THE SHOULDER-HAND SYNDROME: HISTORICAL REVIEW WITH OBSERVATIONS ON SEVENTY-THREE PATIENTS*

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HISTORICAL INTRODUCTION

SHOULDER-HAND SYNDROME is a term which has been applied to a symptom complex comprising stiffness or pain in the shoulder associated with pain and swelling of the hand, in which trophic changes may be apparent.

The clinical picture was first described by Oppenheimer[1] in 1938 in a report on 14 patients with cervical disc degeneration in whom dystrophic changes occurred in the hand associated with pain and stiffness of the shoulder. This author believed that the disc degeneration was the underlying cause of the dystrophy. A comparable clinical syndrome was described by Johnson[2] in 1943, under the term post-infarction sclerodactylia, in a group of 39 patients who manifested symptoms of pain, stiffness and swelling of the hands following myocardial infarction. There was initially a non-pitting œdema of the hand which often progressed until the skin became smooth and tight, with erythema and cyanosis. As the condition progressed, dystrophic changes occurred in the skin with thickening of the palmar fascia. Osteoporosis of the bones of the hand was a constant finding in the advanced state. Pain with stiffness in the shoulder occurred in all instances, but this was felt to be unrelated to the sclerodactylia and was attributed to disuse. Similar changes in the shoulder or hand occurring after myocardial infarction have been reported by other observers,[3-8] the incidence of shoulder involvement varying from 10 to 20%. In these reports it was suggested that the shoulder involved was usually on the side of the cardiac ischæmic pain.

In 1947, Steinbrocker[18] reviewed the literature and described the syndrome in detail. He introduced the term shoulder-hand syndrome; under this heading he included the groups mentioned above and suggested that they represented varying degrees of reflex sympathetic dystrophy and neurovascular response observed at different stages. Steinbrocker divided the course of the syndrome into three main stages according to their clinical manifestations:

Stage 1. This stage, lasting three weeks to six months, is characterized by a painful shoulder with swelling and pain and stiffness in the hands and fingers. It may begin in the hand or shoulder and involve one or both upper extremities. There is diffuse swelling of the fingers and hands with limitation of finger movement but with little pitting œdema. The skin becomes smooth and may exhibit trophic changes, with desquamation in some cases. The hand may be dusky pink, pale or cyanosed.

Stage 2. This might last from three to six months. It is characterized by a gradual lessening of shoulder pain with resolution of the swelling of the hands. The hands become increasingly stiff with flexion deformity of the fingers, atrophy of subcutaneous tissues and wasting of small muscles. Early trophic skin changes may be noted with a thickening of the palmar fascia. Some patchy osteoporosis is often demonstrable.

Stage 3. This stage may last many months. It is characterized by a smooth, glossy skin with trophic changes and atrophy of subcutaneous tissues. The hand becomes atrophic with a thickened palmar fascia and Dupuytren-like contracture. Osteoporosis is usually marked.

MacLean and Stranks[10, 11] suggested a slightly different sequence of events in the clinical course. The initial complaint in the hands of their patients was stiffness which always preceded the swelling. The pain might be localized at first but tended to spread throughout the hand.

Steinbrocker,[9] in a study of 42 cases of reflex dystrophy of the upper extremity, found that 36 of these patients presented the shoulder-hand syndrome due to the following causes or associated factors:

A. Idiopathic
B. Peripheral Lesions
   1. Trauma and suppuration of the extremity (Sudeck's atrophy, causalgia, post-traumatic osteoporosis, acute bone atrophy).
   2. Vascular disease (thrombophlebitis, diffuse vasculitis, periarteritis nodosa).
   3. Intraforaminal osteoarthritis of the cervical spine (Oppenheimer).

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4. Cardiac disease—post-infarctional.
5. Other thoracic diseases (post-pneumonic, etc).
6. Nodular panniculitis (Weber-Christian).

