DUPUYTREN'S DISEASE SECONDARY TO ACUTE INJURY, INFECTION OR OPERATION DISTAL TO THE ELBOW IN THE IPSILATERAL UPPER LIMB – A HISTORICAL REVIEW

D. ELLIOT and R. RAGOOWANSI

From the Hand Surgery Department, St. Andrew's Centre for Plastic Surgery, Broomfield Hospital, Chelmsford, Essex, UK

The aggregated total of 385 cases of Dupuytren's disease arising after acute or specific injury, operation or infection of the forearm, wrist or hand between 1614 and 2003 are documented, including a personal series of 52 cases. The history of this relationship is recorded and the medico legal implications of the association are discussed.
Journal of Hand Surgery (British and European Volume, 2005) 30B: 2: 148–156

Keywords: Depuytren's disease, acute trauma, infection, historical review

INTRODUCTION

Attempts to associate the onset of Dupuytren's disease with repetitive injury as a result of chronic manual labour, including that of Dupuytren himself in 1831, have never achieved credibility. However, there is a body of evidence suggesting that the onset of Dupuytren's disease can be precipitated by an acute or specific injury, infection or operation to the ipsilateral hand, wrist or forearm (Tables 1 and 2). That this occurs only in patients with a genetic predisposition was first suggested by Skoog, proposed definitively by Clarkson and championed by Hueston (Clarkson, 1961; Hueston, 1962, 1963, 1968, 1987; Skoog, 1948).

This study reviews the historical evidence for an association of specific injury, including operations and infections, and the onset of Dupuytren's disease. Although most authors have only witnessed a few cases, the sum total of this combined experience, including our own series, is 385 patients. This study also records our own experience of 52 patients who developed Dupuytren's disease in the palm within 1 year of an acute injury, infection or operation to the same hand, wrist or forearm, having previously suffered no manifestations of this disease (Tables 3 and 4).

PATIENTS AND METHODS

Historical review

Tables 1 and 2 document all of the references we have been able to find in the last two centuries recording an association between the onset of Dupuytren's disease and a preceding acute injury, infection or operation distal to the elbow of the same upper limb which can be reasonably supported from the evidence now available.

We have excluded cases in which the injuring force was a chronic one, often work related. We have also excluded cases in which authors have not identified to our satisfaction that the disease occurred after an acute injury, although the relationship is less definite in the early references, particularly with respect to the relationship in time between injury and onset of disease. We excluded approximately 10 cases quoted by authors which we have been unable to confirm directly from writings by the treating surgeons (Skoog, 1948; de Larrard et al., 1969; James and Tubiana, 1952; Scholle, 1930). We have also excluded an unknown number of cases in German theses referenced by Skoog which we could not verify as we were unable to obtain copies of the theses (Iversen, 1909; Merker, 1897).

In addition to Clarkson's four personal cases, we have also included 37 cases reported by him as a result of direct enquiry (Clarkson, 1961). These cases have been included because the link between Clarkson and his sources is much more definite, was within relatively recent memory, and the source surgeons are recognized today as leading figures in the speciality at that time and were part of a specific attempt to establish the legitimacy of the association considered again in this paper. They include Morley's experience of 25 airmen who had developed Dupuytren's disease after a specific injury of the palm (Clarkson, 1959, 1961).

In 1956, Plewes reported a study in which 37 industrial workers developed reflex sympathetic dystrophy after upper limb injuries, of which 32 were distal to the elbow. Three patients with pre-existing Dupuytren's disease experienced worsening of their disease during the episode of reflex sympathetic dystrophy and 33 of the other 34 patients developed 'palpable thickening of the palmar fascia' in their swollen hands. We have taken this to be the onset of Dupuytren's disease as most clinicians would consider such palmar thickening to be Dupuytren's disease, although Plewes only used the term 'Dupuytren's' in the five patients who went on to develop an actual finger contracture. While it 'is impossible to determine the exact figure, at least 28 of the 32 patients with injuries distal to the elbow developed palmar fascial changes.

Hueston (1962, 1968) reported 16 cases of Dupuytren's disease arising after trauma or infection distal to

Table 1—Dupuytren's disease secondary to acute injury, infection or surgery – before 1950 ($n = 62$)

Author (Year)	*Number of cases*
Plater (1614)	1
Dupuytren (1831, 1833)	2
Goyrand (1835)	3
Smith (1885)	2
Rinne (1888)	1
Kisgen (1889)	2
Anderson (1895)	7
Bähr (1895)	1
Ledderhose (1897)	2
Nichols (1899)	4
Féré and Demanche (1903)	1
Russ (1908)	1
Kaern (1912)	2
Wendenburg (1913)	2
Horák (1914)	1
Marwedel (1927)	2
Scholle (1930)	1
Kohlmayer (1935)	10
Schaefer (1936)	6
Skoog (1948)	11

Table 2—Dupuytren's disease secondary to acute injury, infection or surgery – since 1950 ($n = 323$)

Author (Year)	*Number of cases*
James and Tubiana (1952)	30
Bacorn and Kurtzke (1953)	4
Plewes (1956)	28
Morley (1959)	25
Larsen et al., (1960)	1
Clarkson (1961)	16
Gordon and Anderson (1961)	1
Hueston (1962, 1968)	54
Comtet et al., (1968)	1
Wrobleswski (1973)	7
Fisk (1974)	22
Mikkelsen (1978)	47
Cooney et al., (1980)	3
Stewart et al., (1985)	21
McFarlane and Shum (1990)	7
Kelly et al., (1992)	1
Lanzetta and Morrison (1996)	3
Elliot and Ragoowansi (2004)	52

the elbow in 1962 and 54 cases in 1968. Although not stated in the second paper, we have assumed that the first 16 cases were included in the 54 reported in 1968.

