ANESTHESIOLOGY

The Journal of

THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

Volume 16 MAY, 1955 Number 3

THE RELATION OF SUBCUTANEOUS FOCAL SENSITIVITY TO REFERRED PAIN OF CARDIAC ORIGIN *

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THE nature of the referred pain which occurs in the arm and shoulder as the result of coronary disease has been the subject of much speculation. Pain, usually in the left shoulder and upper arm, but also often about the right shoulder and the neck, is well recognized as a frequent accompaniment of the precordial distress that results from myocardial insufficiency. The literature on the subject is large and has been reviewed many times (1–5).

The usual explanation for the site of the referred pain is that it is central in origin and induced by spread of stimuli within the cord where impulses arriving from the disordered region by way of visceral afferents excite adjacent somatic pain-transmitting neurones within the synaptic systems of the cord. This theory was first elaborated by McKenzie and by Ross, as was discussed recently by White, Smithwick and Simeone (6). In the present instance, it would be considered that impulses of overwhelming intensity from the heart arrive at a central region and there excite adjacent afferent systems from the somatic shoulder area.

In some cases of chronic coronary insufficiency the referred pain becomes the outstanding symptom (1, 3, 7, 8) and the arm and shoulder become rigid and atrophic in consequence of the pain. Marked relief and return of peripheral function may follow procaine injection of trigger areas about the arm and shoulder which manifest the pain. The mechanism whereby relief of pain is brought about by procaine injection of trigger spots in the leg, back and shoulder has been dis-

* Accepted for publication November 23, 1954.

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cussed by Livingston (9) and by Travell and associates (10, 11), and much of the stimulus for the development of the present project is based on their previous work. The present study arose from observations made during the treatment of a number of cardiac cases when it was noted that the trigger areas, abnormally sensitive to pressure, lay in well defined locations which were the same in all patients and that the pain from these trigger spots always radiated in the same directions.

METHOD

Since we saw no obvious reason for the fixity of the trigger sites and no particular structure was apparent there, it was decided to examine a larger group of cardiac patients in an attempt to detect the significance of these trigger areas and their relationship to other signs and symptoms. Control patients were also examined, and were eventually divided into two groups.

The present paper is based on the examination of 72 patients with cardiac disorders, 35 patients with thoracic disorders not related to the heart and 46 patients with nonthoracic disorders. In the first control group were 24 patients with respiratory disease, 7 with epigastric pain, 3 with cholecystitis and one with thoracic pain of thalamic origin. In the second control group were patients with pelvic disorders, leg varicosities and the like, who had no signs or symptoms relative to thorax or diaphragm. All were patients on the wards of the Multnomah County Hospital in Portland, Oregon.

All patients were seen by both authors working as a team and taking turns on alternate days as either the examiner of the patient or the recorder of observations. The patients were all conscious and cooperative although unaware of the import of the procedure. Examinations were carried out as soon as possible after admission but, since examination of the back as well as the chest was necessary, acutely ill patients were not disturbed until permission was given by the medical staff as recovery progressed. All consecutively admitted patients who could thus be examined were included in the study. All were carefully questioned as to the extent, character and duration of their pain. In this series, although pain radiating to shoulder and arm was a usual symptom, there was no instance of the severe shoulder and arm dystrophy which is sometimes seen.

The search for sensitive areas was made, using firm but gentle pressure of the hand and fingers over the entire chest, back and shoulders. As the examiner detected sensitive spots these were marked on a chart according to whether they were slightly, moderately or markedly sensitive. The type of pain, as described by the patient, and its radiation were also noted. In figure 1 the chart used is shown together with the most frequent sites of trigger sensitivity. Since there was some variation in the degree of sensitivity and since

slightly sensitive areas were frequent in both controls and cardiac patients, these were not considered significant and only moderate or marked sensitivity has been considered as related to the pathological process in the tables to follow.

