Shoulder-Hand Syndrome Following Myocardial Infarction with Special Reference to Prognosis

By Joseph Edeiken, M.D.

Shoulder-hand syndrome occurs in approximately 10 to 15 per cent of patients following acute myocardial infarction. Sympathectomy, sympathetic block, cortisone, and various other therapies have been advocated. In the present communication the results of simple measures such as local heat, analgesics, and the regimen of active use of the affected extremity are described in treating 47 attacks in 42 patients with acute myocardial infarction.

In 1936 we reported 14 patients who developed persistent pain in the shoulder region following myocardial infarction. In 1941 Askey studied a similar but larger group and described pain, swelling, and stiffness of the hands in addition to the shoulder symptoms. There have been a number of other reports upon the shoulder-hand syndrome, many of them discussing sympathectomy, sympathetic block, and cortisone, with or without physiotherapy. In most of these publications the postinfarction cases were not separated from those following trauma or hemiplegia or due to diffuse vasculitis, cervical arthritis, or panniculitis.

In 1947, Steinbrocker, describing predominantly advanced and severe cases, observed 3 stages in the evolution of the shoulder-hand syndrome. In the first, which lasted from 3 to 6 months, the patient experienced pain, tenderness, and limitation of motion at the shoulder girdle. Swelling, pain, stiffness, and discoloration of the hands and fingers usually followed, although the sequence is variable and both may occur simultaneously and gradually. In the second stage, which also lasted from 3 to 6 months, the shoulder pain and the hand swelling and discoloration gradually disappeared but the stiffness and flexion deformity of the fingers became more prominent and manifestations similar to Dupuytren’s contracture began to appear. The third stage, which may last for months or become irreversible, is characterized by progressive atrophic changes in the hands, severe atrophy of the interosseous muscles, and limitation in motion at the metacarpophalangeal and interphalangeal joints. Contracture of the flexor tendons often occurs at this stage, particularly on the ulnar side, and rolling up of the palmar and digital fascia, in many ways similar to Dupuytren’s contracture, is common. Occasionally a trigger zone may be present near or on the shoulder, pressure over which causes an increase in pain, suggesting the possibility that it may also be a manifestation of the syndrome. In 1936 we mentioned 2 cases with trigger zones.

The present report is founded upon the study of 42 patients who developed symptoms of shoulder-hand syndrome after myocardial infarction. Of this group 32 were men and 10 women. The youngest was 38 years of age; the oldest, 73. These patients received no therapy except local heat, analgesics, and a regimen of active use of the affected extremity. It is hoped that the information gained may be a useful guide to prognosis for those patients who do not or cannot receive other forms of therapy, or for whom other treatment has not been completely successful, and that it may also serve as a standard against which the effectiveness of a specific therapy may be assessed. Our results indicate that this disorder, managed simply, is essentially self-limited and is usually less severe than might be inferred from some of the reports in the literature.

Material

This study is based upon 42 patients who suffered 47 attacks of myocardial infarction. Shoulder-hand syndrome followed in 43 instances. In all, the diagnosis of myocardial infarction was made on the basis of a typical history and clinical course. In 33, the diagnosis was unequivocally confirmed by...
electrocardiogram; the infarction was anteroseptal in 1, anterior in 16, and posterior in 16. In the remaining 14 instances the electrocardiograms were invariably abnormal, but showed only persistent T-wave changes, which some physicians would interpret as indicative of ischemia without infarction. One patient had 2 attacks, both involving the anterior wall of the left ventricle. Another suffered an anterior infarction and, 6 months later, a posterior infarction. One case had 3 attacks, the first involving the anterior wall of the left ventricle, the second a posterior infarction 3 years later, and the third, 1 year afterward, a recurrent posterior infarction. Another patient had first a posterior infarction, then, 2 years later, an anterior infarction.

Onset. The earliest onset occurred 2 weeks after the attack of myocardial infarction. The majority took place within 4 months. Two occurred 5 months after the attack; 4, 6 months afterward; and 1 was delayed for 14 months. The time intervals are shown in figure 1.

