25 A single injury to the hand

In Chapters 19 and 24 Mikkelsen and Meagher associate Dupuytren's disease (DD) with manual work. The former author found an increased prevalence of DD in heavy manual workers. The significant differences between manual and non-manual workers did not appear until after 60 years of age, that is, until after a lifetime of work and at an age when DD is most common. It is difficult to evaluate work patterns from epidemiological studies because workers change jobs and jobs change with time. Sedentary workers often abuse their hands in their hobbies or sporting activities. However, Mikkelsen's results suggest that manual work is not associated with the early appearance or the rapid progress of disease.

Meagher has made a case to associate DD with manual work on the basis that Dupuytren's tissues are no less susceptible to the aggravating effects of manual labour and hand tool designs than other soft tissues. Unlike trigger finger and carpal tunnel syndrome there are no work patterns — other than heavy manual labour — associated with DD. Further, there is no report in the literature of a series or even a case report of DD related to a sporting activity. Professional golfers, racquet, baseball and cricket players as well as professional musicians apply prolonged and repetitive stresses to their hands. They often develop soft tissue injury at the wrist, elbow and shoulder, but are not prone to DD.

Thus it is doubtful whether the onset of DD is associated with use or abuse of the hands and there is no evidence that the course of the disease is influenced by activity or inactivity of the hands. However, many cases have been reported of DD appearing after a single injury. Clarkson (1961) reported only 2 cases of his own but discussed several cases provided by his authoritative colleagues. His paper is most supportive of a single injury causing DD. Hueston (1962) reported 11 cases of forearm fracture, forearm infection, and elbow and shoulder dislocation in which the onset or progress of DD occurred within months of injury. He attributed this phenomenon to immobilization of the hand with swelling and vascular changes. In the same study Hueston included 21 patients with 'acute invalidism' due to myocardial infarction, lower limb injury, eye surgery, abdominal surgery, pulmonary tuberculosis, and diabetic crisis. These patients also developed DD, presumably because of enforced bedrest. There are no similar reports, although Hueston stated that Plewes (1956) had noted DD in patients with Sudek's atrophy.

AN EPIDEMIOLOGICAL ANALYSIS

As part of the survey reported in Chapter 20, one of the questions asked was of a history of a single injury associated with the onset of DD. The significant variables compared to those patients who did not have a single injury are listed in Table 25.1. Race was not a factor. The prevalence in northern European and Japanese people was the same. More males, and also more males of less than 45 years of age were involved. Most patients were manual labourers. The disease was less severe: more frequently it was unilateral and only one ray was involved.

This analysis identifies a group of young male labourers in whom a single injury to the hand may

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Dupuytren's Disease Biology and Treatment
Churchill Livingstone Edinburg 1990.

Table 25.1 Variables associated with patients who related a single injury to the onset of disease

Variable	Injury (n = 106)	No injury (n = 1114)	p Value
Northern European origin	81%	83%	NS
Japanese origin	12%	10%	NS
Male	91%	76%	<0.02
Age at onset if Male < 45 years	18%	12%	<0.02
Manual labour	58%	42%	<0.002
Unilateral disease	47%	32%	<0.02
One ray involved	46%	35%	<0.05
Outcome at PIPJ V			
Perfect	29%	18%	<0.05
Improved	63%	55%	NS
Worse	8%	27%	<0.05
Recurrence and extension	17%	23%	NS

PIPJ V = fifth proximal interphalangeal joint.
NS = not significant.

have precipitated the appearance of rather mild disease. It is noteworthy that racial origin and the diathesis factors, except age, had no significant influence.

ANALYSIS OF A PERSONAL SERIES

A total of 309 fully documented cases were reviewed in order to select those patients in whom a close relationship between a single injury and the onset of DD was likely. The following guidelines were used to select the patients:

1. History of a single injury to the hand.
2. Objective evidence of tissue damage, such as scarring or healed fracture.
3. DD in the area of injury.

According to these guidelines 18 patients (6%) of the series qualified. These patients had a palpable nodule or cord in the area of the injury, with the exception of 1 patient with a Colles' fracture. She was included because she was the only patient in the series with a close association in time between Colles' fracture and DD. The other 17 patients were male labourers.

Sixteen of the patients were right hand dominant. Only 2 patients were left-handed and contracture was seen in the left hand alone in only 1 patient (who was right-handed). Bilateral disease was noted in 9 patients.