DATA

Nature of the Subcutaneous Sensitivity. If a "normal" person without signs of cardiac or other disorder presses firmly on certain areas of his chest or back he may find some spots which are more sensitive than are other areas. In these same regions, cardiac pa-

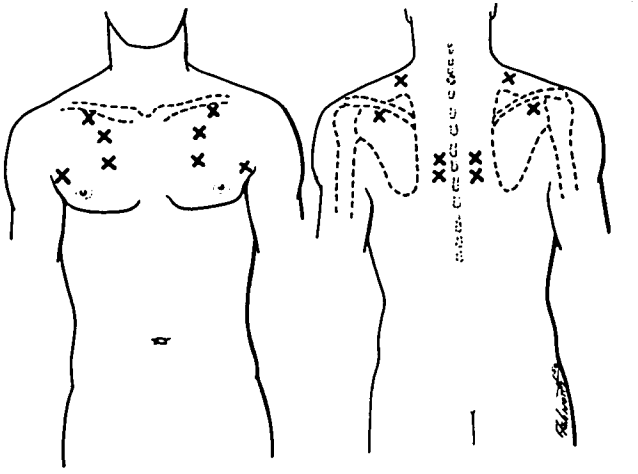

FIG. 1. Diagram of outline used in charting trigger spots, showing areas most frequently sensitive.

tients may be much more sensitive and the pain may be spread over much larger areas so that light pressure alone may cause him to flinch and even light stroking of the skin above the focus may be disagreeable. The character of the pain in both the patients and controls is very definite. On firm pressure it is discrete, stabbing and "hot." After the pressure is removed the pain remains for several minutes in the controls and may even become more painful a few seconds after removal of the stimulus. In the very sensitive cardiac patients this is many times magnified so that their general pain and discomfort is augmented, sometimes for several hours, after the examination. There is always radiation from the trigger spot which

has been stimulated over a fixed geographical region about the area. Hence the name "trigger" spot has its origin.

Both the controls and cardiac patients were completely unaware of these sensitive spots until they were examined. Furthermore, the sensitivity to pressure was not related to their spontaneous chest pain except in a few cases when pressure seemed to produce or augment the spontaneous type of pain being experienced.

Site of Sensitive Spots. The spots which were most frequently sensitive are marked on figure 1. The two most common, which are those most frequently found in a "normal" group also, lie on the chest in the midclavicular line about 1 inch below the clavicle, and on the back of the shoulder at the upper border of the trapezius. Next most frequent were those at the anterior axillary border and those in the second and third intercostal spaces on the chest. Pressure over the flat surface of the scapula also frequently produced pain.

Frequency of Sensitive Spots. Table 1 presents the data with regard to the presence or absence of sensitive spots. A severe degree of sensitivity appeared only in cardiac cases, of which 61 per cent showed sensitivity of either moderate or marked degree. Of the controls who had disease involving thoracic structures, 48 per cent had moderately painful areas, but of the controls who did not have thoracic disease only 20 per cent showed moderate sensitivity. There was thus a significant increase, both in degree of pain and frequency of its appearance in somatic foci, when disease of thoracic viscera, and in particular of the heart, was present.

Lateralization of Sensitive Spots. It is clear from the data presented in table 2 that a positive relationship exists between the side of the spontaneous cardiac pain and the site of trigger sensitivity. Those subjects who had trigger spots about the left shoulder and arm had spontaneous pain on that side, while those having spontaneous pain through to the "middle of the back" had a great number of sensitive spots in the back, and the few subjects who had right-sided pain also had predominantly right-sided sensitivity.

Furthermore, of the 51 cardiac patients who had some degree of sensitivity to pressure in 36 (70 per cent) the sensitivity was either entirely (5 cases) or predominantly (31 cases) on the left side. Two

TABLE 1

TRIGGER AREAS IN CARDIAC AND CONTROL CASES

Group	Total Cases	Trigger Sensitivity				
		None	Sl.	Mod.	Marked	Per Cent Present
Cardiacs	72	11	17	26	18	61
Controls, thoracic	35	8	10	17	0	48
Controls, nonthoracic	46	25	12	9	0	20

TABLE 2

LATERALITY OF PAIN AND TRIGGER SPOTS

Location of Pain	Total Cases	Trigger Sensitivity			
		Bilat.	Rt. Predom.	Lt. Predom.	Per Cent Present
Chest only	24	2	6	10	75
Chest and right arm	6	2	2	1	83
Chest and left arm	22	6	0	12	82
Chest and both arms	13	3	1	9	100

had predominantly right-sided sensitivity. In the controls, however, no such left-sided predominance occurred. Of those 27 who had noncardiac thoracic conditions together with painful trigger spots, 5 were more sensitive on the left and 6 on the right, and of the 21 in the nonthoracic control group, 8 had predominantly left and 3 right-sided sensitivity.

