PERI-ARTHRITIS OF THE SHOULDER

STUDIES OF VEGETATIVE FUNCTION

BY

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Definition.—A diagnosis of peri-arthritis of the shoulder is made when a patient exhibits pain in the region of the shoulder and limited mobility of the joint, apparently of spontaneous onset or following slight injury. The x-ray appearances may show either nothing abnormal or calcific deposits in the capsule or peri-articular tissue.

This syndrome is not a disease *sui generis*, since the underlying pathological changes may no doubt affect either the joint capsule or the peri-articular tissue. Peri-arthritis of the shoulder, therefore, is a collective term for a number of different lesions of the shoulder, the only common features being pain and limitation of movement in the shoulder joint.

A common term may be used for the time being because the clinical features, regardless of the site of the morbid changes, which can hardly be determined without biopsy, are more or less uniform.

Previous Suggestions regarding the Causes of Peri-Arthritis of the Shoulder

Since Duplay (1872) described the syndrome as humeroscapular peri-arthritis, a number of papers have dealt with this lesion. Nevertheless, the actual cause or causes of the disease are obscure, as it is not yet definitely known *why* the morbid changes arise.

The methods used to elucidate these matters comprise (a) direct studies of the shoulder joint and peri-articular tissue at operation, by microscopical examination of biopsy specimens, and (b) clinical studies. In the latter connection it is most interesting to report the papers dealing with the common occurrence of peri-arthritis of the shoulder as a complication of certain other diseases with a constancy which makes it reasonable to presume a pathogenetic relationship.

(a) *Biopsy Studies.*—Neviaser (1945), studying biopsy specimens, found inflammatory changes in the subacromial bursa in seven out of ten cases, tenosynovitis in the long head of the biceps in ten cases, and the capsule adherent to the humeral head in all cases. Microscopical examination of the capsule failed to reveal any definite morbid changes in the synovial cells, but practically all cases exhibited chronic inflammation in the sub-synovial layers, characterized by fibrotic and degenerative processes in the connective tissue and in a few cases by calcification. Culture of the bacteria was negative. Owing to the constant capsular changes, Neviaser interpreted the lesion not as peri-arthritis, but as a thickening and contraction of the capsule which became adherent to the head of the humerus; he therefore suggested the term adhesive capsulitis.

Lippmann (1947), on the contrary, found tenosynovitis of the sheath of the long head of the biceps among twelve operated cases. He did not encounter changes in the sub-acromial bursa or in the peri-articular tissue. Microscopical examination showed inflammatory changes—hyperaemia, oedema, and hyperplasia of the connective tissue with leucocytic and lymphocytic infiltration. In Lippmann's opinion the pain is due to the inflamed tendon sliding freely in the joint proper, and he thinks that fusion of the synovial sheath and the tendon will result in a cure.

Simmonds (1949), working on biopsy specimens derived from operation in four cases, found the initial lesion to be a degenerative process in the supraspinatus tendon and inflammation of the capsule to be a later phenomenon. The cause of this degeneration is unknown, but he found necrotic areas in the tendon surrounded by an inflammatory process.

Though opinions are divided, it seems justifiable to derive from these studies the following conclusion:

The morbid changes in peri-arthritis of the shoulder may affect the joint capsule, or the peri-articular tissue, especially the sub-acromial bursa, the sheath of the long head of the biceps, and the supraspinatus tendon. The changes are nonspecific, suggesting chronic inflammation, and fibrotic and degenerative processes predominate, sometimes with calcification of the degenerated tissue.

Degenerated or necrotic tissue is known to be prone to calcify, and therefore we may assume that there is little fundamental difference between peri-arthritis of the shoulder with and without calcification. This view is held by most workers (Edström, 1936; Kahlmeter, 1936; Dickson and Crosby, 1932), and is supported by the fact that the symptoms often seem to bear little relation to calcifications, which may persist practically unchanged, although the symptoms have subsided. Other workers (Sandström, 1929, 1939; Thyge Madsen, 1948) incline to the view of a nosological entity though without advancing any definite reason.