This study

Fifty-two patients treated in our unit within the period 1989 to 2003 are included in this paper (Tables 3 and 4). The length of time between the injury and onset of Dupuytren's disease varied between 4 weeks and 1 year, with a mean of 5 months. Twenty-five patients were admitted initially as emergencies for treatment of acute injuries of the upper limb and developed Dupuytren's disease during rehabilitation of the original trauma. Twelve patients developed Dupuytren's disease after elective hand surgery in our unit. Fifteen patients were referred by their family doctors with established Dupuytren's contractures, who, on specific questioning, related the onset of disease to a preceding acute injury of the same upper limb.

The patients included 30 men and 22 women with a mean age of 54 (range, 22–75) years. Five had a positive family history, two were insulin-dependent diabetics and 18 were regular smokers. None suffered from epilepsy or were known alcoholics.

Nineteen patients sustained sharp injuries of the fingers, hand or forearm. Thirteen sustained blunt crush injuries to their fingers and hands, resulting in closed metacarpal and/or phalangeal fractures. The other injuries were an amputation of a finger tip, a revascularization of a finger, an abscess in the palm, a mixed thickness flame burn to the whole palm, a Colles fracture, a scaphoid fracture, a hyperextension injury of the wrists without fracture in a fall onto the outstretched hands and a hyperextension injury of two fingers (one case of each). Twelve patients developed Dupuytren's disease following uncomplicated elective surgery, three after trigger finger releases, two after trapeziectomy and seven after open carpal tunnel decompressions. One of the latter developed disease in both hands within 2 months of bilateral carpal tunnel decompressions.

RESULTS

Historical review

In total, a relationship between Dupuytren's disease and a preceding acute injury to the same upper limb distal to the elbow was sustantiated in 333 cases prior to this study (Tables 1 and 2).

This study

The 52 patients in our series had a history of trauma occurring less than 1 year before the onset of Dupuytren's disease. In 34 of the 52 patients, this interval was less than 6 months (Tables 3 and 4).

The 25 patients initially admitted to our unit as emergencies developed disease between 1 and 11 months (average, 6 months) after the injury. The 12 patients who developed Dupuytren's disease after elective hand surgery, developed it between 2 and 7 months (average 4 months) after surgery. The 15 patients who were referred by their family doctors had had an acute injury

Table 3—Dupuytren's disease arising within 6 months of injury ($n = 34$)

Age/sex/hand	*Occupation*	*Risk factors*	*Injury*	*Injury to disease (months)*	*Presentation*	*Progression to surgery*
57/F/Right[2]	Factory worker	Smoker	Carpal tunnel decompression	2	Nodule palm LD4 ray	Nil at 18 months
68/F/Right[2]	Housewife	None	Carpal tunnel decompression	6	Nodule palm RD1 ray	One operation
49 /M/Right[1]	Car mechanic	Smoker	RD5 crush injury + P2 fracture	6	Nodule & Cord RD5 ray	Multiple operations
54/M/Right[1]	Carpenter	Family history	Wrist laceration	1	Nodule & Pits palm RD3 ray	Nil at 18 months
53/F/Right[2]	N/A	Smoker	RD4 trigger release	6	Nodule palm RD4 ray	Nil at 48 months
53/M/Right[3]	Foreman	None	RD4,5 crush injury + P1 fractures	2	Cord RD4 ray	Multiple operations
66/M/ Right[2]	Retired	Smoker	RD1 trigger release	3	Nodule palm RD1 ray	Nil at 53 months
55/ M/Right[1]	Printer	None	RD2,3,4,5 crush + P1 and P2 fractures	4	Nodule palm RD4 ray	Nil at 51 months
33/M/Right[1]	Chemist	Family history	RD3 crush injury + MC fracture	5	Nodule palm RD3 ray	Multiple operations
44/M/Right[1]	Plumber	None	LD3 crush injury + P2 fracture	2	Nodule & cord palm LD3 ray	Nil at 48 months
59/F/Right[2]	Retired	None	Carpal tunnel decompression	4	Nodule palm RD4 ray	Nil at 12 months
41/M/Right[3]	N/A	Smoker	RD4 tip amputation	6	Nodule & Pit palm RD4 ray	Nil at 73 months
61/F/Right[1]	Retired	None	Wrist laceration	4	Nodule palm RD3 ray	Nil at 69 months
26/M/Right[1]	Student	Smoker	Palmar laceration	6	Pits palm RD3 ray	Nil at 96 months
70/F/Right[1]	Retired	None	RD3 extensor laceration	4	Nodule palm RD3 ray	Nil at 25 months
40/M/Right[1]	Carpenter	Smoker	LD3,4 lacerations	6	Nodule palm LD3 ray	Nil at 24 months
48/M/Right[1]	Plumber	Smoker	Palmar laceration	6	Nodule palm RD4 ray	Nil at 10 months
52/M/Right[1]	Carpenter	Smoker	Forearm laceration	4	Nodule & cord RD3 ray	Nil at 3 months
75/M/Right[1]	Retired	None	Palmar abscess	2	Cords palm RD1 ray	Nil at 3 months
43/F/Right[3]	Housewife	None	RD5 crush + metac fracture	2	Cord and Pit Palm RD4 ray	Nil at 18 months
70/F/Right[1]	Retired	IDDM	RD3 laceration	5	Nodule palm RD3 ray	Nil at 33 months
58/F/Right[2]	Housewife	None	Trapezectomy	3	Nodules palm RD3 RD4 rays	Nil at 3 months
32/M/Left[1]	Office worker	Family history	LD1 dorsal laceration	2	Nodule LD1 ray	Nil at 6 months
59/F/Left[2]	Housewife	Family history	Carpal tunnel decompression	4	Nodule palm RD3 ray	Nil at 3 months
70/F/Right[2]	Retired	None	Trapezectomy	6	Nodules palm RD1,3,4 rays	Nil at 3 months
49/M/Left[1]	Timber F"man	Smoker	LD2,3,4 tips crush + fractures/amputs	3	Nodules Palm LD3,4 rays	Nil at 10 months
75/F/Right[3]	Retired	None	Colles Fracture	3	Nodule palm RD4 ray	Nil at 6 months
45/M/Right[1]	Machine op.	None	RD2,3,4 crush + RD2 pp fracture	6	Nodule Palm RD4 ray	Nil at 14 months
57/M/Left + Right[2]	Builder	None	Carpal tunnel decompression (bilat)	2	Cords palm LD1 + RD1 rays	Nil at 3 months
58/F/Right[2]	Retired	None	Carpal tunnel decompression	2	Nodules palm RD3,4 rays	Nil at 3 months
69/F/Right[3]	Housewife	None	Fall with wrist hyperextension	1	Nodules palm RD3,4 rays	Nil at 3 months
38/F/Left[3]	Nurse	None	Scaphoid Fracture	2	Cord L First Web Space	Nil at 6 months
46/F/Right[1]	Interpreter	None	Revascularization RD4	3	Nodule palm RD4 ray	Nil at 4 months
50/F/Right[2]	Housewife	None	Carpal tunnel decompression	3	Nodule palm RD4 ray	Nil at 3 months