C. Lesions of Cord and Ganglia
1. Herpes zoster.
2. Diffuse vasculitis?

D. Higher Lesions
1. Cerebral lesions (hemiplegia).

The age and sex incidence varied with the preciptating factors; for example, following coronary thrombosis the age and sex distribution was the same as in that condition.[12]

A search for the pathogenesis of shoulder-hand syndrome has provoked a wide variety of hypotheses. In discussing causalgia, de Takáts[13, 14] postulated that noxious stimuli might produce an axon reflex which could result in vasodilatation with resultant increased blood flow to the extremity. He further conjectured that stimulation of dorsal root fibres, by producing a painful vasodilator substance, could create a peripheral anæsthesia.

Oppenheimer[1] believed that the swollen atrophic hand found in association with cervical disc degeneration was the result of direct pressure on nerve roots. In his cases the syndrome occurred on the same side as the hypertrophic bony changes. He made no attempt to explain the lack of motor and sensory findings.

Boas,[3] Johnson[2] and Hilker[15] in discussing the shoulder and hand changes following myocardial infarction felt that the entire syndrome might be based on the occurrence of tissue anoxia. Boas believed that the shoulder pain represented cardiac efferent impulses which were not of sufficient intensity to produce angina. Johnson suggested that the factors responsible for the production of local changes in the hands and fingers were: (1) vasoconstriction of the peripheral arteries of the hands resulting from the cardiac pain; (2) pre-existing arteriosclerotic narrowing of the vessels of the hands; (3) anoxæmia of varying duration and intensity resulting from the myocardial infarction.

Most observers make use of the concept of Lorente de No[16] that afferent stimuli enter the cord and travel up and down in the internuncial pool of neurons. Evans[17a, b] has suggested that a prolonged bombardment of pain impulses sets up a vicious circle of reflexes spreading through the pool of internuncial neurons, up, down and even across the cord. In this circling activity, some of the sympathetic motor neurons of the lateral horn which control vasomotor tone and sweat gland mechanism are affected. Spasm in the capillaries, by raising pressure, would produce œdema. Other impulses passing into anterior horn cells might produce pain. Steinbrocker[18] supported these ideas because of the wide variety of precipitating conditions, the involvement of the sympathetic, parasympathetic and motor pathways, the lack of segmental distribution and the therapeutic success from sympathetic block.

Askey[4] observed that the painful shoulder in coronary artery disease was not always on the side of radiation of anginal pain and postulated that there must be an underlying arthritis which determined the shoulder affected. Kelly[19] agreed with the suggestion of a pre-existing joint lesion and stated that local synovial lesions in the hand and arthritic changes in the wrist and small joints of the fingers could explain the limited hand movement.

In patients with anginal pain, Russek[20] felt that abnormal tension in muscles guarding the shoulder might produce the syndrome.

Others have suggested that the use of the shoulder as a weight-bearing joint might be responsible for the development of the syndrome in patients confined to bed.

Finally, Young[21] postulated that the basic fault was a constitutional tendency for fibrous tissue deposition and contracture in the capsule of the shoulder, the small joints of the hand and the palmar fascia.

TREATMENT

Many forms of therapy have been employed in the management of this condition, including shoulder manipulation under anæsthesia, stellate ganglion block, deep x-ray therapy and steroid hormones. In conjunction with these measures, a well-planned physiotherapy program usually has been employed.

MacLean and Stranks[10] have reported good results in the first stage of this syndrome with the use of vasodilator drugs such as Priscoline. In patients where this method of treatment failed and in those in whom the condition had progressed to Stage 2, stellate ganglion block was employed. In more advanced cases in Stage 3, cervical sympathectomy was recommended.

