Dissertation on

STUDY ABOUT THE EFFECTIVENESS OF SERIAL STRETCHING IN POST BURN ELBOW AND KNEE FLEXION CONTRACTURE

M.Ch. DEGREE BRANCH - III - PLASTIC SURGERY

DEPARTMENT OF PLASTIC SURGERY KILPAUK MEDICAL COLLEGE CHENNAI - 600 010



THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI.

AUGUST - 2009

CERTIFICATE

This to certify that this dissertation entitled "STUDY ABOUT THE EFFECTIVENESS OF SERIAL STRETCHING IN POST BURN ELBOW AND KNEE FLEXION CONTRACTURE" is a bonafide work done by Dr.S.Ahamed Rafeeq Meeran, under our guidance and Supervision in the Department of Burns, Plastic & Reconstructive Surgery, Government Kilpauk Medical College, Chennai - 10, submitted for the M.Ch. (Plastic Surgery) Branch III examination, to be held in August 2009, by The Tamilnadu Dr.M.G.R. Medical University, Chennai.

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DECLARATION

I Solemnly declare that the dissertation titled "Study about the effectiveness of serial stretching in post burn elbow and knee flexion contracture" was done by me at Govt. Kilpauk Medical College & Hospital, Chennai-10 during November 2006 to March 2009 under the guidance and supervision of Prof.S.R.Vijayalakshmi., M.S., M.Ch., (Plastic Surgery).,

The dissertation is submitted to **The Tamilnadu Dr.M.G.R.Medical University** towards partial fulfillment of the requirement for the award of M.Ch., Degree (Branch-III) in Plastic Surgery.

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Date :

ACKNOWLEDGEMENT

I express my sincere thanks and gratitude to **Prof. V. Kanagasabai., M.D., Dean,** Kilpauk Medical College & Hospital, Chennai, for permitting me to utilize the clinical materials of this hospital.

I have great pleasure in thanking my Prof. Dr. S. R. Vijayalakshmi. M.S., M.Ch., (Plastic Surgery), Professor and Head of the Department, Department of Burns, Plastic and Reconstructive Surgery, Kilpauk Medical College & Hospital Chennai, for her valuable support in the conduct of the study and for her valuable guidance, suggestion and supervision throughout my career and my period of study. I thank my professor for being helpful in successfully completing this dissertation.

I am extremely grateful to **Prof. T.Mathivanan., M.S., M.Ch.,** (**Plastic Surgery**) who has given his full support and guidance for this study.

I thank all my professors, and Assistant professors who have helped me in this study. I am thankful to all my colleagues for their valuable help.

Finally, I would like to place on record my sincere thanks to all my patients for their immense co-operation without which this study would not have been possible.

Above all I owe my thanks to the ALMIGHTY for the successful completion of my study.

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INTRODUCTION

Human beings are unique creation of god, as they have an upper limb which is distinctly different from the lower limb. Evolution of human race has allowed us to have a complex amount of movements in the limbs. The hands are the eyes of the blind, the tongue of a dumb and the aid of the deaf to communicate. The upper limbs have to extend bend and hold. The lower limbs have to be straight, strong and move.

Burn injury is a systemic illness and its severity is usually assessed, if not by patient's survival, by the consequence of the burn injury i.e. scar hypertrophy, contracture and structural deformities due to loss of body components.

Body deformity is closely related to the magnitude of the injuries i.e. extend and depth of injury, mode of intervention, physiotherapy and follow-up care.

Formation of Scar tissue at the wound site and contraction of the scar tissue are the normal consequence of an injury. Although the exact mechanism accounting for the sequential change in wound healing and scar formation remain incompletely understood, wounds with infection and or allowed to heal spontaneously tend to form scar that are thickened and contracted circumferentially, mediated by various fibrogenic cytokines especially TGF β .

The thickened and contracted scar tissues, changes that are normal and expected consequence of wound healing process are microscopically composed of collagen, arranged in whorls and nodules. The changes may be observed as early as 3 to 4 weeks following the injury and they are cosmetically unsightly and functionally disturbing

Scarring secondary to burns leads to a multitude of adverse medical consequences including loss of function, restriction of joint mobility, restriction of growth, altered appearance and adverse psychological effect

When the upper limb is at rest, it relaxes the muscles, tendons and joint capsules and when prolonged the rest, leads to contracture and deformity.

The hands are no longer capable of full function and the mobile part of man becomes an unaesthetic prong from the body. Similarly when the lower limb is bend, the person is unable to be on his feet. Early mobilization and splinting does miracles, by not only restoring the anatomical shape and size but also retains the function.

The upper limbs which tried to rescue a burning victim needs the supportive care. Proper and timely care of the scar prevent, the formation of the deformity.

This study is an effect to find out the effectiveness of stretching the scar both in the upper and lower limb at the level of elbow and knee.

Though the act of stretching and splinting is tender, the results are dynamic in outcome.

AIM OF THE STUDY

To study about the effectiveness of serial stretching in post burn elbow and knee flexion contracture.

Objective:

- 1. To study about the amenability of the post burn scar to stretching.
- 2. Average time needed for full extension
- 3. Relationship between age of scar and time needed for full extension
- 4. Complications of Stretching
- 5. Effectiveness as an adjunct procedure in a patient with multiple contracture, while the more important areas are getting surgical treatment.

REVIEW OF LITERATURE

Biophysics of thermal injury:

Burns can lead to pyrolysis, disruption and oxidation of tissues. Among the various cell types present in the skin some are more likely sensitive to killing by elevated temperature than others. The degree of temperature elevation at the given site in the skin depends on the rate of heat transfer with in the tissue. The thermal conductivity of Dermis is greater than the subcutis, since fat is a good insulator. Perhaps for this reason thermal injury often leads to necrosis of the entire dermis with little cell death in the Subcutis.

Hair follicle in some sites typically extends well beyond the dermis, into the adipose tissue of the upper subcutis and eccrine sweat glands are also seen in the subcutaneous fat. Despite the presence of adipose tissue around them, often hair follicles are entirely destroyed even though there is little or no apparent necrosis in the upper subcutis. In severe burns the entire subcutis may become necrotic and cell death may occur in the underlying fascia and skeletal muscle or even in the underlying internal organs.



DEGREE OF BURN INJURY



First degree or superficial injury of skin

Superficial burns are those in which part or all of the epidermis is lost but the epidermal basal lamina remains intact and the dermis is uninjured. In these areas epidermal regeneration is required.

Second degree or partial thickness injury

In partial thickness wound, the entire epidermis and the upper part of the dermis become necrosed. The deep portion of the hair follicle remain viable and the keratinocytes lining the hair follicle become migratory, undergo mitosis behind the migrating cells, eventually covering the new epidermis. In severe cases loss of hair follicle may lead to insufficient regenerative activity to cover the surface.

Third degree or full thickness injury

Full thickness burns extend deep enough to destroy the entire hair follicle including the root and some subcutaneous tissue. In this case regeneration from the hair follicle is not possible and the wound can develop an epidermal covering only slowly, as the epidermis lateral to the wound spreads out over the wound surface. During this time the necrotic tissue in the wound bed is at risk of infection and extensive activity of tissue macrophage is required to remove it. Granulation tissue form beneath the necrotic dermal tissue and epidermal migration occur under the eschar formed by the dead tissue leading to restoration of the epidermis and production of dermal connective tissue in the form of thin scar.

Wound heals through three over lapping phases:

1. Inflammatory phase or Reactive phase

Coagulation system activation and formation of thrombus aids in haemostasis. Compliment system activation occurs, resulting in formation of inflammatory cytokines, recruitment of neutrophils, macrophages, resulting in removal of necrotic tissues.

2. Proliferative phase or Regenerative phase

This stage is characterized by the formation of granulation tissue, with massive proliferation of fibroblast and endothelial cells. These cells secrete fine fibers of collagen and ground substance formed of, by multiple mucopolysacchride and proteoglycan that make up the connective tissue matrix. Many peptides like TGF β and FGF and VEGF are involved in the formation of this connective tissue matrix.