[1]Developed after admission for acute injury in our unit.
[2]Developed after elective surgery in our unit.
[3]Presented electively for treatment with a history of Dupuytren's disease developing after acute injury.

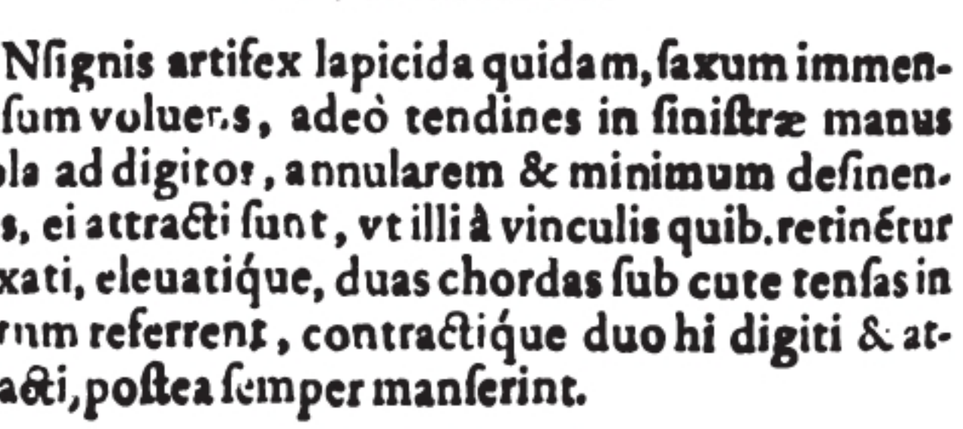
Table 4—Dupuytren's disease arising within 6 to 12 months of injury ($n = 18$)

Age/sex/ hand	*Occupation*	*Risk factors*	*Injury*	*Injury to disease (months)*	*Presentation*	*Progression to surgery*
22/M/Right[1]	Mechanic	None	Forearm laceration	10	Nodule palm RD5 ray	Nil at 14 months
60/M/Left[1]	N/A	Smoker, IDDM	LD5 crush injury + P1 fracture	7	Nodule palm LD5 ray	Nil at 24 months
75/M/Right[1]	Retired	None	LD2,3,4 lacerations	10	Nodule palm LD4 ray	Nil at 60 months
66/F/Right[3]	Retired	Smoker	LD5 laceration	8	Nodule palm LD5 ray	Multiple operations
39/F/Right[3]	Housewife	None	Palmar laceration	10	Nodule & cord palm RD3 ray	Nil at 37 months
60/M/Left[3]	N/A	Smoker	LD5 crush injury + P1 fracture	7	Nodule palm LD5 ray	Nil at 24 months
63/M/Right[3]	Plumber	None	Palmar burn	9	Nodule palm RD5 ray	Nil at 84 months
64/F/ Right[1]	Housewife	None	RD2,3 crush injuries + MC fractures	7	Nodule palm RD2 ray	Nil at 39 months
73/F/Right[2]	Retired	Smoker	RD4 trigger release	7	Nodule palm RD4 ray	One operation
59/M/Right[1]	Plumber	None	RD4 laceration	11	Nodule & Cord RD4 ray	Nil at 24 months
27/M/Right[3]	Carpenter	Smoker	LD 4,5 crush injuries + P2 fractures	7	Nodule palm LD4 ray	Nil at 91 months
40/M/Left[3]	Electrician	Smoker	LD2,3,4 lacerations	7	Cord palm LD3 ray	Nil at 97 months
58/M/Right[1]	Carpenter	Smoker	RD2,3,4 lacerations	11	Cord palm RD3 ray	Nil at 45 months
75/M/Right[3]	Retired	None	Palmar laceration	11	Pit palm RD4 ray	Nil at 48 months
54/M/Right[1]	N/A	Family history	LD2,3,4 lacerations	10	Pit palm RD3 ray	Nil at 40 months
62/M/Right[3]	Retired	None	LD2 laceration	11	Nodule palm LD2 ray	Nil at 81 months
57/F/Left[1]	Teacher	None	Hyperextension LD3,4	10	Nodule palm LD4 ray	Nil at 12 months
45/M/Left[3]	Electrician	Smoker	LD4 tip crush + P3 fracture	8	Nodule palm LD4 ray	Nil at 24 months