Relation of Sensitivity to Spontaneous Referred Pain. Table 3 shows the relationship of sensitivity to spontaneous pain. The cardiac and control groups have been divided according to whether or not the individuals experienced spontaneous pain with their present illness. Only half of the cardiac patients were experiencing present coronary dysfunction *with pain,* the remainder having usually some degree of cardiac insufficiency. Similarly, 20 of the 35 controls who had other thoracic disorders and 19 of those who had nonthoracic disorders had no spontaneous pain during their hospital admission. It is apparent that both thoracic visceral pathologic change without pain and nonthoracic pain are related in some degree to sensitivity in the thoracic subcutaneous areas; that thoracic pain and trigger zones are markedly related, and that those patients who had no pain and no thoracic disorder are remarkably free from trigger areas.

Relation of Sensitivity to Present Pain. Clinically there seemed to be a relationship between degree of spontaneous pain and degree of

TABLE 3

RELATION OF SENSITIVITY TO SPONTANEOUS PAIN

Group	Trigger Sensitivity				
	None	Sl.	Mod.	Marked	Per Cent Present
Cardiac, pain	6	9	10	11	58
No pain	5	8	16	7	61
Controls, thoracic pain	4	6	10	0	50
No pain	7	5	7	0	41
Controls, nonthoracic pain	4	4	7	0	47
No pain	18	7	2	0	8

TABLE 4

RELATION OF SENSITIVITY TO PRESENT PAIN

Group	Trigger Sensitivity				
	None	Sl.	Mod.	Marked	Per Cent Present
Pain present, cardiacs	0	8	10	9	70
Controls, thoracic	6	6	9	0	43
No pain present, cardiacs	11	9	16	9	56
Controls, thoracic	2	4	8	0	57

sensitivity. Those cardiac patients who had severe coronary pain during their present illness demonstrated the greatest tenderness to pressure. In many instances on repeated examination of the cardiac patients the tenderness became less as the spontaneous pain diminished during convalescence. In table 4 the relationship of present pain to sensitive areas is recorded. Those patients, either controls or cardiac cases, who had had spontaneous pain within twenty-four hours of the examination were separated from those without recent pain. There is a significantly higher incidence of sensitivity in the cases with present pain.

Relation of Trigger Spots to Severity of Illness. After a lapse of three years, it was thought that the interval history of the patients might be of interest. Table 5 shows the results of a follow-up study of the hospital records. Those individuals with cardiac disease who had been followed for periods of from nine months to two years after the conclusion of the study were classified as to whether they were: (1) moderately disabled by their cardiac condition (were still attending cardiac clinic); (2) severely disabled (had been sent to a nursing home or had multiple admissions to the County Hospital), or (3) had died. It will be seen in the table that there is no significant correlation between the severity of the trigger pain and the subsequent course of the cardiac disorder.

TABLE 5

FOLLOW-UP OF CASES DURING TWO YEARS SINCE EXAMINATION

	Total Cases	Trigger Sensitivity				
		None	Sl.	Mod.	Marked	Per Cent Present
Moderate disability	28	4	6	9	9	64
Marked disability	6	0	2	3	2	71
Died	22	5	5*	8*	4*	54
No follow-up	15	2	4	6	3	60

* One death of noncardiac etiology.

Relation of Sensitive Spots to Other Factors. Several other factors which might have been significant were found to have little or no importance here. The duration of pain and symptoms might have had an effect on sensitivity but did not appear to be so related. There were as many individuals with severe tenderness among those having their first attacks of cardiac pain as among those who had had several attacks over a period of many months or years. Age might have had an effect and a slight correlation with sensitivity was found. Sex might also have been related, since it has been found that the pain threshold of the female is lower than that of the male (12), but no such difference appeared either as related to spontaneous referred pain or to trigger spots.