The extent of disease was minimal in most patients and consisted of a palpable nodule or cord in the area of injury (Fig. 25.1). All but 2 patients were operated upon and they were reviewed 1–18 years later. The preoperative diagnosis in each case was DD but on review of the pathological specimens (by DTS), the tissue of 9 patients was considered to be scar tissue and not fibromatosis.

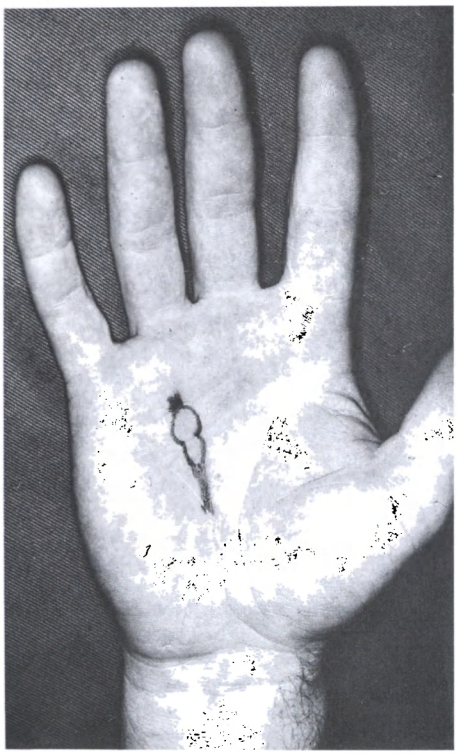
Histological features of DD and scar tissue

Microscopically, distinguishing between scar tissue resulting from trauma and DD may be difficult. This is true when examining specimens of the residual stage of DD when the fibroblastic proliferation has completely subsided and the cellular nodule is replaced by acellular tendon-like collagenous tissue. Similarly, a recent hyperplastic scar can mimic the proliferative stage of DD so that cases of flexion contracture following trauma could be mistaken for DD resulting in trauma being credited as the causal and aetiological factor. The presence of a cellular nodule is necessary to confirm the diagnosis of DD.

It is therefore important to be critical about the histological diagnosis of DD especially in patients with a history of previous trauma. Criteria for differentiating between DD scar tissue are listed in Table 25.2.

Lesions of DD are best delineated from adjacent fibroconnective tissue under lower power examination. The proliferating fibroblastic nodules (Luck 1959) are angiocentric and cellular (Fig. 25.2), whereas in the involutional and residual stages, the tissues tend to be less cellular or fibrous cord-like, and on cross-section may appear nodular but relatively acellular (Fig. 25.3).

Cleft-like spaces which are tissue artefacts from sectioning are frequently encountered in DD lesions. There artefactitious spaces may reflect the non-infiltrative nature and the lack of adhesions between the Dupuytren's lesions and adjacent tissue. Clefting artefacts are seldom noticed in scar tissue. While clefts are peculiar to DD, hyaline change of collagen is almost pathognomonic of



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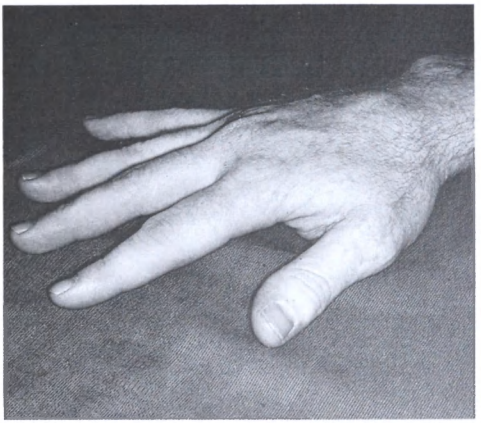

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Fig. 25.1 SG suffered a severe crush injury to the right hand at age 17. There were no fractures and only a minor laceration of the index finger but the hand remained swollen for about 2 months. He first noted thickening of the palm at age 22. **A** The hand at 28 years of age. There is a nodule and cord in the fourth ray. **B** He cannot lay the hand flat. Metacarpophalangeal joint contracture is progressive.

hyperplastic scar tissue. Hyalinization describes a morphological change of collagen characterized by intense eosinophilia. The collagen fibres become widened, homogenized, and assume a pinky-red and almost refractile property (Fig. 25.4). Such a change has never been described in Dupuytren's tissue.