(b) *Clinical Examination.*—This also fails to give a definite explanation, but some of the studies indicate that vegetative disturbances may be at any rate a predisposing factor and that they are in some cases without a doubt the chief cause of the morbid changes in peri-arthritis of the shoulder. In this connection a syndrome, now usually called the shoulder-hand syndrome, which was described by Kahlmeter (1936), is interesting: it comprised brachial neuralgia, peri-arthritis of the shoulder, vasomotor disturbances in the hand, and a state of fear. Kahlmeter advanced the hypothesis that the cause might be found in a local disturbance of the cervical sympathetic trunk, or of more central vegetative centres, or even possibly in neuritis of the sympathetic nerves. This hypothesis is supported partly by anatomical findings, partly by the effect of sympathectomy. The vasomotor disturbances appear in the form of trophic disturbances manifesting themselves as subcutaneous infiltration of the fingers and hand, the normal creases of the skin being smoothed out. A characteristic sign is also cyanosis interspersed with white patches affecting the hand and fingers, when the arm is hanging down. Often, hypaesthesia and hypalgesia may be found in small areas, and an almost invariable sign is marked limitation of motion in the finger joints and perhaps also in the wrist. Characteristically the elbow joint is hardly ever involved. The mobility in the shoulder joint is almost always very slight, but trophic disturbances like those observed in the hand and fingers are never encountered in the region of the shoulder. The disease is very prolonged and fairly incapacitating. As a rule, blocking of the stellate ganglion is tried, often successfully according to Steinbrocker and others (1948), and with improvement in 60 per cent. according to Jespersen (1949). Jespersen affirms, however, that the effect is equally good, whether or not the blocking results in Horner's syndrome, a finding which does not indicate that surgical intervention is wholly satisfactory.

(c) *Concurrent Disorders.*—Peri-arthritis of the shoulder as a complication of *angina pectoris* seems to have been first reported by Howard (1930), and in Scandinavia has

been dealt with mainly by Ask-Upmark (1944). These cases are often accompanied by trophic disturbances in the homolateral hand and fingers, i.e. it is often a shoulder-hand syndrome. Even in the absence of finger involvement, Steinbrocker interprets peri-arthritis of the shoulder in a patient with *angina pectoris* as an abortive shoulder-hand syndrome, claiming that it is a case of reflex dystrophy confined to the shoulder. The frequent coincidence of the two diseases might indicate that vegetative disturbances may predispose to peri-arthritis of the shoulder, since attacks of *angina pectoris* are elicited by vegetative impulses (vascular spasms). It is worth noting that the pains often radiate into the arms, particularly the left, and that patients with *angina pectoris* often suffer from sensations of fear during an attack.

The idea of a vegetative genesis is also supported by the fact that peri-arthritis of the shoulder is common in the presence of *Graves's disease*. Among 298 patients with hyperthyroidism, Duncan (1932) found 29 per cent. with articular symptoms. Iversen, Sindbjerg-Hansen, and Snorrason (1946) found peri-arthritis of the shoulder to be about 7 or 8 times more common among patients with Graves's disease than among normal persons. Snorrason and Duncan have suggested that hyperthyroidism might also be caused by disturbances in the vegetative central nervous system.

Cases of *epilepsy* treated with phenobarbitone sometimes develop peri-arthritis of the shoulder; this phenomenon, first described by Maillard and Thomazi, was later confirmed in Denmark by Lund (1943), who stated that besides peri-arthritis, such epileptics are also apt to develop Dupuytren's contracture, hilodermia, plastic induration of the penis, and fibroma of the soles of the feet. Since all these abnormalities are due to an increased tendency to form connective tissue, Lund is of the opinion that the pathological cause of peri-arthritis is a fibrotic process and calls all four conditions fibroblastic lesions. Fibrotic processes are almost invariably found in biopsy examination.

Peri-arthritis of the shoulder is also a common finding in cases of *psychosis* treated with phenobarbitone (Bandorf-Kullman, 1939), but it is a common complication in psychiatric cases, even without phenobarbitone medication, and it is not yet agreed whether the trophic disturbances are elicited by phenobarbitone, by the mental disorder, or by a combination of the two. Lund thinks it may be assumed that central vegetative disturbances play some part in its development.

Patients with *cerebral haemorrhage* occasionally develop a shoulder-hand syndrome which must presumably be interpreted as a result of damage to the central vegetative system. In five out of 42 patients with shoulder-hand syndrome examined by Steinbrocker and others (1948), the condition had developed after cerebral haemorrhage.

Among other complications, the patients sometimes also suffer from mild *anaemia*. In a series of 42 patients examined by Steffensen (1945), nine were anaemic, and four suffered from a co-existent *diabetes mellitus*; this series was selective, however, in that it comprised only hospitalized patients.