Evans[17a] presented a group of 57 patients treated with sympathetic block and sympathec-

tomy. Eleven patients were relieved by block alone. Twenty-nine were treated by sympathectomy when block failed and 22 of these were interpreted as having an adequate result. The relief of pain with increased mobility of the shoulder and hand was considered an indication of a successful block.[22] Single stellate blocks have usually been unsuccessful; multiple injections have generally been required, the number depending on the recurrence of symptoms. Eighteen of 42 patients who were treated in this manner recovered completely after three or four injections repeated at two to seven day intervals.[23]

ACTH, cortisone, hydrocortisone and prednisone have been used with success in the treatment of the shoulder-hand syndrome. Russek,[20] using hormone therapy, obtained excellent results with early relief of pain in 13 of 17 patients. Sigler[24] reported excellent results in five of seven patients treated with ACTH. More recently, Berger[25] treated 18 patients by injecting 50 mg. of hydrocortisone into tender areas about the shoulder and reported good results in all.

Steinbrocker and associates[26] compared the results of stellate ganglion block with those of cortisone therapy in two groups of 14 patients. They came to the conclusion that both forms of therapy were equally effective but preferred stellate block. With either form of treatment, patients in Stages 1 and 2 invariably had good results in the shoulder while those in Stage 3 usually had a poor result. With regard to the hand, patients in Stage 1 had a good result while those in Stages 2 and 3 experienced some relief of pain but little improvement in function.

REPORT OF SEVENTY-THREE CASES

Material.—Seventy-three patients with shoulder-hand syndrome admitted to the wards of the Toronto General Hospital and Sunnybrook Veterans' Hospital have been reviewed. This figure gives an imperfect indication of the incidence of the syndrome in the general population and even in a hospital population; many patients in whom the symptoms have not been too disabling would not require hospitalization, and the syndrome may easily be overlooked or not recorded in the presence of other serious disease, such as cerebrovascular accident. Recognition of the syndrome is undoubtedly more difficult in patients disabled with other serious disease.

Nineteen of the 73 patients were admitted and observed during the 12-month period July 1954 to June 1955, while the remainder were spread over a seven-year period. An attempt was made to locate this group of 54 patients for re-examination and assessment of their disability. Replies to questionnaires concerning their present status were received from 24 patients; eight returned for follow-up examination; ten patients had died, and 12 could not be traced.

The average age of the group was 63 years, the youngest patient being 31 and the eldest 80 years of age. Only three patients were under 45 years of age and two of these developed the syndrome following trauma; 10 were under 55 years of age.

It is interesting to consider the associated diseases or any special factors that might possibly be precipitating influences. These are listed in Table I. It is obvious that they constitute a heterogeneous group.

TABLE I.—ASSOCIATED CONDITIONS OF POSSIBLE ETIOLOGICAL SIGNIFICANCE IN 73 CASES OF SHOULDER-HAND SYNDROME

Associated conditions	*No. of cases*
A. No noticeable associated illness	10
B. Peripheral lesions:	
(a) Traumatic	5
(b) Severe cervical disc degeneration with symptoms	10
(c) Thoracotomy for tuberculoma	1
(d) Cardiovascular disease:	
(i) *Recent myocardial infarction (within eight weeks)	18
(ii) Old myocardial infarction (six months to two years previously)	3
(iii) Essential hypertension	1
(iv) Cor pulmonale (pulmonary fibrosis)	1
(v) Electrocardiographic evidence of heart disease but no clear clinical manifestation	6
C. Lesions of spinal cord: Quadriplegia	4
D. Cerebral lesions:	
(a) Tumour	1
(b) Cerebrovascular accident:	
(i) Hemiplegia	4
(ii) Subarachnoid hæmorrhage	3
(c) Severe head injury	1
E. Miscellaneous:	
(a) Prolonged bed rest, for six months or more (cirrhosis; diverticulitis)	2
(b) Abdominal surgery	1
(c) Block dissection of the neck	1
(d) Fractured neck of femur	1

*These 18 patients gave histories typical of recent myocardial infarction. In eight patients the electrocardiograms showed significant changes; two patients had normal electrocardiograms; in the remainder (four showing bundle branch block and four showing auricular fibrillation) the electrocardiograms were abnormal but not typical of infarction.