Under higher magnification, fibroblastic proliferation in scar tissue tends to be more disorganized and the fibroblasts are usually more

Table 25.2 Histopathological differences between DD and scar tissue due to trauma

Dupuytren's disease	Scar tissue due to trauma
Lesions tend to be nodular in configuration in the proliferative stage; nodular or cord-like during involutional and residual stages	Irregular in shape and configuration related to type of trauma
Non-encapsulated but usually well demarcated from surrounding tissue; may have clefting artefact (Fig. 25.1)	Infiltrative boundary and merges with surrounding tissue; no fixation artefact (Fig. 25.2)
Predominantly subcutaneous in location with fibrous cord-like extensions to overlying dermis	Predominantly dermal or deep dermal in location
No epidermal reaction	Epidermal reaction, i.e. atrophy or hypertrophy may be present; focal loss of dermal elastic fibres and adnexal structures
Fibroblasts in proliferative lesions tend to be uniform and angiocentric	More pleomorphic fibroblasts, spindle or stellate-shaped; haphazardly arranged
No hyaline change	Hyaline change of collagen is the hallmark of keloid or hypertrophic scar.
Minimal inflammation	Inflammation may be marked; may have foreign body-type granulomatous inflammation.

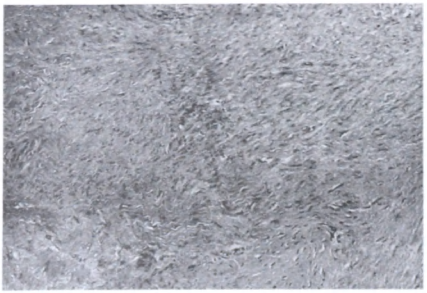


Fig. 25.2 Proliferating fibrous nodule of DD. Note the cellularity and lack of hyaline change of the collagen. Haematoxylin-eosin stained; × 100 original magnification.

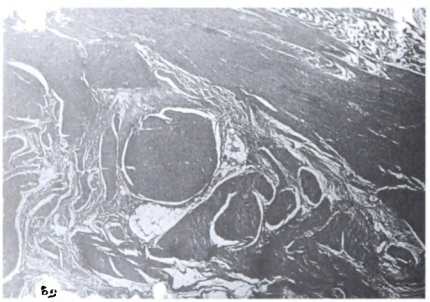


Fig. 25.3 Cord-like and nodular profile of residual tissue of DD. Arrow shows cleft-like space which is tissue artefact commonly observed in this type of lesion. Haematoxylin-eosin stained; × 25 original magnification.

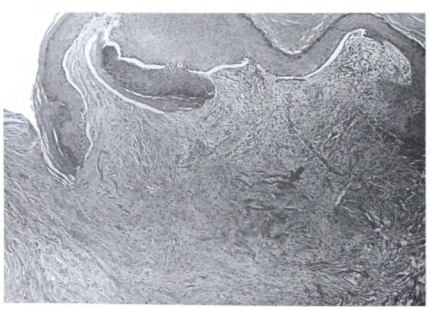


Fig. 25.4 Skin showing hyperplastic scar in dermis. Arrow shows hyaline change of collagen pathognomonic of scar. Haematoxylin-eosin stained; × 40 original magnification.

pleomorphic cytologically with their cell bodies varying from spindle to stellate-shaped. Although haemorrhage and haemosiderin pigment deposition may be seen in both lesions, the finding of more intense inflammation or foreign body-type granulomatous reaction would definitely favour the diagnosis of scar due to trauma.

Differential diagnosis of DD and scar

On the basis of the clinical assessment of the nature and course of the disease, but particularly on the pathological report, the 18 patients were placed into three groups, as shown in Table 25.3.

Seven patients were thought to have DD associated with a single injury. Each was injured and noticed DD before age 30. Four of the 7 patients had a severe laceration (perhaps with an element of crush injury; (Figs 25.5 and 25.6). All had both scar tissue and Dupuytren's tissue removed at operation and 2 needed skin grafting. That is, these patients had both scar contracture and Dupuytren's contracture. Three patients in this group had bilateral disease. The disease in the uninjured hand appeared later and was operated upon after the age of 45 in 2 patients.

Two patients were considered to have DD unrelated to their injury. This judgement was made primarily because the age at injury and onset of disease was in the sixth decade, when DD most often appears. Also both patients had 'typical' bilateral disease and there was no appreciable time interval between the onset of disease in the injured and uninjured hand. Nine had fractures of metacarpal IV and V; JP, who was the only female in the series, had a Colles' fracture.