Pulmonary disorders have also been mentioned as frequently concurrent with peri-arthritis of the shoulder, and prolonged *rest in bed* is also said to be a predisposing factor. These latter phenomena, however, have not been submitted to further study.

Present Investigations

The present study was undertaken to find out whether tests of vegetative function (measurement of skin temperature) might be able to demonstrate vegetative disturbances in peri-arthritis of the shoulder, even in cases where no trophic disturbances were demonstrable. Since manifest vegetative disturbances occur in the presence of peri-arthritis of the shoulder (shoulder-hand syndrome), it is reasonable to try to find out whether abnormal vegetative impulses are demonstrable in the fingers even in the absence of visible vegetative disturbances. Such

studies are also thought to be justified by previous workers' findings regarding the co-existence of peri-arthritis of the shoulder with angina pectoris, Graves's disease, epilepsy, and cerebral haemorrhage.

Methods of Measuring Skin Temperature.—Measurement of the skin temperature as a means of studying the vasomotor reactions has been undertaken in various ways:

In Denmark Ipsen described a procedure which is usually called the cold test:

For 10 minutes symmetrical parts of the extremities are immersed in water at 15° C. to produce arteriospasm. After the extremities have been removed from the water, the spasms will be relieved and the temperature will rise. By comparing the symmetrical sites, it is possible to demonstrate unilateral disturbances. This method, however, is rather inaccurate, since, amongst other reasons, the water remains on the limbs in small drops which evaporate and thereby cool the site after it has been removed from the water. This evaporation may easily vary on the two sides.

Gibbon and Landis recommended the following procedure:

The patient is left in a cool room with the limbs to be examined exposed, so that the skin temperature falls evenly to approximately the room temperature. If the arms are to be studied, the lower limbs are then placed in a foot bath of 44° C. This results in an increase in the skin temperature of the arms after a certain latent period. In normal persons the increase takes place practically at the same time and to the same level on both sides (according to Gibbon and Landis to at least 31·5° in 30 min.).

After using this method in a few cases I abandoned it, because the decrease of temperature was far too slow, and was not marked enough, possibly because the room was not cold enough. Another disadvantage is that air currents may act upon the temperature at the sites to be examined and so cause inaccuracy.

I therefore adopted the method advocated by Christiansen, Fog, and Vanggaard (1939), which was later used for physiological studies by Vanggaard (1941), and formed the basis of the investigations of Haldbo (1942) into post-traumatic reflex dystrophy. The method is as follows:

The arms are placed in a cold box where they remain throughout the experiment. When the skin temperature has decreased sufficiently (12-16° C.), vascular dilatation is induced by placing the feet into hot water (42-44° C.), which makes the skin temperature rise abruptly to a maximum after a certain latent period. The footbath is removed, and another gradual decrease in temperature occurs.

The cold box offers the advantages that air currents are practically excluded, evaporation from the skin is slight, and the same exogenous action is obtained on both sides; thus changes in skin temperature depend solely on variations in the blood flow through the skin. If the blood vessels are normal, the skin temperature must depend on the vegetative innervation. Comparison of the two sides, therefore, will demonstrate unilateral vegetative disturbances.

Haldbo used two boxes, the sides of which were filled with a mixture of ice and water, I used only one box in an endeavour to expose the arms to the same exogenous action.

The walls of the box consist of two layers of plywood separated by an insulating layer of glass wool. A quarter of an hour before the experiment is started, a zinc tray with ice is placed in the ceiling of the box; this tray is covered with a plate of zinc and provided

with a tightly-fitting lid of the same material as the floor and walls. The arms are inserted through two holes at one end of the box, and the holes are provided with felt cuffs which are tied—without pressure—round the patient's arms, so that the insulation remains effective. So that the measurements may be made under visual control, the other end of the box is fitted with double glass panes provided with two small holes, spaced about one shoulder-breadth apart, for insertion of the measuring instrument. These holes, which are plugged with cotton wool when not in use, are placed about 15 cm. above the fingers, so that air currents during the measurement may be presumed to be negligible. I used a thermo-element mounted on a handle and connected with a potentiometer showing the temperature directly. All the measurements were made in symmetrical sites on the right and left, on the dorsal aspect of the middle finger in the most distal extensor crease.