[1]Developed after admission for acute injury in our unit.
[2]Developed after elective surgery in our unit.
[3]Presented electively for treatment with a history of Dupuytren's disease developing after acute injury.

between 1 and 11 months (average, 7 months) prior to the onset of Dupuytren's disease.

In 42 patients (81%) the disease presented in the form of a pit with an associated proximal cord or a nodule and/or cord which grew rapidly over a period of weeks and then remained static, causing no significant morbidity. Ten patients (19%) developed a definite finger contracture with six (12%) of these requiring surgery. Two (4%) underwent a fasciectomy of the affected ray, since which they have remained disease-free. Subsequent to the initial fasciectomy of the affected ray, the remaining four patients (8%) developed further disease in the same ray which was treated by dermofasciectomy. All four have subsequently developed further disease elsewhere in the same hand which has required surgery. Two of the four (4%) have also developed extensive disease in the other hand. There appear to be no particular features, such as young age, predisposing factors or diathesis which identifies patients likely to develop progressive disease (Tables 3 and 4).



Contractio digitorum ſiniſtræ manus, in volam illius.

INſignis artifex lapicida quidam, ſaxum immenſum voluens, adeò tendines in ſiniſtræ manus vola ad digitos, annularem & minimum definentes, ei attracti ſunt, vt illi à vinculis quib. retinétur laxati, eleuatique, duas chordas ſub cute tenſas in altum referrent, contractique duo hi digiti & attracti, poſtea ſemper manſerint.

Fig. 1 Plater's description of Dupuytren's contracture, 1614.
Contraction of the fingers of the left hand into the palm. A certain well-known master mason, on rolling a large stone, caused the tendons to the ring and little fingers in the palm of the left hand to cease to function. They contracted and in doing so were loosed from the bonds by which they are held and became raised up, as two cords forming a ridge under the skin. These two fingers will remain contracted and drawn in forever (Plater, 1614).

DISCUSSION

Plater was the first to record the onset of Dupuytren's disease after an acute injury (Fig 1) (Plater, 1614). The wine merchant from the Quai d'Orsay, described in detail by Dupuytren, also clearly sustained an acute injury prior to developing a contracture (Fig 2) (Dupuytren, 1831). However, it was Goyrand who

En 1811, M. L...., marchand de vins en gros, quai de la Tournelle, nº 25, ayant reçu un grand nombre de pieces de vins du Midi, pièces qui sont ordinairement fort volumineuses, et voulant aider ses ouvriers à les ranger dans son magasin, en les entassant les unes sur les autres, ce qu'on appelle, en termes de commerce, *gerber*, essaya de soulever l'une d'elles, en plaçant la main gauche au-dessous du rebord saillant formé par l'extrémité des douves, et ressentit au même instant un craquement et une légère douleur dans la partie interne de la paume de cette main. Il conserva, quelque temps ensuite, de la sensibilité et de la raideur dans cette même main; cependant peu à peu ces symptômes se dissipèrent, en sorte qu'il y fit peu d'attention, et que ce ne fut qu'au bout d'un certain temps qu'il s'aperçut que l'annulaire tendait à se rétracter et à s'incliner vers la paume de la main, sans pouvoir être relevé autant que les autres.

Fig. 2 Dupuytren's description of the case of the wine merchant of Paris.

In 1811, Mr. L..., a wine wholesaler from number 25 Quai de la Tournelle, having received a large number of casks of wine from South France and wanting to help his workers to store these very large casks in his shop, was piling one on top of the other, which is known in the trade as stacking. Trying to lift one of them by placing his left hand under the projecting rim formed by the end of the staves, he felt, as he did so, a crack and a slight pain deep in the palm of that hand. For some time after, he felt some tenderness and stiffness in the same hand; however, little by little, the symptoms disappeared, until finally he hardly noticed them and only after a while did he realize that the ring finger was starting to contract down towards the palm, without being able to extend as much as the other fingers (Dupuytren, 1831).

should be credited with first identifying a relationship between Dupuytren's disease and an acute injury of the forearm, wrist or hand (Goyrand, 1835; Kelly et al., 1992). In 1891, Anderson discussed what he called 'False Dupuytren's disease – traumatic form'. He appears to have considered this to be an entirely different and milder condition from true Dupuytren's disease, stating that it rarely progressed beyond the site in the palm at which it first appeared and noting other differences in the clinical behaviours of the two conditions (Fig 3).