3. Maturation phase or remodelling phase

Scar remodelling and contraction predominate during this phase. Wound tensile strength gradually increase as the immature disorganised collagen gets reorganised. Although the collagen content in a healed wound is maximum at 21 days, scar tissue is rearranged and replaced by more organised thicker fibers with more cross linking, to obtain a wound that has approximately 80% tensile strength of normal dermis by 6 months. The deposition of ECM and its component is regulated by MMPs and their counter regulatory inhibitors, tissue inhibitors of metalloproteinase.

Scar remodelling continue to occur for upto 12 months following the initial injury, with scar becoming soft, less vascular and less indurated. Scar tissue never achieves the tensile strength of normal skin. Fibroblast become myofibroblast which produces scar contraction.

When there is a tissue loss, wound heals by secondary intention. It is characterized by granulation tissue formation and wound contraction. Wound contraction pulls the nearby tissues towards the centre of the wound resulting in contracture.

MOLECULAR AND CELLULAR BASIS OF HYPERTROPHIC SCARRING

Chemical composition and organization of the extra cellular matrix *Collagen*

Collagen is the predominant extra cellular matrix protein in both normal dermis and hypertrophic scar, where it is responsible for the tensile strength for the tissue. However collagen constitutes a smaller proportion, about 30% less of the dry weight of hypertrophic scars because there are greater increase in other components such as proteoglycan and glycoprotein.

The major genetic form of collagen in skin and scar is type I, which assembles into thick fibrils, fiber and fiber bundles. In normal dermis there are smaller amounts of type III collagen, 15 % of the total, and very small amount of type V and type VI collagens. Pure type III & V collagens are assembled in vitro into thin fibrils but are found in tissues mainly in heterotypic fibrils mixed with larger amounts of type I collagen. Both type III & V collagen are considered to reduce the diameter of the collagen fibers of which they form a part.

Hypertrophic scar generally contains thinner collagen fibrils than normal dermis. The difference might be explained by the higher proportion of type III & V collagen, reported to be about 33% and 10% respectively. Type III collagen appears in healing wounds within few days after injury and its persistence in hypertrophic scars is probably a reflection of their biological immaturity. Type VI collagen does not assemble into fibrils but into thin beaded filaments, 5 to 20 nm wide, that seem to run perpendicular to the fibrils and link them together.

In the light microscope, collagen in hypertrophic scars is arranged in whorls or nodules rather than thick fibers or fiber bundles that are oriented parallel to the surface in normal dermis. In some specimens there are extensive regions of almost hyaline appearance where little organization of the fine fibered collagen is seen. In electron microscopy the narrow collagen fibrils in these region are seen to be more widely spaced than in normal dermis or mature scar and found to be ovoid or irregular in cross section. The interfibrillar space in fibrous connective tissue is occupied mainly by molecules of two other classes, the proteoglycan and glycoprotein.

Proteoglycan and glycoprotein

Proteoglycans influence physical properties of connective tissues such as turgor, resilence and resistance to compression, while glycoproteins such as fibronectin and tenascin are involved in cell-matrix adhesion, and have effects on cell behavior mainly through this mechanism. Proteoglycan also influence cellular activity, through a variety of mechanism including both positive and negative modulation of growth factor activity. The morphology of collagen fibers and their organization is affected by the nature and the amount of proteoglycans present in connective tissues.

Proteoglycans consists of one or more glycosaminoglycan chains, which are linear polymers of anionic disaccharides, covalently attached to a protein core. In glycosaminoglycans like, dermatan sulfate, Chondrotin sulphate, heparan sulfate and hyaluronic acid, one unit of the repeating disaccharide is an uronic acid.

Chemical analysis of the hypertrophic scar shows elevated levels of uronic acid and hence glycosaminoglycans. Since it is an anionic polysaccharide, glycosaminoglycans are mainly responsible for the water holding capacity of the connective tissues. So hypertrophic scars are hyper-hydrated relative to normal dermis or mature scars. However the increase in glycosaminoglycan content is disproportionately high relative to the increase in water content. Since the collagen fiber normally restrict swelling of connective tissues, the high concentration glycosaminoglycan in hypertrophic scar is responsible for their enhanced turgor.

Hypertrophic scars contain on an average only 25% of the total amount of small dermatan sulfate proteoglycan, decorin, the major proteoglycan in normal dermis and six fold higher concentration of large proteoglycan resembling versican. This versican is normally present in the proliferating zone of the epidermis, and is in association with elastin in the dermis. Decorin and versican detected by immunohistochemistry show a striking inverse distribution in the nodules. Decorin is implicated in the regulation of collagen fiber formation and in the organization of fibrils into fibers and fiber bundles. Another proteoglycan, biglycan present in lesser amounts than decorin in normal dermis is found at elevated levels in hypertrophic scars. Normally biglycan is found close to the cell surface but in hypertrophic scars it is associated with collagen in the extra cellular matrix.

The difference in proteoglycan proportions and distributions between normal dermis and hypertrophic scars could result from altered biosynthesis or altered dehydration, the former mechanism more likely. As hypertrophic scars mature, the collagen fiber become coarser and better organized and there is an increase in immunohistochemically detectable decorin. At about 12 months after injury many scars start to resolve spontaneously, with increase in number of cells expressing decorin suggesting that this proteoglycan may play an active role in the resolution. Mature scars show contents of collagen, proteoglycan and water that are indistinguishable from those in normal dermis.

Chemical analysis of hexose and sialic acid contents showed that hypertrophic scars contain elevated concentrations of glycoproteins, part of which is fibronectin. This extra cellular matrix molecule has effects on cell attachment and its activity, that is important in development and organization of hypertrophic scars.

Myofibroblast and delayed apoptosis in Hypertrophic scars

Myofibroblasts are identified by their positive staining for alpha smooth muscle actin and is considered pathognomonic for fibrous tissue, that is prone to undergo contracture. The reduction in cellularity that accompanies maturation of scar is associated with induction of apoptosis. The prevalence of myofibroblast in hypertrophic scar may actually a sign of delay in normal onset of apoptosis and this delay is responsible for the hypercellularity.

The extend and strength of interactions between the attachment dependent cells such as fibroblast and epithelial cells and the underlying extra cellular matrix are important factors determining cell survival. Detachment of cells often leads to apoptosis. In the anchored lattice, which resists deformation in all, but the short vertical dimension, fibroblasts continue to proliferate, while in floating or stress relaxed collagen lattice that are rapidly reorganized, they are triggered to undergo apoptosis. Reorganization of matrix may be impaired as a result of its chemical stabilization by cross linking, by tissue transglutaminase, an enzyme expressed in greater amounts in hypertrophic scar than by normal dermal fibroblasts.

TGF- β stimulates the proliferation of fibroblast and synthesis of extra cellular matrix proteins. It also stimulates the expression of smooth

muscle actin in fibroblast and may delay the onset of apoptosis. Like TGF- β , insulin like growth factor is increased in response to tissue injury. It is mitogenic for fibroblast and endothelial cells, stimulates collagen production by dermal fibroblast. Normally expression of IGF-1 in uninjured skin is restricted to epidermis, sweat and sebaceous gland, but in healing burnt tissues these elements are destructed and epithelial cells migrate to the wound surface could possibly secrete IGF-1 in proximity to fibroblasts and affect their activity.

Histology of hypertropic scar

Abnormal hypertrophic scar shows distinct difference from uncomplicated scar. The striking difference is the presence of rounded whorls of immature collagen that consist of delicate thin collagen fibrils rich in type III collagen, Small blood vessels and plentiful of mucopolysaccharide. These nodules are sharply demarcated from the scar tissue, which may be composed of similar materials or of mature thick collagen fiber, that are oriented parallel to each other and to the wound surface, typical of mature scar. Although they are clearly seen with routine h & e staining, these dermal nodules are distinctly seen with the movat stain which stain mucopolysaccharides, blue green and mature collagen fiber yellow orange. The nodule of hypertrophic scar vary in size from 0.5mm to 1cm in diameter and appear to be sometimes spherical, ovoid or cylindrical in shape. The abnormal dermal tissue is very firm almost like a cartilage in firmness and cutting properties. Both normal and hypertrophic scar are characterized by lack of elastin, which is also visible during movat stain. However there are often residual elastin fiber in the deepest part of the dermis below the zone of hypertrophic scarring.