Duration. Twenty-nine patients, the majority, obtained complete relief within 8 months after the onset of the shoulder-hand syndrome. Nine other attacks (1 patient recovered from 2) were relieved after 10 to 17 months. For 1, the duration was 22 months, and for 2 others, 2 years. One case having a trigger zone over the upper left chest anteriorly has not obtained relief after 3 years; pressure over his trigger zone produces severe pain radiating to the mid-chest and down the left arm. Another patient still has finger stiffness and pain 27 months after his infarction. Since the onset of his disability 4 weeks after a posterior infarction, he has had several periods of complete relief from the syndrome followed by recurrences in one or the other shoulder or hand. He has also had a Dupuytren-like contracture that has greatly improved but has not disappeared. During this time, although he has shown no clinical or electrocardiographic evidence of a recurrent infarction, he has had varying degrees of congestive heart failure. In figure 2 is illustrated the varying durations of disability for the entire group of patients.

Parts Involved. In this syndrome, one or both shoulders may be involved, one shoulder may improve only to have the other shoulder affected, or the shoulders may be involved alternately with frequent recurrences. The hands may be affected in several ways, the most common being stiffness of the fingers of one or both hands. The stiffness may improve in one hand only to become worse in the other, or both hands may improve and then suffer recurrences. In the most severe cases, a condition simulating Dupuytren's contracture may occur. In only 1 of our present group was there marked erythema of the thenar and hypothenar eminences; but we have observed this condition a number of times in patients outside this group.

Severity. In only 5 of our 42 patients did the disability reach the third stage described by Steinbrocker, and the “claw hand” was not seen in any of them. All the abnormalities of Steinbrocker's stages 1 and 2 were uncommonly seen in any 1 patient. In some instances pain in only one or both shoulder regions, or pain or stiffness of one or both hands, was present, often without any objective signs. In 11 instances, both shoulders and both hands were involved. In 12, one or both hands but neither shoulder, and in 8, one or both shoulders but neither hand was affected. In the remaining 12 instances there were various combinations of shoulder and hand involvement. In 1 of the 5 patients having Dupuytren-like contracture, the contracture had been present before the coronary occlusion and persisted after it. In 3 of the remaining 4 patients, the contracture decreased until cure seemed almost complete; in the other, the contracture persisted, though somewhat decreased, for 27 months. One patient had a condition resembling Dupuytren's contracture after the first myocardial infarction. After 2 years the contracture was considerably improved; but 17 months later, when she suffered another infarction, the contracture recurred but improved after 1 year.
CORRELATION OF SYNDROME WITH LOCATION AND SEVERITY OF MYOCARDIAL INFARCTION

We failed to detect any relationship between the transmission of the cardiac pain to the shoulders during the acute attack and the development or location of pain of the shoulder-hand syndrome. Shoulder-hand syndrome occurs in a small percentage (probably not more than 10 to 15 per cent) of cases of myocardial infarction, but the occurrence of the syndrome does not appear to follow recurrent coronary occlusion in any set pattern. For example, one patient remained free of symptoms suggesting the shoulder-hand syndrome after her first coronary attack in 1948; but following her second attack in 1951 she suffered a rather severe shoulder-hand syndrome, only to be free of any such symptoms after her third coronary attack in 1952. Another had an anterior infarction in 1952 and 4 weeks later marked finger stiffness ensued; but following a posterior infarction in 1953 she had neither shoulder pain nor any exacerbation of finger stiffness. One patient suffered stiffness of the fingers and Dupuytren-like contracture after each of her 2 coronary attacks, but her shoulders were never affected. Furthermore, in none of our 42 cases could the severity of the shoulder-hand syndrome be directly related to the severity, the extent, or the location of the myocardial injury. Some of our worst and most persistent cases followed a mild coronary attack in which the electrocardiogram showed no Q waves but merely T-wave changes in the precordial leads that disappeared long before the shoulder-hand syndrome cleared.

CAUSE OF SYNDROME FOLLOWING MYOCARDIAL INFARCTION

The cause of this syndrome is not definitely known. In 1936 Erstein and Kinell suggested that the symptoms of shoulder-hand pain following myocardial infarction may develop merely as the result of relative disuse of the shoulder and abnormal tension of the muscles of the shoulder girdle. It is recognized that the pain, the hand contracture, and in general the late symptomatology may be increased by disuse of the shoulder from immobilization in bed, or by the patient’s fear that the shoulder pain may mean further myocardial damage. Nevertheless, in this author’s opinion, the syndrome appears to be founded upon a complex neurogenic basis.