In 1952, James and Tubiana reported 30 cases of Dupuytren's disease which appeared after injuries distal to the elbow. In 1961, Clarkson reported his research into this relationship, including his own cases and those of 15 very distinguished surgeons in Europe and North America to whom he had sent a questionnaire (Clarkson, 1961). In a study of 220 cases of Dupuytren's disease reported in 1962, Hueston documented 11 cases of hand injury and five of forearm injury occurring less than 6 months prior to the patients developing Dupuytren's disease (Hueston, 1962). The following year, he commented that this and other evidence linking the onset of disease to prior injury appeared 'to be of some statistical significance, being higher than would be allowable as mere coincidence' (Hueston, 1963).

CRITERIA FOR RECOGNITION

OF

DUPUYTREN'S CONTRACTURE AFTER ACUTE INJURY

1. **Onset follows a single injury to the hand.**
2. **The age of the patient is irrelevant.**
3. **Active disease progresses rapidly from the point of injury in the hand then stops.**
4. **Disease does not progress to other parts of the same hand or to the contralateral hand.**
5. **Disease is generally mild.**
6. **Disease does not recur after surgery.**

(Anderson, 1891)

Fig. 3 Criteria for recognition of Dupuytren's contracture after acute injury, (Anderson, 1891).

Although no statistics were presented, Hueston subsequently championed the association of a specific, single injury and the onset of disease and suggested that the appearance of disease within 6 months of a specific injury should be accepted as indicating a causative relationship. Five years later, Hueston had identified 54 cases from a series of 400 of Dupuytren's disease in which the onset of disease was within 6 months of a specific injury to the forearm, wrist or hand (Hueston, 1968). In 1968, Hueston introduced the idea that an operation for Dupuytren's disease is 'the commonest major injury to the hand of these patients with Dupuytren's contracture' and that extension of the disease in the unoperated areas of the hand may be seen within weeks of an operation. Other authors have recorded the onset of Dupuytren's disease after various kinds of surgery to the hand (Lanzetta and Morrison, 1996; Wrobleswski, 1973) and our study identifies a further 12 such cases. Even if papers reporting smaller numbers of cases are ignored, these and other previous studies with substantial numbers of cases, and our own experience of 52 patients, are sufficient to support Goyrand's hypothesis that an acute injury to the forearm, wrist or hand may trigger the onset of Dupuytren's disease in the ipsilateral palm in some individuals.

While early authors often do not specify the exact time interval between the injury and the appearance of the first sign of Dupuytren's disease, the more recent literature suggests that an interval of 1 year or less has been generally adopted as acceptable for making this association. Approximately 65% of cases in our own series presented within 6 months and the remainder during the following 6 months. However, all of Hueston's cases occurred within 6 months of injury

(Hueston, 1962, 1968) and the association is possibly more credible when disease appears within this period.

Most would concur with Anderson's opinion that disease arising after acute injury is milder than true Dupuytren's disease, often remaining confined to the palm and without finger contracture. However, 20% of our series developed progressive and more significant disease, including disease in the contralateral hand. Therefore, patients should be warned of the possibility of progression of the disease.

A number of authors in the past have discussed bilateral cases and some have expressed caution, occasionally disbelief, in accepting such cases as being of traumatic origin (Clarkson, 1961; Fisk, 1959; Hueston, 1962, 1963; James and Tubiana, 1952; McFarlane, 1991; McFarlane and Shum, 1990; Plewes, 1956; Skoog, 1949). Over and above those cases of bilateral disease which occur after bilateral injury (Goyrand, 1835; James and Tubiana, 1952), this study and others (Fisk, 1959; Kelly et al., 1992; Skoog, 1949) would suggest that disease may occur in the other, and uninjured, hand, albeit rarely. The disease in the other hand normally appears later than that in the injured hand, may worsen as time passes and may overtake that of the injured hand.

Beyond the accumulated total of cases reported by surgeons over nearly 200 years, specific evidence to support this association is relatively sparse. Mikkelson found a previous history of acute hand trauma in a higher proportion of the men and women with Dupuytren's disease than in the normal population of a small town on the Norwegian coast in which he worked for many years as an orthopaedic surgeon, although only 26% of 179 patients developed disease within 1 year of the injury (Mikkelsen, 1978). In 1935, Kohlmayer reported an incidence of Dupuytren's disease after the fractures of the radius in 5% of 110 men and 0.75% of 530 women in Vienna during the period 1928 to 1934 (Kohlmayer, 1935). A more recent and prospective study of complications following Colles' fracture reported an incidence of Dupuytren's disease of 4% at 3 months and 11% at 6 months from fracture (Stewart et al., 1985). These authors concluded that this incidence was statistically significantly higher than the predicted incidence of 4.2% for this age-group of patients (Early, 1962).

In Hueston's 1962 and the 1968 series, he also identified six cases in which onset of disease occurred after more proximal injuries of the upper limb. Others have also documented onset of the disease after injuries proximal to the elbow (James and Tubiana, 1952; Plewes, 1956).