In some hypertrophic scarring cases, very hard hypereosinophilic collagen fibers oriented parallel to each other but at varying angle with the skin surface are seen. In some cases such broad dense fibers dominate the wound. Generally they are surrounded by whorls of circularly oriented immature collagen, typical of hypertrophic scar.

Hypertrophic scar contains more of type III collagen and hyaluronic acid than normal flat scar. They appear more vascular and contain more T-cells, macrophages, langerhans and mast cell with high levels of circulating IG-E.

Immunohistochemical staining shows presence of smooth muscle actin, a contractile protein within fibrocystic cells in collagen nodules of hypertrophic scar. The sulfated proteoglycan of hypertrophic scar shows less decorin and more versican. Hypertrophic scar stain more for VEGF and also shows presence of large number of small nerve fibers. Recent studies show presence of bone marrow derived stem cells.

Hypertrophic scar fibroblast behaves differently from mature fibroblast. They secrete more collagen than normal fibroblast when stimulated with cytokines. Moreover there is more of TGF- β secretion in hypertrophic scar and normal apoptosis is delayed, shown by differential expression of apoptosis modulating protein, i.e. increase of antiapoptosis protein such as P-53.

Genetic analysis shows forty-four genes over expressed and 124 genes under expressed. Some notable gene over expressed include genes coding for collagen type X and XVI, thrombospondin-4 and matrix metalloprotein-16. Cultured hypertrophic fibroblast shows reduced response to IL-6 compared to normal fibroblast.

Contracture

The functional disability due to scar contraction is called contracture.

Causes:

- 1. Soft tissue loss, which heals by secondary indention and scarring
- 2. Skin grafting over the joint

- 3. Prolonged immobilisation without splinting
- 4. Congenital
- 5. Spastic paralysis
- 6. Arthritis
- 7. Myositis.

Burn injuries regardless of etiology, rarely involve a joint. However joint function is often impaired in burns due to inactivity, combined with limitation of joint movement due to scar contracture.

Holding bodily joints in flexion, so called position of comfort, a characteristic body posture is seen commonly in distressed individual. Although the exact mechanism is not clear, contracture of muscle fiber at rest and the contractile force difference between flexion and extension muscles may play a role in the genesis of this body posture. Prolonged period of inactivity associated with burn treatment and the scar tissue contraction around the joint structure, further impedes joint mobility.

The regimen of burn management, especially during the period immediately following the injury, seldom include plan for care of the joint. Instead the treatment is focused on the resuscitation, to ensure fluid balance and functional integrity of the circulatory and pulmonary system. The functional consequence of the joint dysfunction is usually left for later reconstruction.

The efficacy of splinting the joint reduces the incidence of contracture. Splints have to be given for a minimum period of 6 months to one year depending on the quality of scar and its maturation.

In response to changes in mechanical environment, chondrocytes alter their pattern of gene expression to remodel the extra cellular matrix. Excessive or insufficient mechanical joint stimulation can lead to cartilage degeneration. Dynamic loading of articular cartilage leads to enhanced synthesis of proteoglycan and collagen, while reduced loading and immobility leads to reduced synthesis of proteoglycan contents. Deprivation of mechanical joint stimulation leads to cartilage destruction as seen in paraplegic patient. Mechanical stimulation is necessary for metabolic activity of chondrocytes and maintenance of cartilage structure and its function.

The pathologic loss of ECM is produced by metalloprotease enzyme which degrades type II collagen and aggrecan, mediated by TNF- α and IL-1. Our body responds by increasing the protective proteins and reduce the rate of degeneration. CH13L1 is a protective protein which inhibit the cellular response to TNF- α and IL-1. G. Trudel and

coworkers have shown increased expression of CH13L1, the protective protein when the joint is immobilized.

Every body joint is susceptible to change. Of all the body joints, major joint contractures are common. Elbow contracture is the commonest and hip contracture, the least involved.

PREVENTION OF CONTRACTURE1) BODILY POSITION AND JOINT SPLINTING

Proper bodily positioning and splinting of joint structure must be incorporated into the regimen of burns treatment and it should be implemented as soon as the patient's condition becomes stable.

a) Acute Phase

Supine position is preferred, but patients can also be placed in lateral position. The head should be placed in neutral position with the head slightly extended, by placing a small pillow between the scapulae. Neck brace may be used if the patient is nursed in any other position.

SHOULDER JOINT

Shoulder joint is kept at $90-120^{\circ}$ of abduction and $15 - 20^{\circ}$ of flexion. This position helps in protecting the brachial plexus from traction injury and also in maintaining the stability of the gleno humeral joint.

This position is best kept, with the use of either a foam wedge or air plane splint. Figure of eight wrapping over a pad around the axilla during the intermediate period of recovery is effective in maintaining shoulder abduction and preventing shoulder flexion.

ELBOW JOINT

Elbow joint flexion is common if left unattended and maintaining elbow joint in extension is essential. An extension brace or three point extension splint across the joint is effective.

KNEE JOINT

Flexion of knee joint is another position commonly assumed by a burn victim and uncontrolled flexion of the knee joint will lead to exposure of joint structure. Maintenance of full extension of the knee joint is necessary to prevent it, and is maintained by knee brace or three point extension splint.

ANKLE JOINT

Planter flexion of ankle joint is common after burns and ankle should be maintained at 90° or neutral position by applying a posterior splint.

HAND

Hand management during the acute phase consists of elevation of upper limb to decrease edema and pain. Dorsal skin has great propensity for swelling due great laxity and edema, which drives the MCP joint into extension. If hand is maintained in this position for too long, the collateral ligaments gets shorter and tight.

Once the hand gets stiff with MCP in extension, the contractual forces of the shortened collateral ligament is difficult to overcome. With MCP in extension, increased tension on the intrinsic and extrinsic tendons drives the IP joint into flexion, thus hand assumes a position of MCP in extension and IP joints in flexion during the course of scar contracture and healing. If unchecked burnt hand contracture result in deformity called claw hand deformity, defined by wrist flexion, MCP hyperextension, boutonniere deformity with PIP in flexion and DIP in extension, and thumb adduction contracture.

Active and passive range of motion exercise is begun as soon as possible. Splinting should be done with hand in protective position, with collateral ligament of MCP stretched to 70 -90° MCP flexion. Wrist is maintained in 30° extension and IP joints in full extension and thumb kept abducted and opposed. Most hand therapy programs advise splint removal at least twice daily for motion exercise.

Postoperatively hand is splinted with k-wires or skeletal traction using Steinman pin through distal radius and attaching hayrake or banjo splint. When dorsum of hand is grafted maximum surface area is achieved in fist position with all joints flexed. While majority favour protective position, the fist position can also be used without adverse effect for the initial immobilization during immediate post grafting period, with resumption of standard rehabilitation after graft take.

b) Intermediate Phase Of Recovery

The period from the second month to fourth month following recovery is considered the intermediate period of recovery in burns injury. The burns victim typically has full physiological function and integumental integrity restored at this time. The cicatrial processes around the injury site are still physiologically active, though healing of the wound is considered satisfactory. At this time the rate of collagen synthesis is maximum and also there is a steady rise in the fibroblast, which produces contraction of scar tissue. Continuous use of splinting and pressure to support the joint and burned site is essential to control changes caused by the scar formation and contraction.

Joint splinting and body positioning are similar during acute phase of burn recovery. The shoulder kept in 15-20° flexion, 80-120° abduction. The elbow and knee joint in full extension, wrist in 30° extension and ankle in neutral position.

2) **EXERCISE**

Primary goal of exercise is to maintain joint functional integrity and muscle strength, attained by manually moving the joint actively or passively.

