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

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

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
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. |
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

<table>
<thead>
<tr>
<th>Patient</th>
<th>Site of disease</th>
<th>Age at injury</th>
<th>Type of injury</th>
<th>Extent of disease</th>
<th>Type of operation</th>
<th>Disease in other hand</th>
<th>Reason for category</th>
</tr>
</thead>
<tbody>
<tr>
<td>SG</td>
<td>Right</td>
<td>17</td>
<td>Crush</td>
<td>Nodule</td>
<td>None</td>
<td>None</td>
<td>Early onset</td>
</tr>
<tr>
<td>RH</td>
<td>Bilateral</td>
<td>29</td>
<td>Crush</td>
<td>Severe</td>
<td>Dermofasciectomy</td>
<td>Severe</td>
<td>Early onset</td>
</tr>
<tr>
<td>AG</td>
<td>Bilateral</td>
<td>29</td>
<td>Laceration</td>
<td>Nodule and cord</td>
<td>Dermofasciectomy</td>
<td>Nodule</td>
<td>Early onset</td>
</tr>
<tr>
<td>RP</td>
<td>Bilateral</td>
<td>20</td>
<td>Laceration</td>
<td>Nodule and cord</td>
<td>Fasciectomy</td>
<td>Progressive</td>
<td>Early onset</td>
</tr>
<tr>
<td>JO</td>
<td>Right</td>
<td>10 ?</td>
<td>Laceration</td>
<td>Nodule and cord</td>
<td>Fasciectomy</td>
<td>None</td>
<td>Early onset</td>
</tr>
<tr>
<td>RDd</td>
<td>Right</td>
<td>10 ?</td>
<td>Laceration</td>
<td>Nodule and cord</td>
<td>Fasciectomy</td>
<td>None</td>
<td>Early onset</td>
</tr>
<tr>
<td>TM</td>
<td>Right</td>
<td>24</td>
<td>Fracture</td>
<td>Nodule</td>
<td>None</td>
<td>None</td>
<td>Early onset</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patient</th>
<th>Site of disease</th>
<th>Age at injury</th>
<th>Type of injury</th>
<th>Extent of disease</th>
<th>Type of operation</th>
<th>Disease in other hand</th>
<th>Reason for category</th>
</tr>
</thead>
<tbody>
<tr>
<td>LG</td>
<td>Bilateral</td>
<td>52</td>
<td>Fracture</td>
<td>Nodule</td>
<td>Fasciectomy</td>
<td>Nodule</td>
<td>Typical disease</td>
</tr>
<tr>
<td>JP</td>
<td>Bilateral</td>
<td>56</td>
<td>Fracture</td>
<td>Severe</td>
<td>Fasciectomy</td>
<td>Progressive</td>
<td>Typical disease</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patient</th>
<th>Site of disease</th>
<th>Age at injury</th>
<th>Type of injury</th>
<th>Extent of disease</th>
<th>Type of operation</th>
<th>Disease in other hand</th>
<th>Reason for category</th>
</tr>
</thead>
<tbody>
<tr>
<td>JE</td>
<td>Right</td>
<td>53</td>
<td>Crush</td>
<td>Nodule</td>
<td>Correct scar contr.</td>
<td>None</td>
<td>Nodule disappeared Pathology</td>
</tr>
<tr>
<td>DB</td>
<td>Right</td>
<td>45</td>
<td>Crush</td>
<td>Nodule</td>
<td>Correct scar contr.</td>
<td>None</td>
<td>Pathology</td>
</tr>
<tr>
<td>RK</td>
<td>Right</td>
<td>51</td>
<td>Crush</td>
<td>Nodule</td>
<td>Excise nodule</td>
<td>None</td>
<td>Nodule disappeared Pathology</td>
</tr>
<tr>
<td>RD</td>
<td>Right</td>
<td>34</td>
<td>Crush</td>
<td>Nodule</td>
<td>Correct scar contr.</td>
<td>None</td>
<td>Nodule disappeared Pathology</td>
</tr>
<tr>
<td>JF</td>
<td>Bilateral</td>
<td>40</td>
<td>Crush</td>
<td>Nodule</td>
<td>None</td>
<td>None</td>
<td>Pathology</td>
</tr>
<tr>
<td>JL</td>
<td>Right</td>
<td>25</td>
<td>Puncture</td>
<td>Nodule</td>
<td>Fasciectomy</td>
<td>None</td>
<td>Pathology</td>
</tr>
<tr>
<td>TR</td>
<td>Bilateral</td>
<td>28</td>
<td>Fracture</td>
<td>Cord</td>
<td>Fasciectomy</td>
<td>Cord</td>
<td>Pathology</td>
</tr>
<tr>
<td>FP</td>
<td>Left</td>
<td>34</td>
<td>Electric burn</td>
<td>Cord</td>
<td>Correct scar contr.</td>
<td>None</td>
<td>Pathology</td>
</tr>
<tr>
<td>RDo</td>
<td>Right</td>
<td>61</td>
<td>Infection</td>
<td>Cord</td>
<td>Fasciectomy</td>
<td>None</td>
<td>Pathology</td>
</tr>
</tbody>
</table>

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
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
A single injury to the hand 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 susceptible 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 fas-
cia. 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 in-
jury 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 his-
tological 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 spontane-
ously 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 pal-
mer 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 agen-
cies and fairer to workers and employers if criteria
were established to serve as guidelines when estab-
lishing 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.