It would thus seem, from the above data, that tenderness in focal somatic areas must be directly related to focal visceral disease and pain and to no other quality here examined.

DISCUSSION

It would be impossible in the present paper to discuss, with all its ramifications, the subject of the pain and tenderness which appears in somatic structures as the result of visceral disease. It has been dealt with by others from many aspects over many years and has been fully discussed recently by White, Smithwick and Simeone (6). There are, however, two points which are of particular interest: first, the structural nature of the trigger spot and second, the structural pathways of the referred cardiac pain. It must be assumed, before these matters are discussed, that the present investigation has confirmed the observations of others to the effect that cardiac disease has a positive relationship both to pain referred to somatic structures about the neck and shoulder and to sensitive trigger areas in the region of the referral.

The Nature of the Trigger Spots. There has been much discussion previously of the anatomy of trigger spots, those of the lumbar region being the most frequently considered. They have been associated with various types of visceral disease of abdominal or pelvic origin (13, 14, 15), but there is no convincing evidence as to the nature of their structure. All observers agree that these areas lie at the edge, rather than in the thick parts of muscle, in regions where loose connective fascial tissue predominates. All agree also that the spots are sharply localized and severely painful to pressure and that the spread of pain from the trigger point always follows a similar pattern.

Copeman and Ackerman (14), discussing the etiology of low back pain in a large series of young males, found a "pain pattern" lying about the lumbosacral regions of the back in normal as well as in affected subjects. These authors noted the appearance of sensitive focal areas up and down the back in individuals who had general

malaise and discomfort associated with virus infections or other febrile disease. They noted, further, that the sensitivity may remain after the pathological process subsides. This might account for the presence of trigger areas in the controls of the present series. It is substantiated further by the results of our examination of some 30 children on the wards of the Doernbecher Hospital carried out at the same time as our investigation of adults. In general these children, some of whom had cardiac disease and others nonthoracic disease, had exceedingly few trigger spots as compared to adults; those few who had painful lesions in cervical or thoracic parts did, however, have severely sensitive trigger areas. As an example, one child, almost recovered from a right mastoidectomy and in no present pain, had exquisitely painful and sharply focal spots on the shoulder and mid-clavicular points of the right side only.

Copeman and Ackerman (14) and Copeman (16) believed that the trigger spots are the result of some form of inflammatory process and resultant fibrosis. They implicate the fat lobules, stating that enlargement and herniation of fat into fibrous structure produces painful foci. These they were able to remove at biopsy and to identify in many, but not all, cases of painful backs. However, no such well-defined structures have been seen in biopsy material from focal painful areas about the shoulder (17).

Travell and her associates (10, 11, 17) have given detailed reports of trigger areas. These, they said, lie in relatively constant regions from patient to patient, usually in myofascial structures. They found that somatic skeletal muscle disorders may produce pain which is identical with that produced by cardiac excitation. The essential characteristics of the trigger zones are carefully defined by these authors: (1) When stimulated by pressure or needling, these areas give rise to a brief episode of referred pain perceived at a distance from the trigger spot. In the case of the precordial musculature, this circumscribes the trigger area. (2) The spread of pain represents a true reference phenomenon since it does not conform to an area supplied by a peripheral nerve. Their postulate is that: "Pain referred from the visceral lesion, in this case the heart, activates the somatic trigger lesion and the continuation of pain then depends on an autogenic cycle of nerve impulses maintained by the secondary sources on the somatic structures." They reported that, during biopsy, light touch of fascial sheaths may reproduce the pain and that in injecting trigger areas about the lower back and joints, there is often an area of increased resistance to needle prick at the site of the pain. In many cases of the present series the fingers of the examiner felt some such increased resistance and firmness above the area of focal tenderness.