The need for therapeutic exercise to enhance mobility of the burn patient begins during the acute phase itself and continues through out the months of healing. Even though painful and extensive, exercise therapy is required during the long rehabilitation process. The results are for the most part depends on the patient and their families understanding, about the importance of exercise therapy, their involvement and dedication to the treatment.

The frequency and intensity of exercise regimen depends on the magnitude of the injury, and the extend of the joint involvement. The treatment if possible should be intensive and as frequent as possible.

The goals of the therapeutic exercise in burn rehabilitation are to.

- 1. Reduce the edema and immobilization stiffness
- 2. Maintain functional joint motion and muscle strength.
- 3. Stretch the scar tissue
- 4. Return the patient to optimal level of function

3) PRESSURE DRESSING

Pressure dressing helps in reducing the tissue swelling, in promoting softening of the scar and provides mechanical support to the joint. Compression of the burn wound, even though healing is still in progress, is most easily achieved by means of wrapping the extremity in elasticized dressing. Wrapping should begin at the hand or feet. The bandage is moved cephaloid in a criss cross fashion. The splint is reapplied over the bandage. It is important to rewrap the extremity 3 or 4 times daily. Wrapping an extremity with elasticized bandage can produce a pressure gradient of 10 -25 mm Hg, and it should be continued for 12 - 18 months.

Mechanism of Action

Kischer and Co-workers, reported a reduction in fibroblast content, total chondroitin-4-sulfate and cohesiveness of collagen fibres in pressure treated hypertrophic scars. Under electron microscopy they noted more rapid disappearance of the collagen nodules that are normally found in hypertrophic scar and reorientation of the collagen bundles parallel to the skin surface. Furthermore, large bundles of collagen fibers convert to smaller groups of fibers arranged in a less compact fashion. The reduction in size and thickness of the hypertrophic scar may also be related to a demonstrated reduction of intralesional mast cell numbers and consequent reduction in histamine production. Most of these changes are postulated to be due to local tissue hypoxia caused by occlusion of the microvasculature.

Baur et al., presented a different hypothesis and proposed that the decrease in capillary blood flow secondary to the pressure, and increased collagenase mediated collagen breakdown. They also found a diminished number of myofibroblasts in pressure treated hypertrophic scars. In addition, compression produces a reduction in tissue oedema with less ground substance production.

Beranek et al. on the other hand state that compression devices neutralize local venous hypertension by preventing a leakage of plasma proteins and by a subsequent amelioration of tissue oxygenation. Pressure is also said to accelerate maturation, decrease erythema, thickness and firmness of the scar.

More recently Krieger et al., proposed that the mechanism of pressure therapy is related to an elevation in skin temperature in the range of 1-3°C caused by the blockage of skin surface heat loss.

a) Elastic garment

Pressure therapy is applied by elasticated garment acting on skin surface with or without pressure padding. Lycra, a spandex fiber material

made of 80 -85% polyurethane materials is commonly used. Pressure therapy should be started early, within 2 weeks after burn wound or skin grafting area has healed. (Larson et al, 1974, Thomson et al, 1974, Leung et al, 1980, and Robertson et al, 1980). The garment should be worn 24 hours a day with short periods for needs of hygienic measures, and should be continued for at least 9 -12 months or till the scar fully matures. Pressure of atleast 24mmHg was considered necessary for effective therapy (Larson et al, 1974; Baur et al, 1976).

Pressure therapy has been demonstrated to be effective in inducing earlier remodeling of hypertrophic scar both clinically (Fugimori et al, 1971, Kischer et al.,1975, Baur et al 1976, Tully et al, 1980) bio-chemically, and mechanically (Tully, 1980). The effective external pressure is taken around arterial capillary pressure of 25mmHg (Larson et al, 1974, Baur et al, 1976).

Clinical improvements can be seen with 5 - 15mmHg. Recent studies have shown that pressure within safety margin of 35 -40 mm Hg, induce more rapid maturation of hypertrophic scar.

Variation with different sites of body relates to the compliance of the underlying tissue and the geometry of that area. Interface pressure tends be very high over bony prominence, in contrast to soft abdomen and buttock where pressure may be low or zero. Geometrically pressure over a convex area with small radius of curvature is higher than an area with large radius, although the garment tension appears same. Concave surface like presternal area, abdomen surface, inter scapular or sacral area, where there is change of surface from convex to concave tends to show zero pressure, no matter how tight the garment is stretched.

This observation is explained by Laplace law which states, Pressure=Tension/ Radius Of Curvature. Concave surface has negative radius of curvature and therefore will not be able to produce any interface pressure despite higher tension provided by the garment.

Continuous fall of pressure at garment scar interface is the result of marked viscoelastic property of the garment material and appears to be unavoidable, unless different material with more favourable extensibility properties are used.

Pressure treatment can be enhanced by

- 1. Regular check on the pressure under the garment using pressure transducer
- 2. Follow up of clinical response
- Tailoring adjustment of garment done regularly when found slack or pressure drops

- 4. New set of garment every month. It was found, 40% drop in the initial pressure after 4 weeks. Every patient is given 2 sets of garments, with 12 hourly change to minimize time dependent drop in pressure
- 5. Garments are worn 24 hours a day, except for hygienic measures and it should be continued for at least one year or until the scar fully matures.
- 6. Appropriate pressure padding to increase the effective convexity of the scarred area. For concave area pressure padding is given to change the effective radius of curvature from negative to positive convex one.

Nilufer Yidiz advices to mark rectangle is on the garment. The stretch needed to obtain the desired compression, of around 25mmHg, should also be marked, a new technique to give adequate pressure, by pressure garments.

Materials available for pressure padding are, soft materials like polyurethane foam (Fujimori et al, 1968) harder material like plastozote, sansplint and orthoplast (Larson et al, 1971, Thomson, 1974, Tolhurst, 1977) are all useful. For even scarred surface elastomer works well by filling up the contour. Theoretically pressure that exceed capillary pressure of 24mmHg is required. However, good clinical results are reported with levels as low as 5 -15 mmHg. Reid and co-workers stated that 15 mmHg is required to accelerate maturation process, and the effects of pressure below 10mmHg are minimal. Pressures above 40mm Hg may produce maceration and paraesthesia .

Undesirable side effects of lycra material include,

1. Anisotropic property resulting from unidirectional lay of the elastic element producing different elastic properties at right angles,

- 2. Non linear response to stress
- 3. Time dependent fabric elastic deterioration
- 4. Abrasive and non absorbing nature of nylon kit component causes intolerance, blistering problem in certain patient, especially in hot humid weather.

b) Silicones

Silicones are synthetic polymers based on dimethyl siloxane monomer. These contain repeated units of (Sio(CH3)2). They therefore have a silica derived backbone and organic groups as SiOC chains attached directly to silicon atom via silica carbon bonds. In wound care and rehabilitation three types of silicones are used.

- 1. Silicone fluids-short unbound ,straight PDMS chains.
- Silicone gel –lightly crosslinked PDMS chains usually formed in the presence of catalyst.
- 3. Silicone elastomer –long and strong cross-linked PDMS chains also formed in the presence of a catalyst, usually silica.

Depending on the amount and type of catalyst, the final product can differ in physical and chemical properties.

In burn wound care the history of use of silicone dates from early sixties, where silicone fluids were used as immersion treatment for burns patient. This method promoted complete separation of eschar, early formation of granulation tissue and early joint motion of a spontaneous healing or grafted burn wound. This method was particularly useful in hand. Unfortunately their use was stopped, when impure industrial grades were used to augment soft tissues, and was banned.

Silicone can be applied without use of pressure, or with pressure as pressure or position device. Pressure pads are individually made using elastomer, putty, or foam and is fitted directly on the patient. They are worn in combination with classical pressure garments, masks and splints. Currently these insert can be custom made based on an imprint of the scarred limb or body part. This elastomer helps in positioning the limbs and fingers in splint.
Remarkable evolution during the last decade was its use as drug delivery medium. This applies either in treating hypertrophic scar with vitamin-E or even burns wound with topical antimicrobial agents. Now silicone coated polyamide dressing are used for skin graft fixation.