Because of technical and other difficulties no measurements were made on the shoulders. It is impossible to exert the same pressure at each measurement with thermo-elements which are not fixed; in the region of the shoulder the ample subcutaneous tissue exerts an effect, but this is of negligible importance in the fingers. It is impracticable to cool the shoulders in a cold box. The local inflammatory changes and consequent hyperaemia may be expected to give rise to vegetative disturbances as a purely secondary phenomenon, so that any findings on the shoulders are of only minor interest.

Material.—Tests of vegetative function were carried out on a total of thirty patients. The patients were selected, since it was not considered justifiable to let persons over a certain age sit with their arms in the cold during the 3 or 4 hours of the experiment. In addition, I preferred females to males, because in my experience females have decidedly more patience for such prolonged examinations. The age and sex of the thirty patients are shown in the following Table.

AGE AND SEX DISTRIBUTION OF PATIENTS

Sex		Age Group			
♀	♂	30-40	40-50	50-60	60-70
26	4	1	4	17	8

Of the thirty patients, one had a unilateral and two a bilateral shoulder-hand syndrome, but the remaining 27 did not exhibit trophic disturbances. At the time of the examination the two patients with bilateral shoulder-hand syndrome were in-patients in the neurological department, and all the rest were attending the department of physical therapy as out-patients.

Procedure.—It is important for the arms to have been exposed to a uniform exogenous action for some time before the experiment begins. This is of particular importance in the case of ambulant patients. Like Haldbo, therefore, I had to let the patients sit with the arms uncovered in the experimental room before the experiment was started.

During the experiment the skin temperature was measured at 5-min. intervals while it was decreasing, and at 2½-min. intervals while it was increasing in response to indirect heating.

The results of these studies have been plotted as curves (Figs 1-10), the abscissa showing the time in minutes and the ordinate the temperature in °C. The thick line parallel to the abscissa gives the duration of the footbath.

Results

A. Cases without Trophic Disturbances (27)

In 21 out of these 27 patients the skin temperature fell almost equally, and the increase in temperature occurred almost simultaneously, reaching the same level on both sides at the same time. The latent period from the time when the feet were immersed in water until the temperature began to increase varied widely—from 15 to 90 min. According to Vanggaard (1941), however, this latent period is subject to physiological variations, so that it does not allow of any conclusions regarding abnormal conditions. An example of one of these 21 curves is given in Fig. 1.

Among the 21 patients with little or no difference between the two sides, are two whose curves deserve consideration (Figs 2 and 3). These showed agreement

FIG. 1.—Typical parallel curves.

FIG. 2.—Peak induced by menopausal flush.

FIG. 3.—Peak induced by menopausal flush.

between the skin temperature on both sides, but after the temperature had fallen to about $20^{\circ}\text{C}.$, there was a sudden "peak" representing an increase of about $5^{\circ}\text{C}.$ In both cases about 30 min. elapsed before the temperature fell to the level at which the increase set in. It turned out that both patients had had menopausal hot flushes at the time when the temperature rose. These flushes resulted in marked vasodilatation on the fingers which lasted for half an hour. No further mention will be made of vegetative disturbances during the menopause.

The curves representing the other six patients, who showed more marked differences between the two sides, are given in Figs 4 to 9: of this group five (Figs 4 to 8) were much alike; the skin temperature on the affected side fell more slowly and not so far as on the healthy side. During the decrease, the difference amounted to 8 or $9^{\circ}\text{C}.$, and in no case was the maximum difference less than $7^{\circ}\text{C}.$

During the decrease in temperature a few "peaks" also occurred on the curve representing the affected side (most marked in Figs 4 and 5), indicating

FIGS 4 and 5.—Higher temperature on *affected* side with slight flushes.

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FIGS 6, 7, 8.—
Higher temperature on
affected side.

small unilateral "flushes" on the affected side. These "peaks" are of almost the same height and duration as those occurring with menopaual flushes.

In a few cases the increase in temperature occurred considerably earlier on the affected than on the healthy side (Figs 6, 7, 8). The most marked time difference will be seen in Fig. 6, in which the increase occurred $7\frac{1}{2}$ minutes later on the healthy than on the affected side.

As a rule the maximum increase in temperature reached approximately the same level on both sides. In Fig. 5,

however, it may be seen that the affected side reached a level 3° higher than the healthy side.

