] reported a similar result of gait analysis. Vikaraitis et al. [16] found that by gait analysis using dynamic foot pressure measurement, in patients with musclocutaneous flaps, static load distribution on the reconstructed bare foot was nearly normal, but dynamic load distribution was pathological. In patients with muscle flaps, both static and dynamic load distribution were close to normal. However, Goldberg et al. [13] noted a correlation between standing foot pressure and gait patterns. They found that patients with abnormal weight-bearing distribution had major gait abnormalities. The contradiction between our results and Goldberg et al. [13] results may be due to the lower number of our patients and the absence of complex injuries in the extremities of them in contrast to that of their patients. The state of the other extremity plays an important role in the visual analysis of the gait. The only patient with poor gait in our study had amputated foot. Though the correlation between weight-bearing pattern and the visual gait analysis is less objective than the dynamic foot-pressure studies, it maintains the advantage of being able to observe the interplay of other parts of the lower extremity and hip girdle during the swing phase of ambulation while also observing the foot during the stance phase [13]. This is very important to be evaluated to improve the postoperative rehabilitation.

The aim of the secondary procedures done for all flaps in our series was either to treat a complication as chronic ulceration or to improve the aesthetic appearance and functional performance by reduction of the flap size for all patients to enable them to wear normal shoes. Other studies reported the need for other secondary ancillary procedures in the form of excision of ulcers, debulking, debridement of bones, correction of bony irregularities, osteotomies, fixation of fractures [9,12,13,15,16]. Some authors needed other flaps to treat severely ulcerating transferred flaps [9,23]. Like other series [9,12,13,15,16], no complications were found after the secondary procedures in all our flaps. These secondary procedures should be done without delay when indicated. The need for these secondary procedures can be decreased by addressing and treatment of all the bony abnormalities in the primary procedures. Also, adequate flap tension and use of skin grafted free latissimus muscle flap decrease the need for debulking procedures [13].

In the past, it was stated that cutaneous sensibility is necessary to prevent breakdown and ulceration in the foot [4]. The concept of using neurosensory flaps as free radial forearm flap was based on this premise [7]. On the same principle, experimental and clinical trials to increase the sensibility of transferred latissimus dorsi muscle flap to the sole of the foot by nerve coaptation of a sensory nerve to the motor nerve of the flap were done. Though sensation was found to be higher in reinnervated flaps, there was no significant effect on the functional outcome and the ulceration rate of the transferred muscle flaps [24,25]. Moreover, a clinical trial was done to compare non-innervated free muscle flaps with sensory to motor reinnervated muscle flaps and reinnervated free fasciocutaneous flaps. The higher sensation in the reinnervated free flaps has been found to have influence neither on the function nor on the rate of ulceration [9]. In our series, on reinnervation of the transferred muscle was done. Only deep pressure sensation could be experienced by our patients with only one patient perceived touch sensation. Similar results were obtained in previous reports with the similar functional outcome and rate of ulceration

[8,12,13,15]. So, we agree with the belief that recovery of deep pressure sensation only is sufficient for acceptable gait. Prevention of ulceration depends mainly on treatment of bone irregularities, proper tailoring and contouring of the flap, suitable footwear and patients education of foot and flap care **[8,12,13,15,16]**.

The present study investigated the long-term histological changes occurring in non-reinnervated free latissimus dorsi muscle flaps transferred to the sole of the foot by light and electron microscopic examination. Denervation related changes have been the subject of some experimental and clinical studies **[10,26,27,28]**. Despite classic observation that denervated muscle will atrophy, the extent of shrinkage of transferred non-innervated muscle flaps remains debated **[18]** and the explanation of flap bulkiness is still controversial **[10]**. In our study, muscle fibers were not only greatly reduced in number, but also diminished in size during the 9-11th month after transfer compared to intraoperative values. Similar observation was reported by Kauhanen et al. **[10]**. This is obviously due to the effect of denervation. Generally, it is believed that reinnervation is mandatory for regaining the initial size of the transferred muscle **[27,28]**. However, in an experimental study, regardless of timing and extent of reinnervation, a considerable amount of muscle atrophy was observed **[29]**. Nevertheless, it has been shown in the rat that sensory reinnervation is capable of preserving the same degree of muscle bulk as motor reinnervation **[18]**. However, Zhang et al. **[27]** found in an experimental study that neither motor nor sensory neurotization was significantly effective in regaining the original fiber diameter in the free flap model in comparison to the in situ model. This means that transplantation may alter the response of muscle to reinnervation.