Manifestations.—All of the patients presented with shoulder pain, limited shoulder movement and inability to fully flex and extend the fingers.

Diffuse pain in the hand, not limited to the joints, was recorded in all but four cases.

The disability involved the right upper extremity in 28 patients and the left in 23, and was bilateral in 22 patients. The shoulder was affected before the hand in 61 patients, the hand being affected first in the remainder. This variability was also noted in the 29 patients with associated cardiovascular disease; nine had involvement of the left shoulder and hand; 10, of the right side; and 10 showed bilateral involvement.

The time interval between the onset of an associated condition and the development of the lesion was analyzed. The shortest recorded was 24 hours, while the longest was four years. In the latter patient, traumatic quadriplegia had occurred four years before the onset of the shoulder-hand syndrome.

There was great variability in the limitation of shoulder movement, but abduction and rotation were most frequently affected. Twenty-five per cent of the patients had no scapulo-humeral movement (frozen shoulder). Movements of the elbow were not impaired. All but three patients had painful limitation of wrist movement. A common finding was the presence of œdema, usually non-pitting, mainly over the dorsum of the hand. There was usually a diffuse thickening of the fingers, with pain on pressure over the soft tissues. Some degree of subcutaneous atrophy was recorded in most patients, being more striking in the more advanced cases. A feature not commonly stressed was vasomotor instability. During an examination the affected hand might appear pale; with activity the colour might change to dusky pink, mottled pink and blue or pale again, all within a short time. The thickening of the subcutaneous tissues in the palm was frequently indistinguishable from early or late Dupuytren's contracture, but not uncommonly the thickening was diffuse, appearing to involve the whole palm. In this series, 40 patients had varying degrees of palmar thickening; no change was recorded in 17 patients, while the remainder were reported as having normal palms.

The sedimentation rate was normal in 49 patients and elevated (20 to 100 mm. in one hour) in 22 patients. In some of these the elevation of the sedimentation rate may have been due to other associated disease.

The hands were examined by x-ray in 39 patients. Osteoporosis was demonstrated in 24 and 15 showed no bony abnormality. Absence of osteoporosis in no way influenced the diagnosis, as it was usually a late finding; several patients had been treated and gained an excellent result before they progressed to this stage.

Treatment and results.—In an attempt to evaluate the efficacy of various forms of therapy we have graded results according to the plan outlined in Table II. Of the 73 patients, 16 received no special treatment; 20 were treated with intensive physiotherapy alone; 15 received ACTH or cortisone in addition to routine physical measures; seven were subjected to stellate ganglion block in addition to routine physiotherapy; of the remainder, four were treated with phenylbutazone, four by shoulder manipulation under general anæsthesia, four by deep x-rays and three by local injection of hydrocortisone into tender areas about the shoulder.

TABLE II.—GRADING OF RESULTS OF TREATMENT

EXCELLENT	Total relief of pain. Full range of movement in shoulder and hand.
GOOD	Relief of pain. Shoulder, hand and wrist movements within 80% of normal. Subcutaneous atrophy and palmar thickening may be present.
FAIR	Persistence of some pain. Extension and flexion of fingers within 50% of normal. Shoulder movement 50% of normal. Subcutaneous atrophy and/or swelling present with palmar thickening.
POOR	Non-functional hand, with limited shoulder movement.

In the group of 16 patients receiving no special form of treatment, 12 had a poor result; in the other four cases the records were not adequate for satisfactory assessment. Of the 20 patients treated with intensive physiotherapy, nine had good or excellent results while 11 failed to respond. The most favourable results were observed in the group treated with ACTH or cortisone; 10 of the 15 patients had an excellent or good result. The dosage of cortisone varied from 100 to 200 mg. daily over a 14-day period, after which it was withdrawn gradually. Results of stellate ganglion block were disappointing. Only one patient had an excellent result, and this after six injections. Six patients derived little benefit after three to 18 injections. Results in the remaining 15 patients treated by various other methods were fair to poor. The results are summarized in Table III.