DISCUSSION

Analysis of our data indicates that the prognosis in shoulder-hand syndrome following coronary occlusion is fairly good, with or without treatment, and that the syndrome is usually self-limited.

In recent years a number of reports have appeared in the literature concerning the efficacy of therapy, in particular of cortisone, sympathectomy, and sympathetic block; the results in general were considered good. We have used cortisone in many patients other than those included in the present study, and in a number of instances the symptoms were relieved; however, in our opinion cortisone did not shorten the course of the disability, and recurrences were quite frequent. Moreover, some cases of shoulder-hand syndrome are not relieved by cortisone, sympathetic block, or sympathectomy. Cortisone may be inadvisable where there are signs of congestive heart failure, and sympathectomy may properly be considered too hazardous in many cases. However,
the material in this study is offered because of its possible value in estimating the long-term prognosis.

None of the 42 patients (43 attacks of the syndrome) received any treatment except heat, analgesics, and exercise of the affected extremity, but only 2 failed eventually to be completely or almost completely relieved (fig. 2). One of these cases who had obtained no relief after 27 months, had both shoulders and both hands involved, the pain seeming to alternate from shoulder to shoulder and hand to hand. Hospitalization was refused and, because of symptoms of congestive failure, including nocturnal dyspnea, cortisone was not given. The other unrelieved patient, having suffered a posterior infarction 5 years previously, had a trigger zone remaining in the upper left chest anteriorly; pressure upon it produced pain that radiated to the sternum and down the left arm. In our experience the prognosis is poor for patients who have a trigger zone, although Steinbrocker suggested that local injection of procaine into the sensitive area may be of benefit in some cases. Berger reported favorable results from the injection of hydrocortisone. In 1936 we mentioned 2 patients with trigger zones in both of whom the results were poor, the syndrome persisting in 1 patient until his death 20 years after the original posterior infarction.

Before cortisone was available we observed a great many patients (none of them included in this series) who recovered from the shoulder-hand syndrome following myocardial infarction after times varying from a few months to several years. It has been our practice to induce these patients to exercise their painful shoulders by "climbing doors" with their arms and increasing abduction gradually; those who have stiff painful fingers are directed to squeeze a rubber ball under hot water. We have not observed that the original pain of the shoulder-hand syndrome is associated with stiffness of the shoulder joint. In some patients the onset of the syndrome is so gradual that they neglect to mention the pain but unconsciously immobilize the joint; others, assuming the pain is of coronary origin, fear to move the painful shoulder. Thus we have made it our regular practice to instruct all patients with coronary occlusion to move their arms and shoulders while in bed and to rest their heads on their hands for 15 to 20 minutes 3 or 4 times daily. Since the establishment of this regimen we have seen the severe types of shoulder-hand syndrome infrequently and the "claw hand" not at all. Of course care should be taken to exclude those patients who have developed a shoulder-hand syndrome following a cerebral accident where paralysis of the extremity may exist.

SUMMARY

Complete or almost complete relief was obtained in 41 out of 43 attacks (95 per cent) of the shoulder-hand syndrome following coronary occlusion in 42 patients. These 42 patients received no treatment other than local heat, analgesics, and exercise of the shoulder and hand. The onset of the disability occurred from 4 weeks to 14 months (the majority within 4 months) after the coronary attack. Relief was obtained in 1 to 24 months (majority within 8 months) after onset. One or both shoulders may be involved and, in most cases, one or both hands also. In some only the shoulders, in others only the hands, were affected.

No direct relationship could be established between the location, severity, or extent of the myocardial injury and the onset, duration, and severity of the shoulder-hand disability. Some of the most obstinate disabilities occurred in patients whose myocardial lesion was considered minor. The involved shoulder may not be the one to which the cardiac pain was originally referred.

Immobilization of shoulder and fingers after onset of the syndrome (usually because the patient fearfully assumes it indicates more coronary damage) seems to increase the severity and duration of the disability, whereas movement in spite of the pain seems to decrease both.

Although some cases had severe pain and swelling of the hands, no instance of "claw hand" was observed in the 42 patients studied, or in the many other cases that have come under the author's observation.