Mechanism of Action:

Various mechanism of action has been proposed. In 1985,Quinn et al found no effects with regard to pressure, change in scar temperature and difference in oxygen tension within scar. They found that hydration of the skin was altered with the sheets, as water vapour loss was of one half of normal skin and they concluded that the stratum corneum provided a reservoir for fluid. Scars have greater trans-epidermal loss of water than normal skin. Reduction in water vapour loss has been postulated to decrease capillary activity, reducing collagen deposition and scar hypertrophy. Beranek suggested that hydration of horny layer results in increased permeability of water soluble compounds, permitting a diffusion of components of inflammation towards the skin surface, which helps in scar maturation.

In 1987, Quinn thought release of low molecular weight silicone would enter stratum corneum and affect the scar maturity. This was supported by the work of Shigeki et al . Sawada suggested hydration and occlusion as principle mode of action of silicone gel sheet and the

presence of silicone is not essential for obtaining clinical benefits. This opinion was supported by the work of Chang and co-workers, who found hydration significantly inhibited the fibroblast proliferation in vitro.

Ricketts et al., found molecular evidence for extensive connective tissue remodelling occuring during occlusive dressing therapy. James et al., demonstrated short-term topical application of tap water on the skin significantly influence its properties, especially of the epidermis.

Hydration should also benefit joint motion when used over a burn wound contracture. This beneficial effect may be due to diminished stress on the tissue.

Hirshowitz et al., proposed silicone sheets produce a static electric field from friction, and this field was responsible for the scar maturity. As hypertrophic scars are associated with increased mast cell, it was suggested that electric stimulation reduces both vascularity and mast cells, thus reducing scarring.

Skin temperature elevation occurs due to blocking skin heat loss and it may also play a role in scar tissue metabolism

Minor complications such as rash, ulcer, erythemia and pruritis do however occur more regularly in children, especially when the dressing is kept in place with pressure garments or adhesive tape. These minor complications usually disappear when the therapy is stopped temporarily or the duration of the therapy is reduced or when hygienic measures are taken.

Most publications support the fact that hydration and occlusion improve both clinical parameter (colour, pliability and thickness) and subjective symptoms such as pain and itching. This is probably also the reason why other occlusive dressing like hydrocolloid, glycerin based gel and occlusive tapes have been introduced for treatment for hypertrophic scar.

c) Combination modalities

The most common combination is that of silicon silastic sheet, gelsheet or pad with a classical pressure garment, using the previously mentioned manipulation technique. Silicone orthosis and garments with varying degree of stiffness or rigidity can be made. The latest development in this regard is that of inflatable silicone insert to treat scars (ISIS) in which the pressure on the scar can be adjusted by means of a pump.

The main advantage of this treatment is that two therapeutic techniques can be combined.

The working mechanism of the combined therapy is evident in that the presumed working mechanism of the individual modalities, pressure, hydration, occlusion and static electricity may combine and reinforce each other.

Eric van den kerckhove and co-workers have suggested that the key to success of the therapy, is by ensuring adequate hygienic precautions. They recommend initial duration of 12 hours treatment per day, particularly when combined with pressure, on children or in warm weather or climate. Further strict guidelines are followed for cleaning and possible disinfection of both the product and the skin to avoid irritation of healthy skin.

4) Burns Scar Massage

Once the scar has matured enough to tolerate sheering forces, scar massage can be started. It aids in softening and remodeling of the scar tissue, to become more elastic and stretchy, thus improving joint mobility. Initially non frictional massage by applying pressure to blanch the scar and mobilization of the skin surface, without friction is done. As the patient begins to tolerate frictional massage, scar tissue can be manipulated with, rotatory, parallel and perpendicular motions using a lubricant. Clinically massage is found to alleviate pain and itching, and can be used for desensitizing purpose. Massage should be done 3 -5 times for 5- 10 minutes.

Other method for scar management is by heat therapy. Heat relaxes tissues and makes them pliable and prepares it for mobilization. Heat modalities include hot packs, paraffin wax, fluid therapy and ultrasound. Heat therapy is rarely used in burns rehabilitation.

TREATMENT OF CONTRACTURE

Elbow and Knee contracture

Flexion deformity is the most common deformity encountered in these joints. The scar formed across the cubital fossa and popliteal fossa frequently aggravates the contracture problem in these joints. The following techniques are frequently used before surgery, to obtain joint movement and joint extension.

1) Serial Casting

Serial casting is frequently used in correction of significant contractures. Joints with over 30° of contracture respond well to casting. The applied cast provides total contact with circumferential and evenly distributed pressure. Casting is a relatively simple, fast and painless intervention and provides an alternative to dynamic splinting, when patients compliance is an issue i.e. in pediatric age group.

Plaster casts are inexpensive, light weight, easy to fabricate and allows for ventilation down to the skin surface, as plaster is porous when cured, thus avoiding skin and wound maceration. Disadvantages of this technique include, decreased resistance to water and will break if not constructed strong enough to withstand patients own muscle strength.

Fiberglass casts are alternative to plaster cast and are fast setting when reacting with water, light weight and are stronger. Because of the fiber abrasive properties, therapist should wear gloves prior to handling it. Recently non-latex polyester materials such as deltacast, are used as alternatives to plaster of paris and fiberglass, as they are light weight and conform well because of their elastic properties. When casting is applied the patient should feel a gentle stretch. The first cast should be removed at approximately 24 hours and thereafter could be applied up to a week time.

2) Splinting

The prolonged gentle stretch aids in tissue elongation without causing micro trauma. Low-torque and long-duration repeated stretching leads to a greater restoration of range of movement with more normal mechanical properties compared to high-torque and short duration stretching - M. Ustuba, et al.

Mechanical properties of elasticity and percent extension or strain were thought appropriate to ascertain any scar maturation. However normal skin is anisotrophic and viscoelastic, thus its properties are directional dependant and load strain rate dependant. The load extension curves of normal skin display an initial compliant phase, during which large extension was produced by applying a low load, less than 100g/cm width, followed by progressive stiffening with increased extension. The time dependent initial behaviour is associated with the response of the elastin content of the skin, whereas stiffening is the result of the collagen content resisting stretching after an initial realignment phase of these fibers. High grade scar subjected to smallest load stiffen abruptly displaying a very different response to that of normal skin, suggesting a different fiber arrangement.-J.A.CLARK et al.

Dynamic extension or flexion splint may be utilized to provide prolonged gentle, sustained stretch for correction of contracture.

Three point extension splint is assembled like prothetic or orthotic device. It has two-side bars hinged at the middle with the bracing trough at the end. A cap pad is attached at the midsection of the sidebar to fit over the elbow or the kneecap. The splint is placed across the antecubital or popliteal fossa. Fitting of the splint is adjusted using Velcro straps.

Extending the amount of movement of the joint is determined by the extend of the preexisting joint stiffness. The magnitude of extension is controlled by tightening the olecranon or patella pad. It is increased gradually as the joint gains its mobility. Problems are uncommon, however breakdown of the skin can occur.

3) Lateral Suspension And Skeletal Traction

Reports of Larson and Evans describe the use of lateral suspension for positioning and for extremity elevation and skeletal traction for prevention and treatment of contractures. Harner and Youel report deals mainly with the management of burns with skeletaly anchored digital traction splint like, banjo, halo and hayrake external fixator. Traction is altered by changing the traction weights. In the suspension mode the distal weight should be such as to maintain the desired position of the extremity, when the patient is sleeping or inactive, but is not so great to prevent active motion. In the traction mode, weight must be sufficient enough to correct a contracture or to maintain the surgically gained positioning.

Skeletal traction requires percutaneous insertion of Steinman pin through the radius, for elbow joint and through the tibia for knee joint. The pin is inserted through both the cortex at the junction of proximal two third and distal third of the radius or tibia. A contracted joint can be mobilized by the continuous and constant pull on the long bone by a gravitational force generated by a 10 -15 pound weight placed on a pulley device.