Winter (13), discussing referred pain, believed that there are tender foci which result from fibrositis, lying in areas similar to those found in the present series. He identified these as being sharply defined,

discretely tender and provoking pain similar to that of which the patient complains, although he has not been aware of the tender areas. The pain, even in remote regions, is abolished by procaine infiltration of the "myalgic" spots. Winter likened the tenderness and pain to that produced by Lewis and Kellgren (18, 19) following the injection of hypertonic saline solution into and about the nerve roots. He believed that the common factor is an inflammatory one and that the local nerve endings develop hyperirritability as the result of a local circulatory disturbance. His conclusion was that overstimulation of vasoconstrictors by the inflammatory process produces local vasoconstriction and consequent malnutrition, the pain being similar in etiology to that produced by insufficient blood supply under other bodily conditions. Eventual trophic changes may follow.

It is our impression also that there are secondary focal changes, probably inflammatory or vascular in nature, about the trigger spots, since increased resistance to pressure is so often present in these sites. No firm focal bodies such as fat lobules, however, were ever felt. A reflex vasomotor reaction as suggested by both Travell (17) and Winter (13) should be the most likely mechanism to produce such changes.

In seeking an answer to the question of structure of the trigger spots our attention has become focused on the similarity in pattern between our diagram of focal trigger areas (fig. 1) and that of figure 2 which shows the "motor points" about the neck and thorax as described by Erb and reproduced by Monrad Krohn (20). It can be seen that all the major spots of tenderness are also major spots at which blood vessels and nerves lie near enough the skin surface to be reached by external stimuli. Since these spots are always between rather than under muscles and in loose fascial structures, the availability of nerve trunks lying there is obvious. Furthermore, the type of pain resulting from pressure there is of the same deep and uncomfortable type that is produced by pressure over nerve trunks elsewhere, such as the sciatic or ulnar nerves. It may appear spontaneously as the result of peripheral neuropathology. The pain produced by Kellgren (19) with hypertonic saline solution is similar in character and also appears in regions related to fairly large nerve trunks. Thus, an inflammatory process of any type, produced by sciatica, hypertonic saline solution or, as in the present instance, by reflex vasomotor changes, might result in a similar pain pattern.

The Nature of Referred Cardiac Pain. To elaborate the above structural concept one must dissect more deeply into the nature of the pain referral. Figure 3, taken from White, Smithwick and Simeone (6), is a diagram of the presently accepted course of sympathetic fibers to and from the heart. This is based on a very large number of clinical observations following either surgical section or anesthetization of these tracts. It will be seen that all cardiac pathways enter or leave

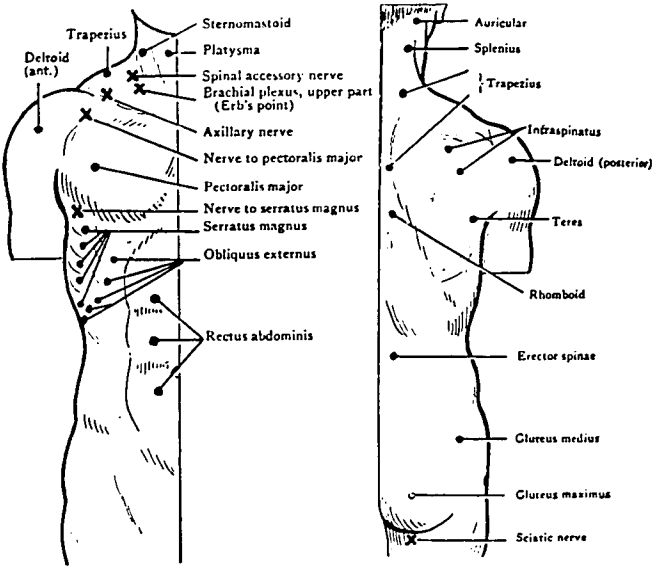

FIG. 2. Diagram of motor points that are excitable through the skin.—From G. H. Monrad-Krohn. New York, Paul B. Hoeber, Inc., 1941, pp. 176 and 177.

the spinal cord through the first to the fourth and sometimes the fifth thoracic segments. Either injection of these dorsal roots with alcohol or section bilaterally will completely and permanently abolish all pain of cardiac origin (6, 21). Removal of the upper thoracic sympathetic ganglia has a similar effect (6). Blocking of the stellate ganglion, on the other hand, will sometimes, but not always, free the subject of cardiac pain (22, 23, 24), and cervical sympathectomy has a similar partial effect (23). Leriche (25) stated that stimulation of the stellate ganglion may produce cardiac pain in all its manifestations, and Ward (26) has seen the coronary pain syndrome reproduced in a number of instances by a herniated cervical disc toward the left side at the seventh or eighth cervical segment. It is evident then that a painful shoulder such as follows myocardial insufficiency may result from several different sources of irritation all related to the lower cervical or upper thoracic cord levels.