The results of the present study confirmed that the main feature of muscle degeneration and atrophy is the replacement of muscle fibers by fat cells. The presence of lipid droplets in the remaining abnormal muscle fibers seen by electron microscopy can be considered as an early stage of the transformation of muscle fibers into fat cells. The presence of pink materials similar to the staining of muscle fibers within some fat cells seen by light microscope denotes a late stage transformation. The presence of few muscle fibers among numerous groups of fat cells indicates complete transformation of large groups of muscle fibers into fat cells. The transformation of muscle fibers into fat cells with the known capability of fat cells to expand may be a factor in the bulkiness encountered in our flaps in spite of the decrease in muscle fiber diameter. Although fat gives the tissue bulk needed in some cases for an acceptable cosmetic results in other areas of the body, this bulkiness will be a problem in an area like the sole of the foot where thin flap is preferred for the cosmetic appearance as well as for the function as the weight-bearing portion of the body.

The loss of cross striations in the longitudinally sectioned transferred muscle fibers observed by LM is clearly explained by the complete myofibrillar fragmentation in most remaining muscle fibers as observed by EM. The presence of different grades of staining of the muscle fibers and different diameters in light microscopic examination may be due to various degrees of degeneration of muscle fibers. This means that some fibers resist degeneration than others. This agrees with the results of Kauhanen et al. **[10]**, who reported a tendency toward type-I fiber specific atrophy with increasing proportion of type-II fibers. This is also confirmed in our EM examination that showed various degrees of myofibrillar degeneration. Two types of fibers could be identified according to the degree of myofibrillar degeneration and the density of the sarcoplasm; the normally appearing fibers and the abnormal fibers. The type-II muscle fibers have lucent sarcoplasm but the type-I fibers have dense sarcoplasm **[30]**. The degree of myofibrillar degeneration in fibers with lucent sarcoplasm was less than the fibers with dense sarcoplasm. This means that type-II fibers resist degeneration than type-I fibers as reported in the study of Kauhanen et al. **[10]**. Another postulation of the observed normally appearing fibers in EM study is that some muscle fibers may regenerate after long period as a result of spontaneous neurotization from the surrounding tissue. However, absence of neuromuscular junctions in our transferred muscle specimens excludes this postulation. The widened I-bands in the normally appearing muscle fibers is due to muscle relaxation as a result of denervation and this is also against the assumption of muscle regeneration.

In this study, excessive collagen deposition

was observed by light and electron microscopy. This was also reported in other studies [10,29]. Increased fibrous tissue within the transferred muscle may be multifactorial. Schultez et al. [31] found higher degree of fibrosis in experimental muscle flaps without neural anastomosis in comparison with transferred flaps with nerve suture. However, other factors as surgical trauma to the muscle and muscle ischaemia before revascularization can not be excluded as a cause of fibrosis because fibrosis was also reported to a lesser extent in reinnervated free muscle flaps [29,31].