Fig. 9 shows an equal decrease in temperature and a simultaneous increase, during which, however, there was a rather marked difference between the two sides, the affected side being lower than the healthy one.

FIG. 9.—Higher temperature on *unaffected* side.

We may now ask the question: Are these curves expressions of unilateral vegetative disturbance or merely of a variation within physiological limits?

Of course, it would have been best to have studied an equally large number of normal patients. I did not, however, consider this necessary, feeling justified in using Vanggaard's experimental results from a normal series as a basis for comparison, since his method is in principle the same as the present one.

Vanggaard's cold box was designed somewhat differently from mine. This is not important, if only the cold box fulfils the necessary conditions. Irradiation of heat from the warmer arm to the colder one cannot take place, since measurements have shown the air between the arms to be of the same temperature as that in the remaining parts of the box.

Vanggaard used fixed thermo-elements, but I used a mobile one; in my experience this does not cause any error worth mentioning in measurements on the fingers where the subcutaneous tissue is so scanty that a major or minor pressure during the measurements gives the same result.

Vanggaard's normal series comprised thirty subjects. In 26 of these he found the curves to be closely symmetrical with regard to the rhythm of their movements, and the absolute values of skin temperature to be close to each other. In the other four instances, however, he found that after the heating was stopped the skin temperature fell much more quickly on one side. Before the heating, on the other hand, the decrease in temperature did not show divergences of more than 2 or 3°.

In other words, it is reasonable to presume that marked differences in temperature during the first falling part of the curve may be interpreted as an expression of abnormal vegetative impulses on one side. Of course, it is not possible to tell from the curves alone which side is affected, but one may assume that the curve from the side affected by peri-arthritis stands for the abnormal findings.

Thus, the decrease in temperature after discontinuation of heating may present rather marked physiological variations on the two sides. Therefore, and also because at this stage the experiment had often already lasted for about 3 or 4 hours, I did not trace the decrease in temperature for a long time in this phase.

According to Vanggaard, the time at which the temperature begins to increase on the two sides is also subject to physiological variations, and may be up to 8 min. later on one side than the other. This occurred in only one of my cases, and in the others the interval amounted to only a few minutes.

On the basis of these observations, I consider Curves 4 to 8 to be abnormal. Curve 9 is probably not abnormal, since the decrease in temperature was equal. The difference between the two sides was not seen until the increase began and the temperature then rose further on the healthy side.

This prompts the question whether the case histories of the five patients represented in Curves 4 to 8 differ from those of the other patients without trophic disturbances. To elucidate this question the case histories of the five patients with abnormal curves are given in some detail, and the findings in the remaining cases are summarized very briefly.

Case Reports

Curve 4, female, aged 56. She had previously been on the whole healthy. Menopause occurred 7 years ago, but she had still hot flushes. She had been treated for a long time with phenobarbitone and stilboestrol, when the shoulder lesion set in.

Present Illness.—Gradually increasing pain in right back of the head, shoulder, and arm; paraesthesia in the thumb and two radial fingers, no temperature sensations, no stiffness of the fingers, no injury. Marked limitation of motion. Duration of disease 8 months, skin temperature measured 5 months after onset. X ray of right shoulder showed nothing abnormal. Wassermann reaction negative, gonococcal complement-fixation test negative, antistreptolysin titre 22, Hb 108 per cent., erythrocyte sedimentation rate 22—30—28—26. She had received physical therapy for 2½ months with little effect, and x-ray therapy for 3 months, as a result of which she had been relieved of pain.

Curve 5, female, aged 59. Five years previously she had had right-sided peri-arthritis of the shoulder which yielded in the course of 3 months to x-ray therapy, and had otherwise been healthy on the whole. Menopause occurred 11 years previously; at the outset she had had hot flushes, but not during recent years.

Present Illness.—Gradually increasing severe pain in the left shoulder and arm, no paraesthesia, no temperature sensations, no stiffness of the fingers, no injury, no phenobarbitone. Severe limitation of motion. Duration of disease 6 months, skin temperature measured 2 months after onset. X ray of left shoulder showed nothing abnormal. Wassermann reaction negative, gonococcal complement-fixation test negative, antistreptolysin titre 64, Hb 89 per cent., erythrocyte sedimentation rate 4—20—22—6. She had received physical therapy for 6 weeks with little effect, and x-ray therapy for 3 months, as a result of which she had been relieved of pain.

