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REGRESSION OF DUPUYTREN'S CONTRACTURE

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The phenomenon of clinical regression of Dupuytren's contracture is described and discussed. It is already recognized and used in fasciotomy where it follows the release of longitudinal tension. The quite extraordinary resolution produced by continuous passive skeletal traction in extension is presented. Regression beneath grafted skin is described and discussed with its clinical implications.

The generally accepted view of Dupuytren's contracture being "irreversible" now presents a challenge for further clinical and pharmacological studies.

The possibility of non-surgical control does exist.

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Regression occurs in Dupuytren's contracture. The formed nodule and cord may soften and even disappear. By considering those situations in which regression occurs, a series of future studies of this condition can be envisaged. Then perhaps we can begin to apply alternative therapeutic modalities or modified surgical procedures to expedite regression and resolution. The present wholesale ablative surgery with its greater than 50% recurrence rate will prove unacceptable to patients of the next millenium.

Some circumstances in which regression and resolution have occurred are anecdotal, but must still be considered; others are experimental, but must still be pursued; others are chance surgical observations which must still be interpreted, and finally some have been induced by surgical enterprise, the principles of which must still be integrated into the overall management of this common condition.

Spontaneous regression

Clinicians rarely see regression of a nodule of Dupuytren's contracture because only a very friendly patient will return to show its disappearance. I have never had such a patient, although often a large fleshy early nodule in the palm has become smaller and firmer and more discrete over a period of months. But that is progression, not regression of the disease.

Disappearance of a palmar nodule has been reported by Gordon (1948), and it was noted that "such observations must be of considerable importance when the aetiology is considered" (Hueston, 1963).

My mentor, Professor E. S. J. King, perhaps Australia's greatest surgeon-pathologist and acute observer of the metaplastic potential of connective tissues, was adamant that he had in his own right palm a nodule of Dupuytren's contracture which disappeared after a few years. Only a patient, prolonged prospective study of the natural history of Dupuytren's contracture, like that of Millesi (1985) but extended to many thousands of cooperative patients, can finally settle this question of natural resolution.

Natural regression

Two instances of resolution in a paralysed arm have been recounted to the author. One has already been reported, in the hemiplegic hand of a close relative of Dr L. D. Howard, student and successor of Bunnell (Hueston, 1963). Recently Dr C. B. Wynn Parry has described a C8, T1 traction injury of the brachial plexus with subsequent disappearance of Dupuytren's contracture in the paralysed hand (personal communication, 1991).

It is possible that this phenomenon is more common. The cooperation of neurologists is needed, because only a prolonged study of many such afflicted patients over a decade can document this. A physiological explanation may then follow, even perhaps indicating a possible new therapeutic pathway.

Drug-induced regression

"Surgery is always the last resort" was a favourite aphorism of Dupuytren throughout his reported lectures. Non-surgical treatment or control of Dupuytren's contracture has been a continuing challenge since Dupuytren's own ineffectual linaments and douches. Changing views of the causation have dictated the vogueish use of systemic drugs, vitamins and steroids, even to the local use of injectable collagenolytic agents. Only a few of these applied histochemical concepts, where regression seemed at least a possibility, will be mentioned.

On the basis that it converts collagen to gelatin, fibrinolysin has been used on Dupuytren's contracture without success (Black, 1915). The same result followed attempts to dissolve newly formed collagen by dimethylsulphoxide (DMSO) (Vuopala and Kaipaineu, 1971), steroids and vitamin E. Procarbazine was noted to have improved not only a patient's Hodgkins disease but also his Dupuytren's contracture (Aron, 1968) in much the same serendipitous way as Murrell et al. (1987a) noted that allopurinol given for other reasons improved Dupuytren's contracture in some patients. This latter led to the development of an hypothesis of pathogenesis (Murrell et al, 1987b; Murrell and Hueston, 1990). A clinical trial has failed so far to confirm this hypothesis (Hueston, 1990), but the use of allopurinol remains the

nearest to a logical systemic drug therapy so far proposed for Dupuytren's contracture.

A local attack on Dupuytren's contracture by enzymic degradation by trypsin injection simply produced a closed but rather clumsy chemical fasciotomy (Hueston, 1971).