As stated earlier, referred pain is thought to be the result of spread of excitation from afferent fibers of an injured visceral area to afferent fibers or synapses from somatic fields lying adjacent and at the same

level within the spinal cord (or possibly within the thalamus). But referred *cardiac* pain is very difficult to explain on this basis. The visceral segments involved by cardiac disease are the first through the fourth thoracic. The "referred" somatic segments involved, however, reach only as low as the first and second thoracic when the pain runs

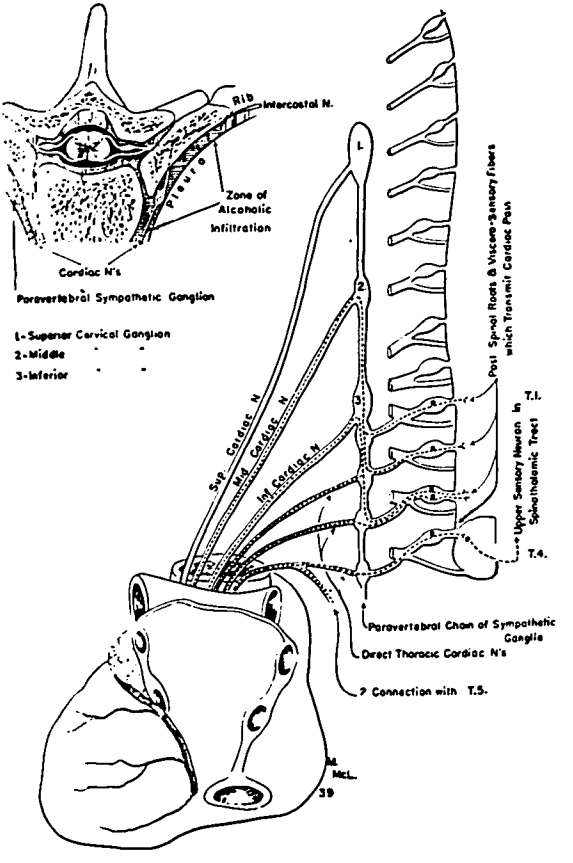

FIG. 3. Cardiac innervation. From J. C. White. Surg. Gyn. & Obs. 73, 334–343, 1940.

down the ulnar surface of the upper arm, and somatic pain is also "felt" at the second, third, fourth and fifth cervical segments in the neck and shoulder while the sixth, seventh and eighth cervical segments in the hand are little if at all affected, although pain is occasionally felt down the arm to the little finger.

If the referred pain is due to spread to somatic afferents within the cord this spread must jump several segments of cord to the higher cervical levels in order to produce neck pain without producing pain in the hand and finger. Some such spotty spread would also have to occur if the referral takes place in the thalamus rather than the cord, since it also has an orderly and segmental representation.

The matter is further defined by the fact that there are no autonomic connections known either to enter or to leave the cord directly through the cervical roots. As shown in figure 3, all the sympathetic fibers from the cervical periphery descend by way of the cervical ganglia to enter the cord in the upper thoracic levels. In these thoracic segments, then, which are also those reached by the cardiac afferents, spread of pain-producing afferent impulses might take place between *autonomic* cardiac afferents and *autonomic* afferents from the upper extremities, neck and shoulder. This would require the use of pain-bearing afferents from the autonomic innervation of somatic structures such as are known to exist in the walls of the larger blood vessels and nerves. Nervi vasorum or nervi nervorum from the regions of referral would then be responsible both for the pain and for the trigger tenderness. If this is so, it might even be possible that the spread of referral of pain occurred at synapses within the sympathetic ganglia at the affected levels as well as in the cord itself.