The effect of treatment appeared to be influenced by the duration of symptoms prior to therapy. Of 39 patients treated within three

TABLE III.—RESULTS OF TREATMENT IN 73 CASES OF SHOULDER-HAND SYNDROME

Treatment	No. of cases	Result: Excellent or good	Result: Fair or poor
No special treatment	16	0	12 (4 no record)
Physiotherapy	20	9	11
ACTH or cortisone	15	10	5
Stellate ganglion block	7	1	6
Other	15	0	15

months of the onset of symptoms, 17 had excellent or good results while 22 had fair or poor results. When treatment was begun after more than three months had elapsed, only six patients achieved an excellent or good result while 24 had a fair or poor response. Four patients could not be assessed. These results are shown in Table IV. Thus of the group treated early, about

TABLE IV.—DURATION OF SYMPTOMS PRIOR TO TREATMENT AND FINAL RESULT

Duration	Result: Excellent	Good	Fair	Poor
1 month or less	3	6	10	3
1 to 3 months	4	4	7	2
3 to 6 months	1	2	5	5
6 months to 1 year	0	1	2	1
Over one year	1	1	6	5

one-half had a good result, and of those treated late only one-fifth had a satisfactory result. The patients who received stellate ganglion block were treated late in the course of their disability, which may explain the poor results. The higher proportion of good results with stellate block in other series might be due to early treatment.

An attempt was made to assess the subsequent course of the 54 patients who had been treated prior to June 1954. Ten had died and 12 could not be traced. Eight patients returned for re-examination and 24 replied to a questionnaire. The patients who had been discharged from hospital with an excellent response to treatment varied in their subsequent course: some had improved; most showed no change; a few had become worse. The patients who had shown fair or poor results tended to improve slightly: the shoulder pain had lessened but they seldom regained a functional hand. The information obtained from this follow-up study is summarized in Table V.

TABLE V.—FOLLOW-UP STUDY OF 32 CASES

Result prior to discharge	Result after 2 to 6 years: Excellent	Good	Fair	Poor
Excellent	5	0	0	1
Good	2	3	2	1
Fair	1	1	5	0
Poor	2	2	5	2
	10	6	12	4

OBSERVATIONS AND DISCUSSION

The shoulder-hand syndrome occurs in association with a wide variety of disorders. Some of these are relatively minor in nature and not sufficiently serious to require hospitalization. For this reason the true incidence of the syndrome is not represented in any hospital series. Over one-third of our patients with shoulder-hand syndrome (29 of 73) had cardiovascular disease. Eighteen of the 29 had suffered myocardial infarction within eight weeks of admission. This, however, represented only a small group of the patients treated for infarction in the two hospitals, where patients with cardiovascular disease comprise about one-third of the total hospital admissions.

Thirteen patients had a lesion of the central nervous system, but it is probable that this figure underestimates the true incidence in this group as the shoulder-hand syndrome is often seen in association with hemiplegia.

Relatively few developed the syndrome following trauma, and in none was it associated with infection. In 10 cases there was no apparent associated disease.

It appears that many unrelated conditions may be associated with the development of the shoulder-hand syndrome with no single feature common to all. Tissue anoxia with resulting hypotension, while a factor in a few, is not present in most patients. Many patients (over 25%) developed the syndrome with no antecedent history of pain. Though pain may perpetuate the syndrome once established, it is not likely the initiating factor. Pressure on nerve roots might explain the origin in some cases of severe degenerative disc disease in the cervical spine. The absence of palmar fascial thickening and permanent limitation of shoulder movement in many cases tends to weaken the theory that the syndrome is a result of constitutional fibrous dysplasia.