For a flexion deformity of the elbow, the patient is placed in the supine position and the pulley traction device will provide a horizontal and then vertically downward pull. For knee contracture, patient is placed prone and weight placed around the ankle.

SURGICAL TREATMENT

Pre-Surgical Evaluation

The following features are assessed

- 1. Extent of contracture
- 2. Magnitude of scarring and scar thickness
- 3. Location and size of the uninjured skin
- 4. Point and axis of joint rotation

1) Incisional Release

The incisional release is in alignment with the axis of joint movement. The contracted joint is freed by making an incision in the scar across the joint surface. Prior infiltration with lignocaine containing 1:400000 adrenaline is useful in obtaining haemostasis and later pain control. This must be made with caution to avoid injury to major vessels and nerves. In rare instances scarring could involve capsule and reconstruction may be necessary. After contracture release wound is covered by the following techniques,

- 1. Partial thickness skin graft
- 2. An interposition skin flap mobilized from adjacent area.
- 3. Combination of interposition skin flap and split skin grafting
- 4. Muscle or fasciocutaneous or myocutaneous flap mobilized from adjacent area
- 5. Free Skin flap or fasciocutaneous or myocutaneous flap harvested from a distant site and transferred via microsurgical technique.

Skin Grafting technique

Skin grafting is technically simple and results in minimal morbidity. A partial thickness graft of 15/1000 to 20/1000 inch thickness is harvested from an unburned area. Thighs, leg and scalp are the common donor sites. Full thickness skin graft can be harvested from inguinal region, post auricular area or supraclavicular area, without leaving unsightly scar. The donor defect is closed primarily. The graft is cut to fit into the defect, and the edges are anchored with sutures. The suture ends are left long to tie over a bolster, to immobilize the graft. Several quilting sutures are placed in the center of the graft to immobilize the graft against the bed. Haemostasis around the recipient site is essential as haematoma found underneath the graft will hinder its take.

The bolster is removed on the 4th or 5th day after the procedure. Bodily fluid or blood accumulated under the graft i.e. seroma or haematoma should be evacuated, by making a small nick in the graft. The fluid is rolled out with a cotton tip applicator. The joint is immobilized immediately and the pressure dressing is used to minimize the consequence of contracture. Physical exercise is resumed 3 weeks after the surgery.

Good post operative care is given to the grafted area and also to the donor site. Liquid paraffin massage is given to the grafted skin to prevent dryness of the graft and it also helps in graft maturation. Grafted area has to be splinted till the graft matures, to prevent recurrence of contracture.

2) Ilizarov External Distraction:

Ilizarov distraction technique is very useful in severe contracture were surgical release and cover is not possible. In this method Illizarov external fixation frames are built around the elbow joint contracture or knee joint contracture. The frames are fixed perpendicular to the diaphyseal axis of each segment. Olive wires are used for stabilization and to prevent drifting of bone. The proximal and distal Ilizarov rings are interconnected by lateral and medial hinges, taking into account the correct axis for the movements of the joints.

Additional threaded long push rods are placed on the posterior aspect of the rings in case of knee joint contracture, and in the anterior aspect in case of elbow joint contracture. These rods help in gradual distraction of the contracture scar.

This method does not add scars as do distant flaps, and the bulkiness typical to the flaps in the recipient area is also avoided. Caution is needed while introducing the pins as neurovascular damage can occur.

Slow distraction helps in regaining full extension of the elbow and knee joint without complications.

POST-STRETCHING









MATERIALS AND METHOD

From the patients admitted, or attending the out patient department, detailed history about the following are taken,

- 1. Information about the nature of the injury
- 2. Date of the injury
- 3. Treatment history of the wound
- 4. Previously done Surgical procedure
- 5. Whether splinting was done while wound was healing and after wound has healed and,
- 6. Follow up care

Local examination of the joint include assessing the,

- 1. Extent of the scar
- 2. Maturity of the scar.
- 3. Presence of blister, raw area, ulceration or scar breakdown, if present is noted.
- 4. Degree of Contracture.
- 5. Active and passive range of joint mobility
- 6. Condition of the proximal and distal joints and,
- 7. Associated other deformity

POST - STRETCHING





POST - STRETCHING WITH STATIC SPLINT





ASSOCIATED DEFORMITY INTER THIGH ADHESION



Investigations like, X-ray of the joint is taken to assess the joint condition and to rule out heterotopic calcification. Other routine blood and urine investigations are done to assess the general condition of the patient.

After ruling out heterotopic calcification and joint abnormality, informed consent about the therapeutic procedure is obtained. Gentle massage to the scar is started using liquid paraffin and the joint is mobilized passively, by fully flexing and extending the joint and the maximum extent of the extension is noted. Dry absorbable dressing with good padding is given to the scar and firmly bandaged. Plaster of paris splint is given and the joint held in maximum extendable limit till the plaster of paris hardens. Distal part should be looked for any neuro vascular compromise. Maximum extendable limit of the joint is noted by the pain tolerability and by the extension initially obtained passively, before the application of the splint.

Paracetamol tablet is given to give symptomatic relief to the patient. After seven days or when the patient is comfortable without pain, the dressings are removed and any complications like blistering, scar break down, if present are noted. Blister if present should be allowed to settle on its own by puncturing with a sterile needle and letting out the fluid. If scar break down has occurred stretching is stopped temporarily and the splinting is continued in a slightly lesser extension till the ulcer







ASSOCIATED DEFORMITY

RAW AREA RIGHT - FOREARM



SKIN GRAFTING



POST-STRETCHING

heals. Mercurochrome is applied to the wound. Liquid paraffin massage is given to other areas of the scar. During the next visit, or when the scar break down has healed in case of scar breakdown, plaster of paris splint is changed, after giving liquid paraffin massage to the scar and mobilizing and further extending the joint. These procedures are repeated till the patient attains full extension.

When full extension was obtained, plaster of paris splint is removed and pressure garment with static extension splint is given and the patient is advised to continue it for one year. Both the Pressure garment and the splint are removed once in 4 hours and massage to the scar is given with liquid paraffin. Gradual flexion of the joint actively and passively is done, and both the splint and the pressure garment are reapplied. When full extension and flexion was obtained, patient is advised to use the joint for full range of movement with the pressure garments on. Splints are applied during the night time. This splinting and pressure garments are continued for one year or till the scar fully matures.

Patient is asked to attend the out patient department for regular follow up, once in 15 days for first 3 months and then once a month after 3 month to ensure they are regularly using the splint and no recurrence of contracture has occurred.

RESULTS

Total of 23 cases were selected for the study during the period, November 2006 – March 2009. All the 23 patients were corrected by serial stretching

- Average time at which the patients report to the hospital, after developing contracture was 4.31 months, and it ranges from 20 days to 10 months
- 2. Flame burn was the commonest cause of burns
- Female gender was commonly affected and the age group was
 16 25 years in Elbow contracture and 5 15 years in knee contracture.

TABLE -1

Age	Male	Female	Total	
< 5 yrs	1	0	1	
5 -15 yrs	0	1	1	
16 - 25 yrs	2	7	9	
26 - 35 yrs	1	5	6	
36 - 45 yrs	1	1	2	
> 46 yrs	0	0	0	
TOTAL	5	14	19	

ELBOW CONTRACTURE - AGE / SEX DISTRIBUTION





TABLE -2

Age	Male	Female	Total
< 5 yrs	0	0	0
5 -15 yrs	1	2	3
16 - 25 yrs	0	1	1
26 - 35 yrs	0	0	0
36 - 45 yrs	0	0	0
> 46 yrs	0	0	0
TOTAL	1	3	4

KNEE CONTRACTURE - AGE / SEX DISTRIBUTION

- Elbow contracture being the commonest one, account for 82.6%
 of the total contracture
- Degree of contracture commonly reported was, more than 60° for the elbow joint and 30-60° for the knee joint.