Curve 6, female, aged 48. She had previously been healthy, but had always been "nervy" and moody. Menstruation regular, no hot flushes.

Present Illness.—Acute onset of pain in right shoulder and arm, paraesthesia in all fingers and thumb, no stiffness of the fingers, no injury, had received phenobarbitone.

Marked limitation, particularly of active motion. Appeared to be exaggerating her symptoms. Duration of disease 3 months, skin temperature measured 8 days after onset, when she had had a sensation of heat in the right upper arm for 2 days; this subsided 4 or 5 days after the experiment. X rays of right shoulder showed nothing abnormal, of cervical column showed spondylitis deformans of C5, C6, and C7. Wassermann reaction negative, gonococcal complement-fixation test negative, Hb 90 per cent., erythrocyte sedimentation rate 7. She had received physical therapy for 3 months, and as a result was almost free from pain.

Curve 7, female, aged 67. Long-standing asthma for which she had periodically taken ephedrine. Menopause occurred 20 years ago, with hot flushes at the outset, but none at present.

Present Illness.—Gradually increasing, but not particularly severe, pain in left shoulder and arm, paraesthesia, stiffness, and slight swelling of the fingers of the left hand, particularly in the morning, no temperature sensations, no injury, no phenobarbitone. Moderate limitation of motion, left-sided Dupuytren's contracture, no hiloderma, no trophic skin disturbances, especially nothing suggesting reflex dystrophy. Duration of disease 6 months, skin temperature measured 5 months after onset. X rays of shoulders and hands showed nothing abnormal, particularly no halisteresis, lungs showed sequelae of right-sided interlobar pleurisy. Electrocardiogram showed nothing abnormal. Wassermann reaction negative, gonococcal complement-fixation test negative, anti-streptolysin titre 80, Hb 90 per cent., erythrocyte sedimentation rate 23—40. She had received physical therapy for one month, and as a result was almost relieved of pain. Dupuytren unchanged.

Curve 8, female, aged 62. Had always been somewhat nervous, but otherwise healthy. Menopause occurred 15 years previously, and she still had periodical hot flushes.

Present Illness.—Gradual onset with a sensation of chill at the back of the head, increasing pain in the right shoulder and arm and two ulnar fingers, no paraesthesia, no stiffness of the fingers, no injury, had received phenobarbitone. Slight limitation of motion; movements of cervical column somewhat restricted. Duration of disease 7 months, skin temperature measured 2 months after onset. X rays of right shoulder showed nothing abnormal. X rays of cervical column showed spondylitis deformans. Wassermann reaction negative, gonococcal complement-fixation test negative, anti-streptolysin titre 40, Hb 95 per cent., erythrocyte sedimentation rate 12—15. She had received physical therapy for 3 months with little effect, and then x-ray therapy for 3 months with good effect, but still suffered from mild pains.

Observations on Five Abnormal Cases

- (1) Only two patients had temperature sensations, one at the back of the head, and the other in the upper arm, but no such sensations had occurred in the hands. This finding, however, does not rule out the occurrence of abnormal vegetative impulses at this site, as I have a few times recorded temperature differences of 7-8° without the patients having noticed a difference on the two sides. As a rule the patients themselves notice the difference, but at other times patients may report a difference, although the measurements show exactly the same temperature, so that subjective statements are of doubtful value.
- (2) All the patients had radiating pain and three had paraesthesia in the fingers.
- (3) One patient exhibited Dupuytren's contracture, but no other trophic disturbances.
- (4) In three cases the lesion was right-sided and in two left-sided, but the course of the curves was found to be independent of the side affected.

- (5) The duration of the disease had been fairly long in all cases and had proved rather refractory to treatment which in three cases had had to be supplemented by x-radiation.
- (6) Two patients exhibited spondylitis deformans of the cervical vertebrae. It must be stated, however, that only those patients who showed restricted mobility of the cervical column were x-rayed from this point of view. Opinions are divided as to the significance of this finding; among fourteen patients with rheumatic pain in the region of the shoulder and trophic disturbances of the hands, Oppenheimer found spondylitis deformans of the cervical vertebrae in each case, whereas this phenomenon was observed only in exceptional cases by Steinbrocker who does not attribute much importance to it.
- (7) The sedimentation rate was slightly enhanced in most cases.
- (8) Two patients suffered from menopausal hot flushes.