One of the prominent observations in this study is marked increase in the vascularity of the transferred latissimus dorsi muscle flaps. The blood vessels were more numerous and larger in size than those of the control muscles. The same observation was reported by other studies [29,21]. It has been assumed that denervation with loss of muscle contraction will lead to increased arterial flow and venous pressure and decreased vascular resistance with consequent neovascularization to cope with these pressure changes [21,32]. The increase in size and wall thickness of the blood vessels can be explained by the blood pressure changes because the thoracodorsal artery (the pedicle of the flap) was anastomosed to the posterior tibial vessels in the most dependant part of the body with consequent higher pressure changes. This explanation is supported by the results of Stark et al. [33] who described subendothelial proliferation of smooth muscles as an adaptive process after vein grafting into the arterial circulation. The increased vascularity of the transferred muscle has its clinical importance. Healing of wounds would be expected to be rapid in this area of the body liable to trauma.

The histological changes occurring in the transferred latissimus dorsi muscle flap to the sole of the foot in the form of fatty changes, increased vascularity and fibrosis transform the flap into a structure nearly similar histologically to the subcutaneous tissue of the sole of the foot. This makes the non-reinnervated free latissimus dorsi muscle flap an ideal choice for reconstruction of the pressure area of the sole of the foot.

REFERENCES

1- Noever G., Bruser P. and Kohler L.: Reconstruction of heel and sole defects by free flaps. Plast. Reconstr. Surg., 78: 345, 1986.

2- Morris A.M. and Buchan A.C.: The place of cross-leg flaps in reconstructive surgery of the leg and foot. Br. J. Plast. Surg., 31: 138, 1978.

3- Woltering E.A., Thrope W.P. and Reed J.K.: Split thickness skin grafting of the plantar surface of the foot after wide excision of the neoplasm of the skin. Surg. Gynecol. Obstet., 149: 229, 1979.

4- Harrison D.H. and Morgan B.D.G.: The instep island flap to resurface plantar defects. Br. J. Plast. Surg., 34: 315, 1981.

5- Baker G.L., Newton E.D. and Franklin J.D.: Fasciocutaneous island flap based on medial plantar artery: clinical applications for leg, ankle and forefoot. Plast. Reconstr. Surg., 85: 47, 1990.

6- Hartrampf C.R., Scheflan M. and Bostwik J.: The flexor digitorum brevis muscle island pedicle flap: a new dimension in heel reconstruction. Plast. Reconstr. Surg., 66: 264, 1980.

7- Stock W. and Biemer E.: Sensible transplantate and der fussohe. Handchirurgie, 16: 215, 1984.

8- Milanov N.O. and Adamyan R.T.: Functional results of microsurgical reconstruction of plantar defects. Ann. Plast. Reconstr. Surg., 32: 52, 1994.

9- Potparic Z. and Rajacic N.: Long-term results of weight-bearing foot reconstruction with non-innervated and reinnervated free flaps. Br. J. Plast. Surg., 50: 176, 1997.

10- Kauhanen M.S.C., Salmi A.M., Von Boguslawsky E.K., Leivo I.V.V. and Asko-Seljavaara S.L.: Muscle fiber diameter and muscle type distribution following free microvascular muscle transfers: A prospective study. Microsurgery, 18: 137, 1998.

11- Sommerlad B.C. and McGrouther D.A.: Resurfacing of the sole: Long-term follow-up and comparison of techniques. Br. J. Plast. Surg., 31: 107, 1978.

12- Yucel A., Senyuva C., Aydin Y., Cinar C. and Guzel Z.: Soft-tissue reconstruction of sole and heel defects with free tissue transfer. Ann. Plast. Surg., 44: 259, 2000.

13- Goldberg J.A., Adkins P. and Tsai T.: Microvascular reconstruction of the foot: Weight bearing patterns, gait analysis and long-term follow-up. Plast. Reconstr. Surg., 92: 904, 1993.

14- Harris P.G., Letrosne E., Caouette-Laberge L. and Egerszegi E.P.: Long-term follow-up of coverage of weight-bearing surface of the foot with free muscular flap in pediatric population. Microsurgery, 15: 424, 1994.

15- Rainer C., Schwabegger A.H., Bauer T., Ninkovic M., Klestil T., Harpf C. and Ninkovic M.M.: Free flap reconstruction of the foot. Ann. Plast. Surg., 42: 606, 1999.