The injection of triamcinolone acetate into keloid scars and nodules of Dupuytren's contracture nodules has been shown to induce regression (Ketchum et al, 1974). The steroid appears to inhibit the synthesis of collagen by blocking fibroblast receptors. Resorption proceeds without new synthesis of collagen, so the tissue mass is diminished. Ketchum combined radical fasciotomy with steroid injection into residual palmar and digital nodules, knuckle pads and plantar nodules (Ketchum, 1991).

The most recent studies of the control of fibroblast contractility in Dupuytren's contracture have been pharmacological (Naylor et al, 1991); prostanoids have been shown to induce contraction in strips of Dupuytren's nodule. Isolation of the precise cell mediator would bring the possibility of producing an antagonist. "Tissue-specific" control of Dupuytren's nodular activity and induction of regression would then be possible.

Regression after tension release

There have been many instances of continued regression after traumatic release of a tight band (Grace and McGrouther, 1984). Fasciotomy under local anaesthesia has a well defined role in the management of Dupuytren's contracture (Colville, 1983). The resolution of the proximal palmar segment takes up to six months, the distal segment may never disappear and even later progress if its digital attachments remain as a source of intermittent physical traction transmitted to the remaining Dupuytren's tissue.

On a bigger scale, the whole width of the palm may be incised down to the flexor tendon sheaths with relief of MP joint flexion deformity (McGregor, 1985; Ketchum, 1991). The risk of incomplete MP correction or recurrent MP deformity is avoided by careful release of the deep paratendinous attachments of the affected palmar aponeurosis and interposition of a skin graft to prevent reunion across the wide palmar defect. Resolution of Dupuytren's contracture is noted proximal to this wide fascial release over some months. Distally any digital disease will remain and progress unless further release is made in the finger, and the risk of reunion of the band again removed by a transverse skin graft. There is a risk of digital nerve damage during fasciotomy within the digit, but even this does not deter Gonzalez (1985, 1988) who stresses the difference between a primary operation and dissection in a scarred digit with recurrent disease. Total division and lengthening of all tight bands in a localized area can be achieved by a Z-plasty with large flaps including and thus lengthening the underlying fascia (Watson, 1984; McGregor, 1985), and leading to regression.

Skin graft interposition in the digit has been termed a

"firebreak" (Hueston, 1984) but perhaps "fibrebreaK" would be a more appropriate term. This way lies the surgery of the future.

The precise cellular control mechanisms involved in the production and resorption of collagen in growth, work hypertrophy and other connective tissue remodelling, are not yet clear. A mechanism for the transmission of physical forces through the matrix to the cells by means of intercellular linkage has been proposed by Tomasek et al. (1987), probably through extracellular fibronectin to intercellular actin bundles. This provides a possible explanation for the hypothesis of work hypertrophy in Dupuytren's contracture proposed by Luck (1959). Reversal of this process by release of tension thus allows resorption to exceed production of collagen, leading to clinical regression.

Regression after skin exchange

The palmar skin appears to play an unexplained role in the behaviour of underlying fibroblasts. Only empirical observations can be made until the cell-controlling factors have been further elucidated. Some clinical facts elude explanation.

In severe Dupuytren's contracture where the inherited tendency (diathesis) is so strong that extensive recurrent disease has occurred in a digit—or even if it is anticipated—a total clearance including both the fascial mass and the overlying skin is sometimes performed. Such a "dermofasciectomy" leaves large digital skin defect which is resurfaced by a full-thickness (Wolfe) skin graft (Hueston, 1969, 1985, 1991). The protection that this provides against recurrence suggests that an inducing factor has been removed.

Even more relevant to the subject of resolution is the fact that the heavily infiltrated skin excised from the digit in dermofasciectomy softens to its normal suppleness within two months when transplanted into the donor-site defect (Hueston, 1974). This still awaits explanation.

A cooperative elderly Russian patient whose digits had been corrected by skin grafts consented to an experiment on his remaining palmar plaque. Here, under local anaesthesia, 1 cm² of palmar skin was excised from over the centre of the plaque and a split skin graft applied directly to the exposed bed of diffuse Dupuytren's tissue left in situ. Over the following six weeks, this originally flat graft was seen to sink, becoming a depression which on palpation was softer than the surrounding remaining Dupuytren's plaque. A biopsy at two months showed only a thin layer of scar over the flexor sheaths.