Structurally there is little that can interfere with this concept of referred pain pathways. The afferent autonomic pathways are known to exist (6, 27) and since it is always easier, as far as is known, to transfer or refer action potentials to structures of similar rather than dissimilar nature, the fine, little myelinated or unmyelinated, branching or multisynaptic neurones such as occur either in the somatic structures mediating painful impulses or throughout the sympathetic system are, functionally, excellent media for such spread of impulses.

The above described course of afferent autonomic fibers transmitting pain is further confirmed by the observations of Sweet and White (28) presented in a paper before the American Neurological Society, June 16, 1953, and published in abstract form in the program of that meeting. These authors stated that electrical stimulation of the superior cervical ganglion or of the trunk below it in man causes pain in the chest and arm more often than in the neck and head. But that, when the trunk is divided, stimulus to its rostral end either is painless or causes pain only in face and head, whereas any pain from stimulation of the caudal end is referred to the neck, arm or chest. The above data would appear to establish beyond a doubt the afferent pain path-

way by way of the cervical sympathetic chain. It would be interesting to know whether stimulation of the intact cervical chain produces trigger spot sensitivity as well as focal pain.

One final fact must be explained in order to complete this concept. Why are the proximal rather than the distal portions of the extremity involved by the pain referral? Here again, the concept of sympathetic afferents from the extremity is of assistance for it is always the large vessels and nerve trunks that lie proximally which have marked pain sensitivity, rather than the small distal branches.

Various methods of altering cardiac pain also strengthen this theory. Cold stimuli applied to the skin have been shown to induce cardiac attacks (29). Sympathectomy has relieved them (6). Trophic changes frequently accompany such pain (1, 2, 17), and treatment of focal areas with injection of procaine seems to have lessened cardiac pain and diminished frequency of attacks (11, 24, 29).

The results of upper thoracic ganglionectomy (inferior cervical through the fourth thoracic) in a large series of patients as reported by Lindgren (23) are of interest here. Although 100 per cent of patients treated in this way were relieved of their *referred pain*, only 80 per cent found relief from their *precordial pain*. According to our postulate this would be the result of having sectioned all of the incoming autonomic fibers from the areas of referral by way of cervical ganglia while the "direct" precordial pain remained because of untouched afferents entering from the heart through the fifth thoracic ganglion (fig. 2).

Finally, if it is confirmed by further observations, the concept offered here of pain referral as being chiefly autonomic-to-autonomic rather than autonomic-to-somatic may be useful in explaining some of the obscure pain syndromes occurring elsewhere in the body. If there is a system of afferent nerve fibers within the extremities which is related to autonomic distribution by way of the blood vessels and deep nerves rather than through segmental somatic patterns (27), the relief of causalgia by sympathectomy is explained and many of the deep and phantom pains which do not appear in dermatomal patterns must then be the result of this transmitting system which will spread and "refer" pain throughout its multiple synaptic ganglia in a much more effective manner than can the more discretely constructed segmental somatic system.

SUMMARY

The painful trigger spots of the upper thorax have been examined in both cardiac and control cases, and have been found to have direct relationship to pain of cardiac origin.

These focal points occur in specific areas which are of identical location in all subjects examined.

Although they appeared in some individuals who had no thoracic pain (20 per cent), they were more frequent in patients who had thoracic disease not of cardiac origin (48 per cent) and were much more prevalent in association with spontaneous cardiac pain (61 per cent).

In the cardiac cases laterality of spontaneous pain was associated with trigger tenderness of the same laterality.

It is suggested that the trigger areas are the result of focal inflammatory changes based on reflex vasomotor processes and that this fact accounts for their persistence when the visceral source of the pain is no longer present.

It is further suggested that the sensitive areas lie in close relationship to the larger and more superficial nerves.

Finally, it is suggested that the syndrome of referred cardiac pain can best be explained by the use of pathways of referral from the visceral autonomic source of excitation to other *autonomic* afferents from the shoulder and neck which enter the same ganglia and the same cord segments as do the cardiac afferents. Such a concept of referral from autonomic-to-autonomic, rather than autonomic-to-somatic would explain many other types of severe deep-lying pain which do not follow the usual segmental somatic patterns.

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NOTICE OF THE ANNUAL MEETING

The American Society of Anesthesiologists, Inc.

October 30–November 3, 1955

Boston, Massachusetts