16- Vikaraitis S., Norkus T., Astrauskas T., Kaikaris V., Rimdeika R. and Averkina S.: Free musculocutaneous and muscle flaps for foot reconstruction: a clinical and gait analysis study. Europ. J. Plastic Surg., 23: 111, 2000.

17- Godina M.: Early microsurgical reconstruction of complex trauma of the extremities. Plast. Reconstr. Surg., 78: 285, 1986.

18- Dollen A.L.: Muscle sense or nonsense? Ann. Plast. Surg., 26: 444, 1991.

19- Salmi A., Ahovuo J., Tukianen E., Harma M. and Asko-Seljavaara S.: Use of ultrasonography to evaluate muscle thickness and blood flow in free flaps. Microsurgery, 16: 601, 1995.

20- Salmi A., Tukiainen E., Harma M. and Asko-Seljavaara S.: A prospective study of changes in muscle dimensions following free-muscle transfer measured by ultrasound and CT scanning. Plast. Reconstr. Surg., 97: 1443, 1996.

21- Clarke H.M. and Chen G.: Peripheral neovascularization of muscle and musculocutaneous flaps in the pig. Plast. Reconstr. Surg., 89: 109, 1992.

22- Hijortdal V.E.: Microcriculatory profile in myocutaneous island flaps: An experimental study in pigs. Scan. J. Plast. Surg. (Suppl. 24), 1992.

23- Caffee H.H.: Treatment of late ulceration in free muscle flaps to the foot. Plast. Reconstr. Surg., 103: 1247, 1999.

24- Goldberg J.A., Trabulsy P., Lineaweaver W.C. and Bunke H.J.: Sensory reinnervation of muscle flaps for foot reconstruction. J. Reconstr. Microsurg., 10: 7, 1994.

25- Hattori Y., Chuang D.C. and Lan C.T.: Sensory restoration of the skin graft on a free muscle flap: experimental rabbit study. Plast. Reconstr. Surg., 108: 132, 2001.

26- Becker M.H., Lassner F., Dagtekin F.Z., Walter G.F. and Berger A.: Morphometric changes in free neurovascular latissimus dorsi flaps: experimental study. Microsurgery, 16: 786, 1995.

27- Zhang F., Lineaweaver W.C., Ustuner T., Kao S.D., Tonken H.P., Campagna-Pinto D. and Buncke H.J.: Comparison of muscle mass preservation in denervated muscle and transplanted muscle flaps after motor and sensory reinnervation and neurotization. Plast. Reconstr. Surg., 99: 803, 1997.

28- Becker M.H., Wermter T.B., Brenner B., Walter G.F. and Berger A.: Comparison of clinical performance, histology and single fiber contractility in free neurovascular muscle flaps. J. Reconstr. Microsurg., 16: 525, 2000.

29- Kostakaglu M., Terenghi G., Manek S., Batchelor A.G., Polak J.M. and Green C.J.: Reinnervation and neovascularization in prefabricated free muscle flaps. Microsurgery, 16: 388, 1995.

30- Ustunel I. and Demer R.: A histochemical, morphometric and ultrastructural study of gastrocnemius and soleus muscle fiber type composition in male and female rats. Acta. Anat., 158: 279, 1997.

31- Schultez G., Gaggle A., Karcher H. and Kleinert R.: Histologic results of neuronal anastomosis of the microvascular latissimus dorsi transplant. Plast. Reconstr. Surg., 105: 526, 2000.

32- Sasmor M.T., Reus W.F., Starker D.J. and Colen L.B.: Vascular resistance consideration in free tissue transfer. J. Reconstr. Microsurg., 8: 195, 1992.

33- Stark G.B., Hong C. and Futrell W.: Rapid elongation of arteries and veins in rats with a tissue expander. Plast. Reconstr. Surg., 80: 570, 1987.