The role of the palmar skin in the control of the subcutaneous palmar-digital space awaits explanation. These phenomena—resolution when transplanted, resolution beneath skin transplanted from elsewhere, and the freedom from recurrence beneath new skin, whether a free graft (full or split thickness), or a flap—all must be telling us something. Future investigations may elucidate

the factor or factors involved in this dermis/Dupuytren's contracture relationship.

Whatever the factor in the transplanted skin that induces resolution, a dermal "anti-Dupuytren" factor may eventually be available.

Regression during continuous traction

The intractable nature of an established severe Dupuytren's contracture is such that radical surgery, even amputation, may seem to be the only solution. However, Messina (1989) with the aim of avoiding amputation if at all possible, has for five years now been applying *continuous* skeletal traction to severely flexed digits with Dupuytren's contracture—often recurrent and often to more than one digit (Fig. 1). The patient is then given a small spanner to rotate a turnbuckle to pull the transfixed finger into extension at the rate of 2 mm each day. Full extension of MP and IP joints is obtained in 2 to 3 weeks. A visit permitted examination of patients who had been subjected to this pre-operative passive *continuous* traction. The phenomenon of regression was fascinating and prompted this paper, rather than a conversion to the technique at present.

When questioned the patients had three interesting comments: (1) it is quite painless during this continuous traction; (2) after 4 or 5 days, there is a feeling of "release" in the Dupuytren's cord and extension up to 4 or 5 mm is then possible each day until near-full extension is reached at 2 weeks; (3) on removal of the traction at 2 weeks there is a sensation of retraction returning in the region of the Dupuytren's cord over the next day or two.

At this stage of near full extension the previously indurated cord and nodule feel soft (Fig. 2) and almost flat. Clearly regression has been induced by continuous traction. Fasciectomy is then performed of the soft flat pale ribbon of former Dupuytren's contracture. No rupture or haemorrhage has been found so far.

A possible explanation for this is that, during the first three or four days, there is a realignment of collagen bundles allowing some lengthening, followed by "slippage" of collagen protofibrils into a staggered longer arrangement, as cross-linkages are lost and reformed under continuous traction. The "release" at four or five days may be due to cell-mediated dissolution of more major cross-linkages under the influence of collagenase released by the physical force of continuous traction acting on the cell matrix linkage system mentioned earlier (Tomasek et al, 1987).

The postulated early "slippage" of collagen protofibrils can occur within milliseconds, allowing disaggregation and a rapid "ratchet" rearrangement within a collagen fibril without structural changes in the collagen molecules (Scott, 1990). So far only studied in the collagen of echinoderms, this ability of collagen fibrils to slide past one another is well described by Bailey (1985). This phenomenon has not yet been sought in human collagen