Observations on 21 Normal Cases

Symptoms.—Nervous, five; menopausal hot flushes, three; gradual onset, twelve; acute onset, six traumatic and three non-traumatic; temperature sensations, one patient ("chill" at the back of the head); stiffness of fingers, one; paraesthesia, eight; radiation of pain, seventeen; spondylitis of cervical vertebrae, three; peri-articular calcifications, three; raised erythrocyte sedimentation rate, six.

Duration of Disease.—This ranged from 1 month to several years, the patients being fairly equally distributed between these two extremes. The time at which the temperature was measured varied also, with the extremes at a fortnight and several years after the onset. In the majority of cases, however, the measurement took place after the disease had lasted for 3 to 5 months and usually 2 months before treatment was discontinued.

Therapy.—Two had previously received x-ray therapy, both with no improvement worth mentioning. Three were referred for x-radiation because physical therapy had failed.

B. Cases with Trophic Disturbances (3)

Two cases of bilateral shoulder-hand syndrome were included to find if there was any difference of degree between the two sides. In both the curves fell and rose again equally.

The third case, one of typical right-sided shoulder-hand syndrome, had arisen after a "wrong" movement performed by the patient in picking something up

FIG. 10.—Curves characteristic of healing phase.

from the floor. At the time of examination the disease had persisted for 6 months; the patient was improving on ordinary physical therapy, and one month after examination the pain had virtually disappeared. In other words, one would expect the curve to correspond to the findings during the healing phase, and this it did (see Fig. 10). The difference in temperature between the two sides was not particularly marked, but the increase on the affected side occurred a few minutes later, a sign of

vasospasms. While the disease was in the active stage, the patient had complained of a sensation of heat in the hand, but she was not examined during this phase, as I did not then possess the necessary apparatus.

Discussion

It is evident from these data that striking, let alone constant, differences were not observed between patients with normal curves and patients exhibiting differences in temperature on the two sides during cooling.

To evaluate the findings of the vegetative studies in patients without trophic disturbances, it would be reasonable to make a comparison with findings in patients with trophic disturbances of the fingers. As my own series is rather inadequate in this respect, I shall also report the results of others.

As emphasized by Steinbrocker and others (1948), the shoulder-hand syndrome belongs to the group of diseases ordinarily named "reflex dystrophies", a term which refers in the main to the characteristic vasomotor and trophic disturbances accompanying these conditions. The term "shoulder-hand syndrome" refers merely to the peculiar localization of the changes.

It is generally agreed that reflex dystrophies run through certain phases with varying vegetative disturbances. According to Steinbrocker and others (1948), the shoulder-hand syndrome has three different phases, corresponding in principle to the phases of reflex dystrophy, regardless of site.

Haldbo (1942) surveyed the vegetative disturbances in two phases, elucidated by tests of vegetative function. Since his procedure was the same as mine, it seems reasonable to use his experimental results as a basis for comparison. In the first or developmental stage—characterized by severe oedema, pain, and restriction of movement—there are always disturbances referable to paralysis of the sympathetic nerves, i.e. hyperaemia and anhidrosis. Correspondingly, Haldbo observed slower and less complete cooling of the affected than of the unaffected side. In the second or healing stage—characterized by tissue atrophy—arteriospasm manifest themselves in a greater decrease and a slower increase of skin temperature on the affected side. The affected side also exhibits hyperhidrosis. Both findings indicate increased sympathicotonia.

I can see no essential difference between Steinbrocker's Phases 2 and 3, which are both stages of healing, and his description of the shoulder-hand syndrome may thus be said to correspond to Haldbo's observations on reflex dystrophies in general, especially since several of Haldbo's cases belong, in fact, to the shoulder-hand syndrome group.

The findings in the present study correspond in principle to the experimental results found during the development of reflex dystrophy. Not one of my five abnormal cases, however, showed trophic disturbances at any time, so that the condition is probably interpretable as an abortive shoulder-hand syndrome, which infers the presence of abnormal vegetative impulses in the fingers of the affected side without manifest trophic disturbances.

Arteriospasm did not occur on the affected side. This is due either to chance,

or to the fact that any arteriospasm were so slight as to be undemonstrable by the method used. A third possibility—and probably the most likely one—is that vasospasms do not occur until the time when the patients are recovering. In this respect I have made the regrettable error of not following-up the patients by repeated measurements of skin temperature.