Nine of the patients who were diagnosed as having DD did not have histological evidence of the disease. In 4 of these patients, the nodule disappeared (Fig. 25.7). In the other 5 patients, the surgical specimens revealed scar tissue rather than fibromatosis. Two of these 9 patients were thought to have bilateral DD. In JF the nodule disappeared on the injured side but a nodule remains on the uninjured side 14 years later without progression. In TR the cord removed from the little finger was scar tissue (Fig. 25.8). A palpable cord in the other hand is unlikely to be DD because he had similar injuries to that hand.

Table 25.3 The relationship of injury to DD

Patient	Site of disease	Age at injury		Operation	Type of injury	Extent of disease	Type of operation operation	Disease in other hand	Reason for category
		Injury	Disease						
Dupuytren's disease related to injury									
SG	Right	17	22	—	Crush	Nodule	None	None	Early onset
RH	Bilateral	29	29	35	Crush	Severe	Dermofasciectomy	Severe	Early onset
AG	Bilateral	29	29	35	Laceration	Nodule and cord	Dermofasciectomy	Nodule	Early onset
RP	Bilateral	20	21	40	Laceration	Nodule and cord	Fasciectomy	Progressive	Early onset
JO	Right	10	?	39	Laceration	Nodule and cord	Fasciectomy	None	Early onset
RDd	Right	10	?	51	Laceration	Nodule and cord	Dermofasciectomy	None	Early onset
TM	Right	24	24	26	Fracture	Nodule and cord	Fasciectomy	None	Early onset
Dupuytren's disease not related to injury									
LG	Bilateral	52	55	61	Fracture	Nodule	Fasciectomy	Nodule	Typical disease
JP	Bilateral	56	56	59	Fracture	Severe	Fasciectomy	Progressive	Typical disease
Not Dupuytren's disease									
JE	Right	53	53	53	Crush	Nodule	Correct scar contr.	None	Nodule disappeared
DB	Right	45	45	45	Crush	Nodule	Correct scar contr.	None	Pathology
RK	Right	51	51	56	Crush	Nodule	Excise nodule	None	Pathology
RD	Right	34	34	34	Crush	Nodule	Correct scar contr.	None	Nodule disappeared
JF	Bilateral	40	41	—	Crush	Nodule	None	Nodule	Nodule disappeared
JL	Right	25	25	34	Puncture	Nodule	Fasciectomy	None	Pathology
TR	Bilateral	28	28	28	Fracture	Cord	Fasciectomy	Cord	Pathology
FP	Left	34	34	34	Electric burn	Cord	Correct scar contr.	None	Pathology
RDo	Right	61	61	61	Infection	Cord	Fasciectomy	None	Pathology

It is likely that all patients who develop DD, including those with a single injury, have a genetic predisposition to the disease (Hueston 1987). Of the 7 patients in Table 25.3 whose disease was related to injury, 3 had bilateral disease and 2 others had a positive family history. The absence of a family history in the remaining 2 patients is meaningless because it is well known that most patients with DD do not know the correct status of even close relatives.

Both series reveal that age is a factor in relating DD to a single injury. The average age of onset is 48.3 ± 14.5 years in men and 57.6 ± 14.2 years in women (Chapter 20). In the large series, a single injury was associated with young men with minimal disease. In my personal series all 7 patients whose DD was related to a single injury were under 30 years of age when the disease appeared. Perhaps a single injury could precipitate DD in an older person, but there is no evidence from these studies to support such a theory. If DD appears within the expected age group, it would not be possible to assign a causal relationship to a single injury.

Age is an important factor when assigning a causal relationship between DD and a single injury. But age is also a feature of an increased diathesis in which the patient develops early and aggressive disease. The diathesis factors associated with severe disease are discussed in Chapter 22. Before accepting and rejecting a causal relationship, the other diathesis factors must be considered:

1. *Race.* Most people with DD are of northern European origin, although the disease is not