TABLE - 3

Degree	Male	Female	Total
< 30°	3	3	6
30-60°	0	6	6
> 60°	2	5	7

DEGREE OF CONTRACTURE - ELBOW





TABLE - 4

Degree	Male	Female	Total
< 30°	0	1	1
30-60°	0	2	2
> 60°	1	0	1

DEGREE OF CONTRACTURE - KNEE

6. All patients had full correction of flexion deformity

Average time taken for full correction of flexion deformity was
 37.94 days for elbow contracture and 47.25 days for knee contracture.

TABLE - 5

ASSOCIATED DEFORMITY CORRECTION

	Elbow Joint Contracture	Knee Joint Contracture	Total
Associated Deformity	13	4	17
Simultaneous correction	10	2	12





8. 13 patients amounting to, 68.4% of the total elbow contracture patients and all the patients with knee contracture had associated deformity. 10 patients with elbow contracture and 2 patients with knee contracture had simultaneous correction of the associated deformity.

TABLES - 6

COMPLICATIONS	MALE	FEMALE	TOTAL
Scar Breakdown	1	0	1
Blister	1	4	5
Neuro-Vascular Compromise	0	0	0
Recurrence of Contracture	4	2	6

COMPLICATIONS - ELBOW CONTRACTURE

TABLES - 7

COMPLICATIONS - KNEE CONTRACTURE

COMPLICATIONS	MALE	FEMALE	TOTAL
Scar Breakdown	0	1	1
Blister	0	2	2
Neuro-Vascular Compromise	0	0	0
Recurrence of Contracture	1	1	2

- 9. 5 patients with elbow contracture and 2 patients with knee contracture had developed blister. One patient with elbow contracture and one patient with knee contracture had scar break down. All of them settled with conservative management
- 10. 6 patients with elbow contracture and 2 patient with knee contracture had discontinued the splint and had developed recurrence of contracture after correction by serial stretching, which was again corrected with serial stretching.







ASSOCIATED DEFORMITY

HAND CONTRACTURE



CONTRACTURE RELEASE WITH SSG



AXILLARY CONTRACTURE



CONTRACTURE RELEASE WITH Z PLASTY



DISCUSSION

Burn injuries, regardless of the etiology, rarely involve the joint. However, the joint function is often impaired because of burns. The joint problems and joint deformities noted in burn patients are mostly due to physical inactivity combined with limitation of joint movement because of scar contracture.

Initial management of burns during the acute stage is concentrated in the resuscitation of the patient from the burns shock ,with intravenous fluids, analgesics and antibiotics. Management of the burn wound depends on the depth of the injury. Collagen dressing is given to the superficial wound. Wound that does not heal by twenty one days needs surgical intervention, to prevent the wound from healing by secondary intention and contracture deformity. Wound that needs more than 2 weeks to heal have very high chance of developing hypertrophic scar, so pressure garments has to be given to prevent hypertrophic scarring, till the scar matures.

Limb that has burnt is elevated during the acute stage to lessen the edema formation and is splinted in the appropriate functional position. Later active and passive mobilization of the joints are done to prevent stiffness and contracture formation.



POST-STRETCHING



ASSOCIATED DEFORMITY

RIGHT ELBOW FLEXION CONTRACTURE

CONTRACTURE RELEASE WITH SKIN GRAFTING



COMBINED INTERVENTION



Most of our patients have very poor general condition and nutritional status. They are very reluctant for mobilization and these are the patients who are very prone to develop contracture, if physiotherapy is not initiated at the proper time.

Nutritional status of most of our patients is very low, moreover the food intake is also very poor. Both of these factors prolong the wound healing process. Apart from the smelly discharge from the wound, pain associated with the dressing change and mobilization, these patients are very much depressed, which all add to their woes, making them reluctant to mobilization, splinting and to have adequate food intake. When these patients recover, contracture and other deformity like hypertrophic scarring has already developed.

Holding bodily joints in flexion, so called posture of comfort, a characteristic body posture is commonly seen in distressed individual. Although the exact reasons are not entirely clear, contraction of muscle fibers at rest and the contractile force difference between the flexor muscles and the extensor muscles may play an important role in the genesis of this body posture. The magnitude of joint flexion, furthermore, increases as the individual loses voluntary control of muscle movement, as frequently seen in burn victim. Prolonged periods of physical inactivity, associated with burn treatment and scar tissue contraction

POST - STRETCHING





POST - STRETCHING WITH STATIC SPLINT





ASSOCIATED DEFORMITY INTER THIGH ADHESION



around the joint structure, as the recovery ensues, further impedes the joint mobility.

Analysis of our study shows flame burn was the commonest cause of burns. Female gender was commonly affected by burns, 17 out of the total 23 patients included in our study were female, and the age group commonly involved was between 16-25 years in elbow contracture. Knee contracture was commonly seen in 5 - 15 years.

Elbow joint was commonly affected by contracture in our study, accounting for 82.6% of the total cases. Literature review also shows elbow joint being commonly involved in post burn contracture.

Degree of contracture commonly reported was more than 60° for elbow joint and $30-60^{\circ}$ for knee joint. The average time taken for full correction of elbow contracture was 37.94 days and 47.25 days for knee contracture.

13 patients amounting to 68.4% of total elbow contracture and all the knee contracture patients involved in the study had associated other deformity.

When multiple joints get deformed, important areas get priority in contracture correction. Neck contracture is initially treated, as a secure airway is needed for subsequent surgical correction of other joint



POST-STRETCHING



ASSOCIATED DEFORMITY

NECK CONTRACTURE

NECK CONTRACTURE RELEASE WITH SSG





contracture. Limb joint contractures are corrected from proximal to distal manner, starting from axilla proceeding distally to elbow, wrist and finger contracture.

Serial surgery of the involved joint with a minimum period of 3 month interval in-between surgery takes prolonged time to rehabilitate these patients. These patient especially from poor socio economic status and with severe deformity finds employment difficult. With no income and rehabilitation their life becomes even more harder to survive.

Serial stretching helps in simultaneous correction of multiple joint contracture, thus reducing the overall time spent in correction of deformity and fastens rehabilitation. Of the 23 cases studied 17 patients had associated other deformity. Ten patients with elbow contracture and 2 patients with knee contracture had simultaneous correction of associated deformity.

Maturity of the scar affects the response to stress and strain applied to it, lesser the maturity greater the response. Immature scars respond to pressure dressing and to the strain applied favourably, by increased suppleness, softening, lengthening of the scar and by decrease in vascularity. All the patients included in our study had immature scar and all of them responded favourably to pressure dressing and stretching.

COMPLICATIONS OF STRETCHING

BLISTER WITH SUPERFICIAL ULCERATION



SCAR BREAKDOWN



Though contractile properties of the scar decreases with the maturity of the scar, the contracture corrected by stretching has to be maintained by proper splinting and pressure dressing for a prolonged time, as long as one year or till the scar matures to retain the beneficial effect.

Continuation of the splint for longer period especially after serial stretching needs good motivation and every patient had a good chance of discontinuing the splint and pressure garment at an earlier date. In our study 8 patients discontinued the splint and developed recurrence of contracture. Serial stretching was again started and contracture was corrected. All the patients were advised to continue the splint and asked to report for regular follow up .

Scar tissue is lined by thin layer of epithelium and it doesn't have dermal support. Hence scar epithelium is very prone to develop blister and ulceration, with minimal shearing force. Scar massage should be done in a circular motion with no shearing strain between epithelial lining and scar bed. Blister formation was seen in 7 patients in our study. Of these 7 patients, 3 patients developed blister following vigorous scar massage with liquid paraffin. Other patients developed blister while using pressure garments.
Hot humid climate commonly seen in our city makes the patient sweat a lot. This added with the shearing strain during the application of the tubular pressure garment makes them prone to develop blister and superficial ulceration.

Patients find wearing pressure garment cumbersome and discontinued at an earlier date as it is prone to develop blister and ulcer, resulting in scar hypertrophy and recurrence of contracture, if not splinted properly. Of late pressure stocking was replaced with elastocreep bandage which the patients found easy to use and more acceptable, and blistering was reduced by application of cotton pad as the first layer to absorb the excessive moisture especially during hot climate to prevent maceration.