Two mechanisms whereby acute injury might precipitate the onset of Dupuytren's disease have been suggested. In 1949, Skoog found 'microruptures' in the diseased fascia and suggested that hyperextension resulted in tears of the palmar fascia which triggered a chronic inflammatory reaction leading to Dupuytren's disease. Gordon and Anderson reported a clinical case in which such an injury appeared to have precipitated Dupuytren's disease (Gordon and Anderson, 1961) and Larson et al. (1960) were able to reproduce these lesions in the palmar aponeurosis of the monkey as a result of partial rupture of the palmar fascia mechanically. Hueston was of the opinion that the association was due to hand swelling and immobility, coupled with local vasomotor disturbance (Hueston, 1968). In support of this opinion, he quoted Plewes' study of industrial workers who developed palmar Dupuytren's disease as a result of reflex sympathetic dystrophy after upper limb injury (Plewes, 1956).

Under circumstances in which any one hand surgeon is only likely to see a few cases of a phenomenon in a working lifetime, historical aggregation of the total surgical experience merits at least the attention we accord to single case reports of rare events and pathologies. This evidence, which appears to have been volunteered largely without hope or intention of material gain over a period of more than two centuries, suggests that the scepticism with which Fisk viewed the 22 cases of disease arising after a single injury to one hand which he reported in a study of 66 men occupied in the maritime trade was unjustified (Fisk, 1974). Contrary to his conclusion, which has been held up repeatedly as the definitive evidence that acute trauma cannot precipitate the onset of Dupuytren's disease, the facts of this paper support the association. Skoog emphasised that there was no reason to doubt the truth of his own patients, 22% of whom made the association spontaneously and without question of compensation. Perhaps as pertinent was his statement that the majority of patients with this disease deny the influence of trauma as an aetiological factor (Skoog, 1948).

Hueston supported Clarkson's qualification of the relationship as one in which disease had been precipitated at an earlier point in time in a susceptible individual who might have developed Dupuytren's contracture at a later stage, rather than one of direct causation of the disease by the trauma, as have the other most influential writers on this subject during the last 100 years (Clarkson, 1961; Hueston, 1963, 1987; McFarlane, 1991; Skoog, 1948). Although Hueston's earlier writings acknowledge this as only opinion, later this had moved to a more definite statement of an association "in those who are predisposed racially to the condition" (Hueston, 1987). Largely as a result of his considerable writings on this subject, his view of the relationship (and its legal consequences) is commonly held worldwide. Although propagated most enthusiastically by Clarkson and Hueston, it is likely that it originated from Skoog's statement that "accidental lesions of the hand are extremely common, but are only exceptionally followed by Dupuytren's contraction, thus demonstrating the decisive importance of predisposition ….." (Skoog, 1948). Unfortunately, this supposition cannot be substantiated from the documentation of the

recorded cases in the literature and would be difficult or, possibly, impossible to prove. It is questionable whether seeking a familial presence of disease in individual cases in the future would be helpful as individual patients' histories, in this respect, are notoriously unreliable (Ling, 1963).

An association between acute injury and the onset of this disease has obvious medicolegal significance. The data summarized in our paper does not constitute definite proof of fact. Nevertheless, we should advise the legal profession that Dupuytren's disease has been observed to appear after acute injury, infection or surgery distal to the elbow of the same upper limb in several hundred cases and that weighted opinion supports the concept that this only occurs in genetically predisposed individuals. That nearly 20% of the cases in this study progressed to contracture and a lesser number to surgery might also be considered appropriate information. Whether the patient can prove that there was no pre-existing palmar disease in the injured upper limb might then be a legal point of contention (Ross, 1999). That these individuals might already have had an inherited predisposition and that they might have developed the disease at a later date also begs the legal questions as to how much earlier the disease had been brought on in that particular individual and, therefore, how much additional disability had been caused by earlier onset of disease as a result of the injury. The literature does not reveal whether acute onset disease which remains confined to the palm and without contracture does eventually progress later in the patients' lives as no authors have included long-term follow-up of their cases.

In an attempt to establish criteria which would identify cases in which a relationship between a single injury and the onset of Dupuytren's disease is a reasonable supposition, McFarlane and Shum (1990) suggested seven criteria which should be fulfilled. In 1991, McFarlane modified these slightly and reduced them to six (Fig 4). We feel that this list is confusing, particularly in its attempt to exclude diathesis patients and regulate the age of patients in whom this association may be made. Our own cases and those in the literature with adequate documentation, other than McFarlane's own series, would suggest that limitation of the association to any particular age grouping of patients and/or to those without a diathesis is not justified. The place of histological proof in confirming the presence of Dupuytren's disease is also probably small. Most clinicians make a diagnosis of Dupuytren's disease and continue management of most cases on clinical grounds and without biopsy. Biopsy for legal purposes seems unwarranted, particularly as most of these cases will only have palmar disease, with no contracture, and no clinical indication for surgery. When disease remains in its earliest stage, the likelihood of progression in the short term is small and the legal cost implication small. Where the disease has progressed beyond a palmar nodule before or during the legal process, the diagnosis of Dupuytren's disease is unlikely to be in doubt and the need for compensation greater. However, the possibility of surgery, and the possibility of providing material for

CRITERIA FOR RECOGNITION

OF

DUPUYTREN'S CONTRACTURE AFTER ACUTE INJURY

1. **The appearance of Dupuytren's disease before age 40 in men and 50 in women suggests a causal relationship unless the individual expresses a strong diathesis to Dupuytren's disease.**
2. **If the Dupuytren's disease is bilateral, the disease in the *uninjured* hand should have appeared after age 40 in men and age 50 in women (to exclude a strong diathesis).**
3. **There is objective evidence of injury in the hand.**
4. **Dupuytren's disease is in the area of the injury within the hand.**
5. **Dupuytren's disease appeared within 2 years of injury.**
6. **When scar contracture is present, histologic proof of co-existing Dupuytren's disease is advisable.**

(McFarlane, 1991)

Fig. 4 Criteria for recognition of Dupuytren's contracture after acute injury, (McFarlane, 1991).