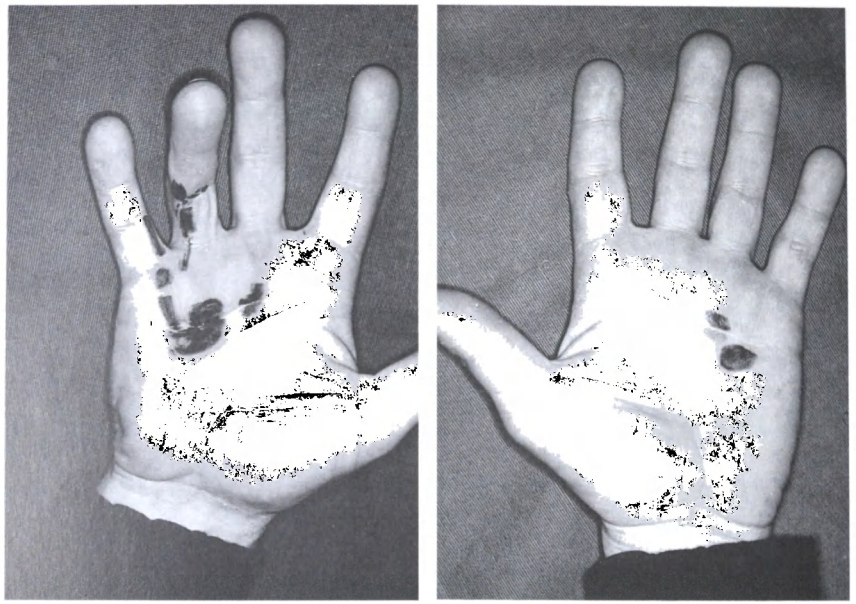


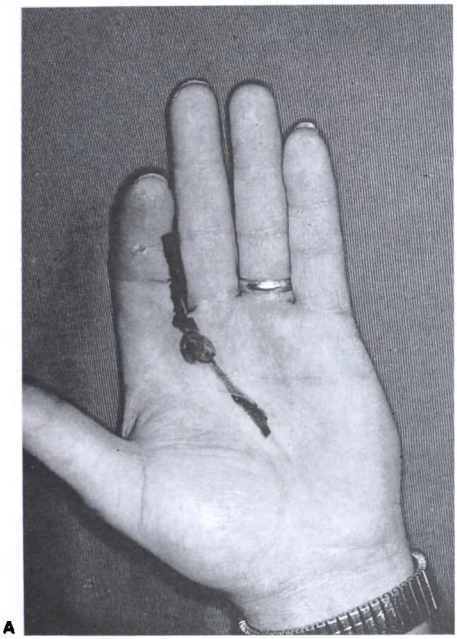
Fig. 25.5 Patient AG: a 35-year-old labourer who had a severe laceration of the right ring finger at age 29. This healed with a flexion contracture of the finger and shortly after he noticed thickening in the palm and finger. **A** Appearance of the right hand with scarring in in the finger and DD in the palm. He was treated by excision of DD and correction of the scar contracture. A full thickness skin graft was applied to the finger. He has remained free of disease for 8 years. **B** There are two nodules in the palm of the left hand which were discovered at examination at age 35. It is assumed that this patient's right hand disease appeared early because of the injury but also because of a predisposition to disease.

uncommon in the Orient and India and has been reported in black Africans. In the large series (Chapter 20), the prevalence of a single injury was the same in Japanese and northern Europeans. Thus, racial origin is not a factor which would determine whether DD is associated with a single injury.

2. *Epilepsy and diabetes mellitus*. The prevalence of both these diseases is increased in patients with DD. If the patient had either of these diseases, it would not be possible to establish a causal relationship with a single injury.
3. *Bilateral disease*. Most patients have bilateral disease when first seen, but bilateral disease in a young person is evidence of a strong diathesis. If a person had bilateral disease within 2 or 3 years of injury, it would be unreasonable to assume any causal relationship between the injury and disease.
4. *Knuckle pads and plantar nodules*. Because ectopic deposits are evidence of an increased diathesis to disease, a causal relationship could not be accepted if either was present.

Thus, to qualify for consideration of an association between a single injury and the onset of DD, the individual must be younger than the usual age of onset (less than 40 years old), be free of epilepsy or diabetes, have unilateral disease and have no ectopic deposits.