The basic nature of the process in the shoulder and hand remains unknown but findings suggest that the basic reaction occurs in the connective tissues of the shoulder, hand and, occasionally, the lower extremity. In some respects this might be compared to rheumatoid arthritis and other

collagen diseases where there appears to be an abnormal connective tissue reaction to unknown factors. We have been unable to obtain conclusive evidence for or against the idea that abnormal stimuli arising in sympathetic nerve fibres might initiate the chain of reactions in the connective tissues.

Interest in this lesion of the shoulder and hand should not detract from a similar train of events which may occur in other areas. One patient, 11 years of age, presented an analogous clinical picture in the left hip and foot after a minor fall. In two other patients an identical picture developed in the lower extremity following injury, and a similar lesion has been frequently observed after application of plaster casts. The symptoms in such cases are often attributed to "arthritis"; much valuable time may be lost because of failure to recognize the true nature of the lesion.

SUMMARY

A study of 73 patients with shoulder-hand syndrome is presented. The pathogenesis of the condition remains unknown, but prompt recognition and institution of therapy may prevent needless disability in many cases. The average age of the group was 63 years. Approximately 40% of the cases were associated with cardiovascular disease, most commonly myocardial infarction. In the remainder, associated diseases varied widely. There was an equal incidence of left-sided, right-sided and bilateral involvement. Predominance of left-sided lesions, suggested by others, was not found in this series.

The essential diagnostic features of the condition are pain and limitation of shoulder movement with swelling and tenderness of the hand and inability to fully flex or extend the fingers. Osteoporosis of the hand was found in over two-thirds of the patients examined and may be an indication of the severity and duration of symptoms.

Early recognition is important, for results are poor if treatment is long delayed. No form of treatment is entirely satisfactory, but the use of cortisone and ACTH in addition to judicious rest with early passive and increasing active movement gave the best results.

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RÉSUMÉ

Cet article porte sur l'étude de 73 cas du syndrome épaule-main. La pathogénie de cette affection demeure inconnue mais un diagnostic posé dès l'abord et suivi d'un traitement précoce peuvent prévenir l'installation d'une infirmité évitable dans plusieurs cas. L'âge moyen des malades de ce groupe était de 63 ans. Environ 40% d'entre eux souffraient du cœur—l'infarctus pour le plus grand nombre. Les autres présentaient une diversité d'états associés. Il y avait une distribution égale des lésions à droite, à gauche et des deux côtés à la fois. On ne put corroborer la prédilection pour le côté gauche qu'ont déjà avancée certains auteurs.

Les traits essentiels du diagnostic de cet état sont la douleur et la fixité de l'épaule ainsi que le gonflement et la sensibilité de la main et l'incapacité de pleinement fléchir ou étendre les doigts. Une ostéoporose de la main fut observée dans plus de deux tiers des cas; elle est un indice de l'intensité et de la durée des symptômes. Il importe de dépister la lésion dès son début car les résultats son piteux si le traitement est longtemps différé. Aucune forme de traitement n'est pleinement satisfaisante, mais l'emploi de cortisone et d'A.C.T.H. en plus de repos judicieux, avec mouvements passifs d'emblée suivis plus tard de mouvements actifs progressifs, a donné les meilleurs résultats.

LYMPHOCYTIC INFILTRATION OF THE SKIN (JESSNER)

Seven cases of this disease are presented by Calnan (*Brit. J. Dermat.*, 69: 169, 1957). The primary lesion is an erythematous rose-pink papule. There may be many papules and sometimes these are arranged in plaques or crescents. The usual sites are the malar region and the back or the chest. There is a heavy preponderance of males. The course is variable, with recurrences and remissions. Histologically the characteristic findings are a normal epidermis and a dense mantle of lymphocytes about the entire network of skin blood vessels. Differential diagnoses include discoid lupus erythematosus, early lymphocytic reticulosis, annular erythema and insect bites. The etiology of this condition is unknown. No case has been reported to progress to lymphoblastoma or leukæmia.