CRITERIA FOR RECOGNITION

OF

DUPUYTREN'S CONTRACTURE AFTER ACUTE INJURY

1. **There is objective evidence of injury with no evidence of Dupuytren's disease prior to the injury.**
2. **The injury was within the same hand, wrist or forearm as the first hand to develop disease.**
3. **The patient may be of any age and may or may not exhibit conditions predisposing to Dupuytren's disease or indicative of a diathesis.**
4. **Disease appears within 1 year of injury.**
5. **A single nodule or band appears first in the palm of the injured hand.**
6. **Disease commonly remains limited to the part of the hand initially involved but may progress within the same hand or to the other hand and may occasionally become significant in degree.**

(Elliot and Ragoowansi 2004)

Fig. 5 Criteria for recognition of Dupuytren's contracture after acute injury, (Elliot and Ragoowansi, 2004).

histology, is also correspondingly greater. Therefore, we would suggest that criteria closer to those originally proposed by Goyrand and by Anderson (Anderson, 1891; Goyrand, 1835) be used to substantiate this association (Fig 5).

References

Anderson W (1891). Contractions of the fingers and toes; their varieties, pathology and treatment. Lecture 1 Part 2. Lancet, 2: 57–59.

Bacorn RW, Kurtzke JF (1953). Colles fracture. A study of two thousand cases from the New York State Workman's Compensation Board. Journal of Bone and Joint Surgery, 35A: 643–658.

Bähr Von F (1895). Kurze Bemerkung zu dem Artikel Bieganski's über die Dupuytren'sche Contractur. Deutsche Medicinische Wochenschrift, 21: 540.

Clarkson P (1959). The role of trauma in the aetiology of Dupuytren's disease. *Presented at the eighth meeting of the Second Hand Club in 1959*, Proceedings of the Second Hand Club, Westbury Press, 1975, p. 45–46.

Clarkson P (1961). The aetiology of Dupuytren's disease. Guy's Hospital Reports, 110: 52–62.

Comtet JJ, Auffray Y, Creyssel J (1968). Le traitement chirurgical après échec du traitement non sanglant dans les fractures de l'extrémité inférieure du radius. Acta Orthopaedica Belgica, 34: 505–522.

Cooney WP, Dobyns JH, Linscheid RL (1980). Complications of Colles' Fractures. Journal of Bone and Joint Surgery, 62A: 613–619.

de Larrard J, Hitier CP, Dervillee P, Doignon J, Robert M (1969). Le role du traumatisme dans l'etiologie de la maladie de Dupuytren. Archives des Maladies Professionnelles de Medecine du Travail et de Securite Sociale, 330: 721–724.

Dupuytren G (1831). De la rétraction des doigts par suite d'une affection de l'aponévrose palmaire—Description de la maladie—Operation chirurgicale qui convient dans ce cas. Compete rendu de la clinique chirurgicale de l'Hopital Dieu par MM, les docteurs Alexandre Paillard et Marx. Journal Universel et Hebdomodaire de Médecine et de Chirurgie Pratiques et des Institutions Médicales, 5: 349–365.

Dupuytren G (1833). Revue de la clinique chirurgicale de M. Dupuytren durant les mois de décembre et de janvier. Gazette Medicale de Paris, 2s, 1: 111–113.

Early PF (1962). Population studies in Dupuytren's contracture. Journal of Bone and Joint Surgery, 44B: 602–613.

Féré C, Demanche R (1903). Note sur un cas de rétraction de l'aponevrose palmaire. Revue de Chirurgie, 28: 324–328.

Fisk G (1959). The relationship of trauma and Dupuytren's contracture. *Presented at the 8th meeting of the Second Hand Club in 1959*, Proceedings of the Second Hand Club, Westbury Press, 1975, p. 481–483.

Fisk G (1974). The relationship of trauma and Dupuytren's contracture. In: Hueston JT, Tubiana R (Eds) *Dupuytren's disease*. Churchill Livingstone: Edinburgh, 43–44.

Gordon S, Anderson W (1961). Dupuytren's contracture following injury. British Journal of Plastic Surgery, 14: 129–131.

Goyrand G (1835). De la rétraction permanente des doigts. Gazette Médicale de Paris, 2s, 3: 481–486.

Horák J (1914). Über Dupuytren'sche Kontraktur. Zentralblatt für Chirurgie, 41: 78–79.

Hueston JT (1962). Further studies on the incidence of Dupuytren's contracture. The Medical Journal of Australia, 1: 586–588.

Hueston JT. *Dupuytren's Contracture*. Edinburgh and London, Livingstone, 1963: 73–75.

Hueston JT (1968). Dupuytren's contracture and specific injury. Medical Journal of Australia, 1: 1084–1085.