The types of injuries considered were crush injuries, lacerations and fractures (Table 25.3). It



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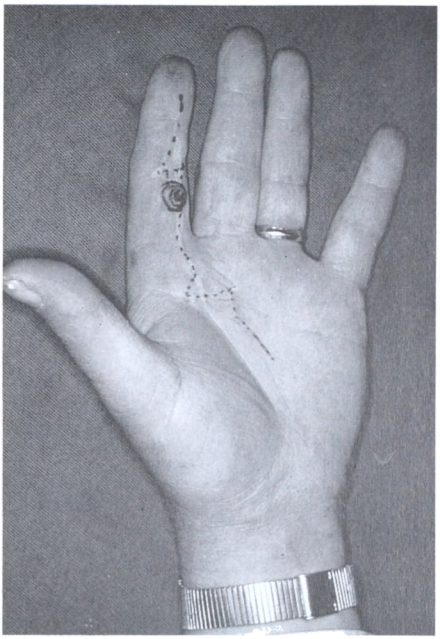

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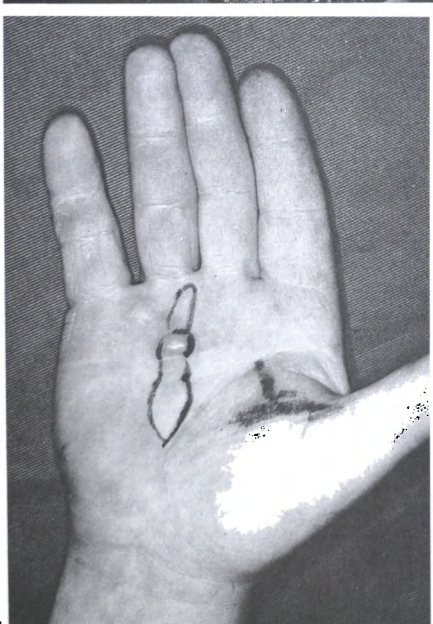

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Fig. 25.6 Patient RP: a 40-year-old farmer who cut his left hand with a chain saw at age 20. There has been gradual contraction since then. **A** Note the unusual pattern of disease which corresponds to the skin scar. There is 55° contracture at the proximal interphalangeal joint of the index finger. **B** Appearance of his hand 5 years after fasciectomy. He has full flexion and extension but has a recurrent nodule over the proximal phalanx. This has now been removed. **C** The disease in his right hand. There is a causative relationship between injury and disease in the left hand but also a predisposition to disease.

would be expected that most damage would occur from crush injuries and fractures, but lacerations produced the most convincing evidence of an association between DD and injury. These 4 patients had severe lacerations, perhaps associated with an element of crush. The lacerations themselves produced flexion contracture and this was compounded by Dupuytren's contracture. Both scar and DD were confirmed histologically.

The mechanism by which DD is precipitated by injury is not explained by our studies, although it might be due to the tethering effect of scar tissue

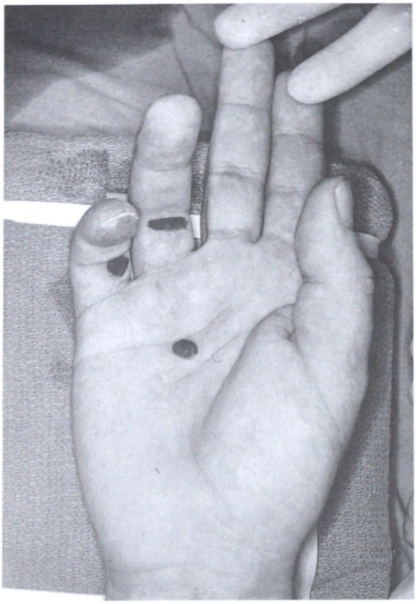

Fig. 25.7 Patient DB: a 45-year-old diesel mechanic. He had avulsed the tip of his right little finger in a drive shaft 3 months before. A split thickness skin graft was applied; this was slow to heal. He was unable to return to work because of extreme pain in the fingertip and flexion contracture of the finger. Appearance of the hand 6 months after injury when a cross-finger flap is to be applied to the fingertip. Three supposed Dupuytren's nodules are marked. The nodule within the little finger was removed and histologically found to be scar tissue. Three months later the remaining two nodules had disappeared.