Abnormal vegetative impulses without trophic disturbances occurring after injury were described by Leriche (1923), who found by oscillometric experiments that any injury to a limb, open or closed, is followed by vasomotor disturbances; at the outset these take the form of vasoconstriction, but this usually changes to dilatation which may persist for a few months without clinical manifestations. These phenomena do not appear to have been studied previously in ordinary peri-arthritis of the shoulder without trophic disturbances.

The pathogenesis of reflex dystrophies, and thus also of the shoulder-hand syndrome, is obscure. Ordinarily Steinbrocker's explanation is accepted:

Impulses causing the trophic and vasomotor disturbances issue from a focus of irritation through an axone reflex or by antidromal stimulation of sensory autonomic nerves. The focus of irritation may be an area of damaged tissue (trauma), the heart (angina pectoris), or the central vegetative centres (cerebral haemorrhage). A reflex genesis is considered most likely because interruption of the efferent pathway in the reflex arc (blocking of the sympathetic ganglia) seems to improve the condition.

In the present writer's opinion, the following objections may be raised:

(1) Sympathetic blocking produces no convincing effect in these conditions. If the blocking did affect an essential factor of the disease, the therapeutic result would be far more striking. Jespersen's results have been reported above. As to Steinbrocker's results, the blocking failed in some cases, and in most instances the changes persisted for years regardless of blocking or sympathectomy. The idea of blocking the sympathetic ganglia in these conditions also appears paradoxical, since vasomotor tests have shown that the developmental stage is marked by reduced sympathetic function, manifesting itself as an abnormally increased hyperaemia, which would only be further increased by sympathetic blocking. This is pointed out by Steinbrocker as well as by Haldbo, but both workers find it important first to interrupt the presumed reflex mechanism.

(2) If the idea of a reflex be disregarded, it would appear more logical to try the opposite procedure, viz. to reproduce the vasospasms encountered in the healing phase. This has seemingly not been tried, presumably because of the great practical difficulties. A possible procedure might be iontophoresis of an adrenergic substance. A reflex mechanism is almost certainly indispensable to start the process, but at a later stage the essential factor is probably damage to the vegetative nerve centres. Such damage was found by Sunder-Plassmann in the form of degenerative processes in the ganglion cells of sympathetic trunks from patients suffering from these conditions.

Summary

In five out of 27 patients with peri-arthritis of the shoulder, measurements of the skin temperature revealed abnormal vegetative impulses in the fingers of the affected side despite the absence of trophic disturbances. This must be interpreted as a condition which might be called an abortive shoulder-hand syndrome, the pathogenesis of which is probably analogous with that of a manifest shoulder-hand syndrome, i.e. a result of vegetative disturbance.

The experimental results support the idea that peri-arthritis of the shoulder may, in some cases at least, result from disturbances in the vegetative nervous system.

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Périarthrite scapulaire—Étude de la fonction végétative

RÉSUMÉ

Chez cinq sur 27 malades atteints de périarthrite scapulaire la mesure de la température cutanée montra l'existence des impulsions végétatives anormales dans les doigts du coté atteint, malgré l'absence de troubles trophiques. Ceci doit être interprété comme indiquant une affection qu'on pourrait appeler "syndrome scapulo-manual abortif", et sa pathogénie est probablement analogue à celle du syndrome scapulo-manual manifeste, c'est à dire une conséquence d'un trouble végétatif.

Les résultats expérimentaux viennent à l'appui des théories selon lesquelles la périarthrite scapulaire pourrait, dans certains cas tout au moins, dériver des troubles du système nerveux végétatif.

Periarthritis escapular—Estudio de la función vegetativa

SUMARIO

En cinco de los 27 enfermos con periartritis escapular la determinación de la temperatura cutánea mostró la existencia de impulsos vegetativos anormales en los dedos del lado afectado, a pesar de la ausencia de disturbios tróficos. Al interpretar este hecho se llega a la conclusión de que se trata de una afección que merecería el nombre de "síndrome escapulo-manual abortivo", y su génesis es probablemente análoga a la del síndrome escapulo-manual manifeste, es decir el resultado de un disturbio vegetativo.

Los resultados experimentales sostienen las teorías según las cuales la periartritis escapular puede, en ciertos casos por lo menos, resultar de los disturbios del sistema nervioso vegetativo.