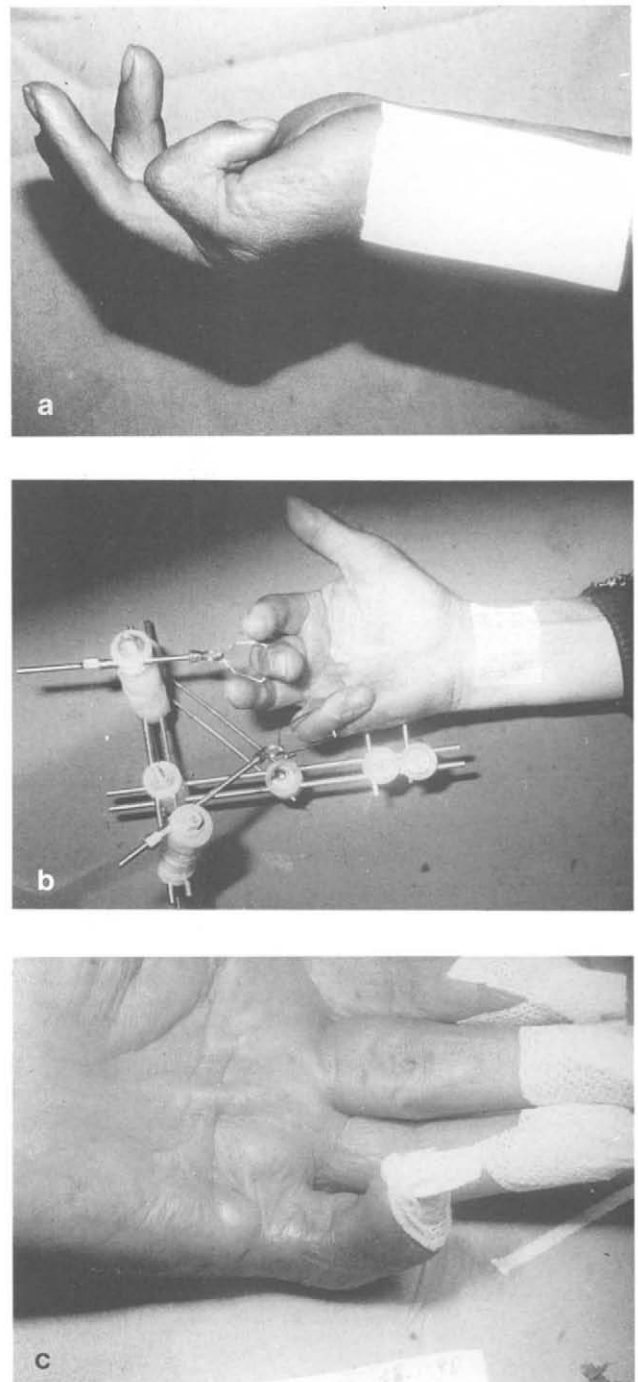


Fig. 1 (a) Dupuytren's contracture before traction: Severe flexion deformity of little finger and moderate deformity of middle finger. (b) Traction frame in place. Two pins across shafts of both fourth and fifth metacarpals; traction wires transfixing neck of middle phalanges and meeting at small turnbuckle at end of rod fixed to frame. Continuous passive traction is then applied daily using a small spanner on each turnbuckle. (c) Ample exposure for fasciectomy. Same patient (despite reversal in printing from Dr Messina's transparency) after 3 weeks of continuous passive traction, and two weeks then of pre-operative hand therapy. Some relapse has occurred in the two weeks since traction has been discontinued.

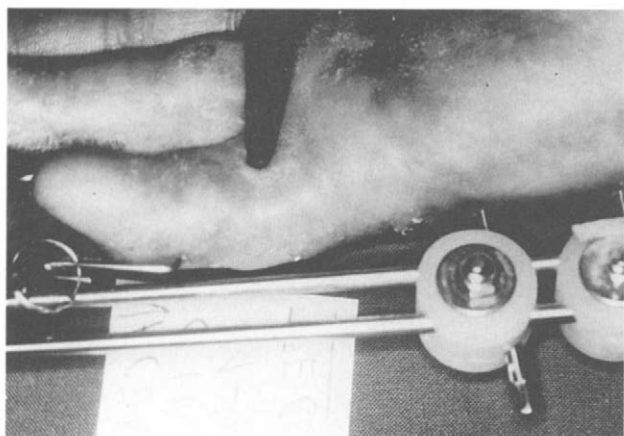


Fig. 2 Another patient immediately after discontinuation of two weeks of continuous traction. A black biro is being pressed into the region of the former Dupuytren's nodule to show how soft it has become.

but it opens the door to speculation in relation to the regression induced by continuous passive traction.

The far slower breakdown over hours and days of collagen molecules to aminoacids and the formation of new collagen molecules is probably the mechanism of connective tissue remodelling in growth, scars and in Dupuytren's contracture (Brickley-Parsons et al, 1981). This resorption and remodelling may be effective in the later stages of the astonishing regression/resolution clinical phenomenon.

What would happen if the extended digit were kept extended? What has happened to the joint ligaments? Messina operates at two weeks because his aim is to correct the deformity to a state where safe surgery through non-macerated skin can be performed and amputation avoided. Some of the patients examined had significant residual IP joint stiffness and one had a rupture of the volar plate, but none had an amputation. So far, Messina has used continuous traction purely as a pre-operative manoeuvre (Messina, 1989).

The implications of this technique at a histological and chemical level are immense in the ultimate understanding of Dupuytren's contracture, and the physiology of regression may well be a guide to the pathogenesis of contraction.

Comment

Attempts to explain or to interpret these observations have been simplistic. One fact is clear: regression occurs in Dupuytren's contracture.

No "final common pathway" of chemical or cellular action links these clinical observations as yet, but it might be profitable to divert attention from the mechanism of contraction in Dupuytren's disease to its reversal under certain environmental and physical provocations.

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