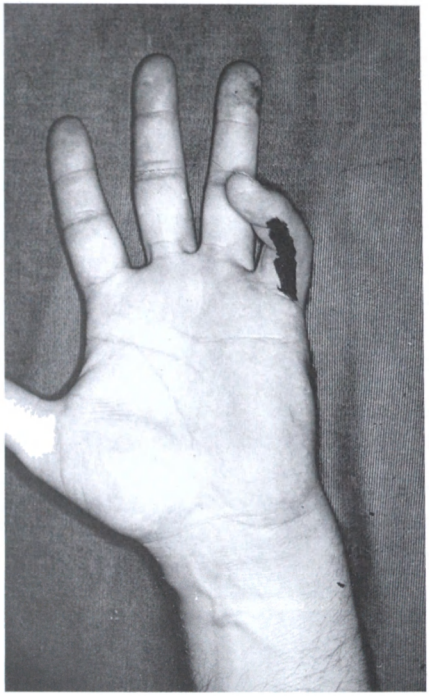

Fig. 25.8 Patient TR: a 28-year-old farmer who hit a wall with his fist, suffering a fracture of the fifth metacarpal. The finger was immobilized in flexion for three weeks. A palpable cord was present but there was no nodule. Note the slight hyperextension at the metacarpophalangeal joint. The tissue removed at operation was scar tissue.

on the fascia involved in DD. This would occur with lacerations and perhaps crush injuries. The alteration in the biomechanics at the site of injury could hasten the onset of DD in the genetically susceptable fascia. It is unlikely that swelling and immobilization of the hand were the causal agents. Both are common after severe injury, especially fractures and particularly following Colles' fracture. More permanent scarring resulting from tissue disruption is the likely cause.

The single patient with Colles' fracture (JP) in our series of 309 patients reflects the experience of Stewart et al (1985) who noted only 2 patients with joint contracture due to DD in 235 patients after Colles' fracture. On further questioning, it was found that JP had DD before her injury. Thickening of the palmar fascia following injury such as Colles' fracture is not uncommon. It is also seen in patients with reflex sympathetic dystrophy as well as after an operation for DD. This type of thickening is transient.

Only three metacarpal V fractures were selected

for this study even though many other patients in the series gave a history of a previous fracture in the hand or finger. Malunion of a fracture of metacarpal V frequently results in a compensatory flexion contracture at proximal interphalangeal joint V. A fibrous band often develops on the ulnar side of the finger originating near the tendon of insertion of the abductor digiti minimi muscle and extending distally to be attached to the skin. This band is the result rather than the cause of the flexion contracture. It is also seen in other states of flexion contracture such as malunion of the proximal phalanx, camptodactyly, and burns, and simply represents foreshortening of the normal fascia. TM, TR, and FP are examples of patients with this condition. A lateral cord is frequent in patients with DD but invariably a palpable nodule is also present.

There was no example of a hyperextension injury leading to DD although there may have been an element of hyperextension in some of the crush injuries. Gordon & Anderson (1961) presented a well documented case report supported by histological evidence of DD and Hueston (1962) reported 6 cases. Hyperextension with forceful tearing of a contracting cord can also overcome joint contracture as reported by Grace et al (1984) and many years ago by Adams (1878). It seems that hyperextension can cause or cure the disease.

A nodule in the palm or finger is thought to be a pathognomonic sign of DD, and yet in 9 of our patients the nodule either disappeared spontaneously or histologically was not DD. The Dupuytren's nodule does not disappear although it becomes less obvious as the disease progresses to joint contracture and the cords become more prominent. Thus, the disappearance of thickened fascia or discrete nodules indicates that the process was not DD.

CONCLUSIONS

Although the vast majority of injuries to the hand do not result in DD or even thickening of the palmer aponeurosis, we have shown that occasionally a single injury can precipitate the onset of DD. Presumably this occurs in genetically susceptible individuals and our studies suggest that a causal relationship can only be established in young people.

Thickening in the palm, nodule formation, or a palpable cord does not necessarily constitute DD. If a definite decision is to be made that a specific injury has precipitated its onset, a histological diagnosis of the tissue should be obtained. In this regard, it would be helpful to compensation agencies and fairer to workers and employers if criteria were established to serve as guidelines when establishing a relationship between a single injury and the onset of DD. The following are suggested:

1. The appearance of DD before age 40 in men and 50 in women suggests a causal relationship unless the individual expresses a strong diathesis such as the presence of epilepsy, diabetes, bilateral disease or ectopic deposits.
2. If the DD is bilateral, the disease in the uninjured hand should have appeared after age 40 in men and age 50 in women.
3. The injury was within the hand.
4. There is objective evidence of injury.
5. DD is in the area of the injury in the hand.
6. DD appeared within 2 years of injury.
7. Histological proof of fibromatosis is needed to make a definite diagnosis of DD.