Two patients developed scar break down during the process of serial stretching. Both of them had severe hypertrophic scarring and when stretched to their maximum extension resulted in scar break down. Both of them settled with conservative management. Stretching was stopped temporarily but splinting was continued in lesser extension till the ulcer healed and stretching was again started but at a slower rate and stretched just short of the maximum extension during each session. Literature review also shows low torque of longer duration gives better result when compared with high torque of shorter duration. (M. Usuba et al) Even though burn injury mostly involve skin and subcutaneous tissue, except in very rare cases where deeper tissue is involved, contracture involve all the soft tissues upto the joint including neuro vascular bundle and joint capsule.

Knee joints have powerful flexors provided by the hamstring muscles, are very prone to develop flexion contracture and once they get contracted, full correction with single surgery is difficult to obtain, as not only muscles, tendon and joint capsule but also, neurovascular bundle contract. Serial stretching helps in gradual correction of contracture. Slow stretching helps in gradual lengthening of the contracted soft tissues which help the patient to regain full extension without neuro vascular compromise.

CONCLUSION

Serial stretching is a good modality of treatment for correcting post burns flexion contracture of the knee and elbow. It can be used as an out-patient procedure without anesthesia and can be applied to all age group.

Slow progressive and prolonged stretching helps in full correction without serious complication.

Patients and their parents need good motivation, as prolonged follow up and after care, in the form of pressure garment, splint, scar massage and exercise are necessary.

Cotton padding with elastocreep bandage helps in reducing blister formation which is very common with compression stocking. Blister formation is the commonest cause for discontinuation of pressure garment and splint.

Serial stretching being another tool in the armamentarium of burns surgeon helps in the simultaneous correction of multiple deformities or in patients with high chance of hypertrophic scarring or when surgical correction is not possible.

Early splinting, proper positioning and mobilization helps to prevent development of contracture. Splinting and pressure therapy has to be continued till the scar fully matures, to prevent scar hypertrophy and recurrence of contracture.

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Prof. Dr.S.R.VIJAYALAKSHMI M.S. M.Ch. (Plastic Surgery)

PROFORMA

NAME :	AGE/SEX:
IP/PS NUMBER :	OCCUPATION :
ADDRESS	
HISTORY	
HISTORY OF BURNS	
TREATMENT HISTORY	
DURATION OF CONTACTURE	
LOCAL EXAMINATION:	ELBOW JOINT / KNEE JOINT
SKIN OVER THE JOINT :	NORMAL / SCAR / SSG
EXTENT OF THE SCAR / SSG:	
TYPE OF SCAR :	IMMATURE / MATURE
SCAR HYPERTROPHY:	YES / NO
SKIN ABOVE THE JOINT :	NORMAL / SCAR / SSG

SKIN BELOW THE JOINT:

NORMAL / SCAR / SSG

DEGREE OF CONTRACTURE:

MOVEMENT OF THE JOINT:

ACTIVE

PASSIVE

DISTAL NEURO VASCULAR EXAMINATION :

ASSOCIATED OTHER DEFORMITY / MORBIDITY:

SURGERY DONE FOR ASSOCIATED DEFORMITY:

DATE OF SURGERY:

PROCEDURE :

INVESTIGATION:

URINE :

BLOOD:

X-RAY :

Date	Deformity angle	Complication		

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MASTER CHART

S. No.	Name	Age	Sex	IP No/ PS No.	Knee / Elbow	Maturity of Scar	Scar - Hypertrophy	Duration of contracture	Degree of contracture	Days needed for correction	Complication	Recurrence	Associated deformating	Associated correction
1	David Gunaseelan	23	М	21649	Elbow	Immature	+	7 months	30°	32	Scar Breakdown	+	HT Scar	-
2	Ramesh	28	Μ	1746/08	Elbow	Immature	+	3 months	30°	14	-	+	-	-
3	Latha	23	F	24650	Elbow	Immature	-	2 months	60°	36	-	-	PBC neck	Neck Contracture Release + SSG
4	Sathyanarayanan	4	Μ	24613	Elbow	Immature	-	20 days	80°	28	-	+	-	-
5	Kannagi	34	F	23606	Elbow	Immature	-	3 months	90°	56	-	+	-	-
6	Durga Devi	15	F	17399	Elbow	Immature	+	4 ¹ / ₂ months	30°	21	-	-	-	-
7	Jenifer	22	F	17466	Elbow	Immature	+	4 months	40°	34	Blister Superfical ulcer	-	HT Scar PBC Neck	-
8	Divya	17	F	2179	Elbow	Immature	+	8 months	60°	48	Blister	-	PBC neck	Neck Contracture Release + SSG
9	Alamelu	24	F	2528	Elbow	Immature	+	10 months	70°	55	-	-	PBC Left elbow	Left Elbow Contracture Release + SSG
10	Pachiammal	27	F	2898	Elbow	Immature	+	3 months	40°	30	-	-	PBC neck HT Scar Microstomia	Neck Contracture Release + SSG
11	Vijayababu	25	М	21931	Elbow	Immature	+	4 months	80°	68	Blister Superfical ulcer	+	PBC both axilla and hand, HT Scar	1) Axillary Contracture release + Z plasty 2) Hand Contracture release + SSG
12	Kamatchi	18	F	3519	Elbow	Immature	+	4 months	30°	24	-	-	-	-
13	Sathya	26	F	1038/08	Elbow	Immature	+	7 months	70°	52	-	-	PBC (L) elbow, PBRA (L) Forearm	-
14	Sumathi	20	F	99/09	Elbow	Immature	+	3 months	60°	42	-	-	-	-
15	Vijayalakshmi	27	F	27188	Elbow	Immature	-	2 ¹ / ₂ months	40°	14	-	-	PBRA (R) Forearm	SSG
16	Mohanraj	36	м	185/05	Elbow	Immature	+	2 months	30°	23	-	-	PBC (R) Axilla, (R) elbow, HT scar	Right Axillary Contracture release + SSG
17	Kasthuri	44	F	22980	Elbow	Immature	+	5 months	20 °	14	-	-	Eversion of Lower Lip	Eversion of lower lip correction with SSG
18	Chandra	30	F	21842	Elbow	Immature	+	5 months	80°	60	Blister Superfical ulcer	-	PBC (R) elbow	Right Elbow contracture Release + SSG
19	Radha	23	F	6477	Elbow	Immature	+	7 months	90°	70	Blister	+	PBC (R) elbow	Right Elbow contracture Release + SSG
20	Dinesh	7	М	21680	Knee	Immature	+	2 months	90°	72	-	+	HT scar, Interthigh adhesion, Mental Retardation	-
21	Marianisha	10	F	25489	Knee	Immature	+	5 months	40°	36	Blister	+	PBRA (R) Leg	SSG
22	Maheswari	20	F	5428	Knee	Immature	+	2 months	60°	46	Blister	-	HT Scar, PBC (L) Hip	-
23	Gunamangai	6	F	6475	Knee	Immature	+	5½ months	30°	35	Scar Breakdown	-	PBC Right Hand, alopecia	Contracture Release Right hand + SSG

LIST OF ABBREVIATIONS

TGF-β	-	Transforming Growth Factor - β
FGF	-	Fibroblast Growth Factor
VEGF	-	Vascular Endothelial Growth Factor
IGF - 1	-	Insulin like Growth Factor - 1
IG - E	-	Immunoglobulin - E
IL - 6	-	Interleukin - 6
SSG	-	Split Skin Grafting
HT Scar	-	Hypertrophic Scar
PBC	-	Post Burn Contracture
PBRA	-	Post Burn Raw Area
PDMS	-	Poly Dimethyl Siloxane
МСР	-	Metacarpophalangeal
IP	-	Inter Phalangeal
DIP	-	Distal Interphalangeal
ECM	-	Extra Cellular Matrix
TNF - α	-	Tumor Necrosis Factor - α
IL - 1	-	Interleukin - 1
MMP	-	Matrix Metallo Proteinase
CH13 L1	-	Chitinase 3-like protein 1