Hueston J (1987). Dupuytren's contracture: medicolegal aspects. The Medical Journal of Australia, 147: S2–S11.

Iversen J. Über die traumatische Entstehung der Dupuytren'schen Kontraktur über deren Localisation am Daumen. Inaugural Dissertation, Kiel, 1909.

James JIP, Tubiana R (1952). La maladie de Dupuytren. Revue de Chirurgie Orthopedie, 38: 352–406.

Kaern H (1912). To tilfaelde af Dupuytrens Kontraktur, opstaaet efter en akut betaendelse I Fascia palmaris. Hospitalstidende, 55: 25–32.

Kelly SA, Burke FD, Elliot D (1992). Injury to the distal radius as a trigger to the onset of Dupuytren's disease. Journal of Hand Surgery, 17B: 225–229.

Kisgen P. Über Dupuytren'sche Fingerkontracturen infolge spontaner narbiger Retraktion der Aponeurosis Palmaris. Inaugural Dissertation, Würtzburg, 1889.

Kohlmayer H (1935). Zur Frage der tramatischen Entstehung der Dupuytren'schen Fingerkontraktur. Zentralblatt für Chirurgie, 62: 1928–1931.

Lanzetta M, Morrison W (1996). Dupuytren's disease occurring after a surgical injury to the hand. Journal of Hand Surgery, 21B: 481–483.

Larson RD, Takagishi N, Posch JL (1960). The pathogenesis of Dupuytren's contracture. Journal of Bone and Joint Surgery, 42A: 993–1007.

Ledderhose von (1897). Zur Pathologie der Aponeurose des Fusses und der Hand. Archiv für Klinische Chirurgie, 55: 694–712.

Ling RSM (1963). The genetic factor in Dupuytren's disease. Journal of Bone and Joint Surgery, 45B: 709–718.

Marwedel P (1927). Zur traumatischen Genese der Dupuytrensche Contractur. Zentralblatt für Chirurgie, 54: 1246–1248.

Merker E. Die Dupuytrensche Fingerkontrakur. Inaugural Dissertation, Berlin, 1897.

McFarlane RM, Shum DT (1990). A single injury to the hand. In: McFarlane RM, McGrouther DA, Flint MH (Eds) *Dupuytren's disease: biology and treatment*. Churchill Livingstone: Edinburgh, 265–273.

McFarlane RM (1991). Dupuytren's disease: relation to work and injury. Journal of Hand Surgery, 16A: 775–779.

Mikkelsen OA (1978). Dupuytren's disease-the influence of occupation and previous hand injuries. The Hand, 10: 1–8.

Morley GH (1959). The role of trauma in the aetiology of Dupuytren's disease. *Presented at the eighth meeting of the Second Hand Club in 1959*, Clarkson P (Ed.), Proceedings of the Second Hand Club, Westbury Press; 1975, pp. 45–46.

Nichols JB (1899). A clinical study of Dupuytren's contracture of the palmar and digital fascia. American Journal of the Medical Sciences, 117: 285–305.

Plater F. *Observationum in hominis affectibus*. Basel, Konig & Brandmyller, 1614; vol. 3, p. 140.

Plewes LW (1956). Sudeck's atrophy in the hand. Journal of Bone and Joint Surgery, 38B: 195–203.

Rinne (1888). Ueber eine seltene Aetiologie der Dupuytren'schen Fingercontractur. Duetsche Medicinische Wochenschrift, 14: 761–762.

Ross DC (1999). Epidemiology of Dupuytren's disease. Hand Clinics, 15 (1): 53–62.

Russ R (1908). The surgical aspects of Dupuytren's contraction. American Journal of Medical Science, 135: 856–864.

Schaefer V (1936). Die Genese der Dupuytren'schen Kontraktur (Die Bedeutung der Anlage, des chronischen Traumas und des Unfalles). Zentralblatt für Chirurgie, 63: 1712–1716.

Scholle W (1930). Uber die Dupuytren'schen Fingerkontraktur unter besonderer Berücksichtigung ihres Vorkommens bei Jugendlichen. Deutsche Zeitschrift für Chirurgie, 223: 328–339.

Skoog T (1948). Dupuytren's Contraction with special reference to etiology and improved surgical treatment. Its occurrence in epileptics Note on knuckle pads. Acta Chirurgica Scandinavica, 96 (suppl): 139.

Smith N (1885). Seventy cases of Dupuytren's contraction of the palmar fascia. British Medical Journal, 1: 275–278.

Stewart HD, Innes AR, Burke FD (1985). The hand complications of Colles' fracture. Journal of Hand Surgery, 10B: 103–106.

Wendenburg F. Die Dupuytrensche Fingerkontractur. Inaugural Dissertation, Berlin, 1913.
Wrobleswski BM (1973). Carpal tunnel decompression and Dupuytren's contracture. The Hand, 5: 69–70.

Received: 29 December 2003
Accepted after revision: 13 August 2004

Mr D. Elliot, MA, FRCS, Hand Surgery Department, St. Andrew's Centre for Plastic Surgery, Broomfield Hospital, Court Road, Chelmsford, Essex CM1 7ET, UK. Tel.: +44-1621-857-362; fax: +44-1621-841-127; E-mail: info@david-elliot.co.uk

doi:10.1016/j.jhsb.2004.08.002 available online at http://www.sciencedirect.com