Cortisone, sympathetic block, and sympathectomy have all been reported to be of value
in the treatment of the shoulder-hand syndrome following myocardial infarction and other causes. The present study, however, where the treatment consisted only of local heat, analgesics, and exercise, indicates that the shoulder-hand syndrome following coronary occlusion is a self-limited disability for which the prognosis is, in general, good. The regimen of exercise of the shoulders and fingers soon after the coronary attack and during the early stages of the syndrome increases the favorableness of prognosis.

**SUMMARIO IN INTERLINGUA**

Complete o quasi complete alleviamento esseva effectuate in 41 ex 43 attaccos (95 pro cento) de syndrome humero-manual post occlusion coronari in 42 patientes. Iste 42 patientes recipeva nulle tractamento excepte application local de calor, analgesicos, e exercito del humero e del mano. Le declaration del invaliditate occurreva in 4 septimanas e 14 menses (in le majoritate del casos intra 4 menses) post le attacco coronari. Le alleviamento esseva effectuate intra periodos de inter 1 e 24 menses (in le majoritate del casos intra 8 menses) post le declaration. Un sol o ambe humeros pote esser afficite; in le majoritate del casos etiam un o ambe manos. In alicun casos solmente le humeros es afficite; in alicun alteres, solmente le manos.

Nulle relation directe poteva esser establite inter loco, severitate, o magnitude del lesion myocardial e declaration, duration, e severitate del invaliditate humero-manual. Le plus obstinate invaliditates includeva alicunes in patientes con lesions myocardical que habeva essite considerate como de severitate minor. Le humero afficite non es necessarmente le humero originalmente associate con le dolor cardiac.

Immobilisation de humero e digitos post le declaration del syndrome (usualmente effettuate per le patiente proque ille time que il se tracta de un signo de insulto coronari additional) pare augmentar le severitate e le duration del invaliditate, durante que movimento in despecto del dolor pare reducer ambes.

Ben que certe casos esseva characterisate per sever dolores e tumescencia del manos, nulle caso de "mano-facula" esseva observate inter le 42 patientes studiate o inter le numero altere casos que le autor ha habite le opportunitate de observar.

Cortisona, bloco sympathetic, e sympathectomia ha omnes essite reportate como mesuras de valor in le tractamento de syndrome humero-manual post infarimento myocardial e altere factores causal. Tamen, le presente studio—in que le tractamento consisteva exclusivemente de calor local, analgesicos, e exercitio—indica le syndrome humero-manual post occlusion coronari es un invaliditate auto-restRICTIVE in que le prognose es generalmente bon. Un regime de exercitios del humeros e digitos promptemente post le attacco coronari e durante le prime phases del syndrome mesme augmenta le character favorabile del prognose.

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WHY DO YOU SUPPORT THE AMERICAN HEART ASSOCIATION?

Comments upon Its Origin, Development, and Aims

From Address of Dr. H. M. Marvin before Officers of Affiliates of the American Heart Association, Monday Evening, October 29, 1936

Having been involved in this great movement in a modest way for more than 25 years, it is not strange that I should have known many fine, dedicated people, a number of them sitting in this room, whose lives bear eloquent testimony to the truth of the prophetic words of Mrs. Browning: "The sick man thou hast served will make thee strong; the poor man thou hast served will make thee rich."

I know that many of you have found deep satisfaction in giving yourselves partly or wholly to unselfish efforts on behalf of those who are unfortunate, handicapped and sick. If there is anything more rewarding, except possibly ministering to man's spiritual needs, I have not heard of it. In a world filled with doubt, disappointment and failure, here is something to which sensitive people may turn in the full assurance of receiving great emotional rewards. William James once said, "The great use of a life is to spend it for something that outlasts it." He did not need to add that in spending it thus we can fill it with joy, with dignity, with precious memories; we can justify the qualities that make us human beings; we can in truth, add glory to our dust.

Let me ask, how many things do you know, subject to your control, which lift the heart and give wings to the spirit? How many do you know that deepen the significance of life and widen its horizons? How many do you know that bring joyous and abiding satisfaction? I can tell you one! It is participation in those activities which have as their aim the lessening of human suffering.
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