A Method of Treatment for Pericardial Pain

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The usual case of acute idiopathic pericarditis presents relatively little problem in management, except for the relief of pain during the first few days of illness. Some patients, however, suffer a prolonged and relapsing course, which can result in considerable discomfort, and at times incapacitation. Therapy for this disorder is symptomatic, reliance generally being placed on salicylates, opiates, and sedatives. There are reasons for this. The mechanism of pericardial pain is not understood, making it impossible to provide other than nonspecific modes of relief. Moreover, the discomfort is rarely of sufficient intensity to cause the patient great concern.

We recently encountered a patient in whom the pain of acute pericarditis was so severe and prolonged that frequent doses of opiates were required for alleviation. This patient and a subsequent one received immediate, complete, and long-standing relief from a left stellate ganglion block, a method of treatment that, to the best of our knowledge, has not been previously utilized.

It is hoped that this report might stimulate re-exploration of the much neglected field of the mechanism of pericardial pain. In addition, a trial of this technic would seem worthwhile in the patient with the initial severe pain of acute pericarditis, and more particularly in the individual whose discomfort is recurrent and incapacitating.

Case Reports

Case 1

A 46-year-old white woman was admitted in August 1960 to the Medical Service of the Twenty-Eighth General Hospital. Twelve hours previously she had been awakened by severe, crushing, precordial pain, radiating to the neck and into the left shoulder, markedly aggravated by lying supine and with deep respirations. She noted that sitting up and leaning over offered partial relief; salicylates, however, were ineffective.

Temperature on admission was 99.4 F. The patient was extremely uncomfortable with rapid, shallow respirations; a "paradoxical pulse" of 12 mm. Hg was present. Physical examination was otherwise normal.

On admission serum glutamic oxalacetic transaminase was 0 units and serum glutamic pyruvic transaminase was 3 units. A complete blood count including a sedimentation rate was normal. Serial electrocardiograms (fig. 1) confirmed the diagnosis of acute pericarditis. Initial chest x-ray, compared with previous films, showed generalized enlargement of the cardiac silhouette (fig. 2), which returned to normal 2 weeks after admission. Skin tests for histoplasmosis, coccidioidomycosis, and tuberculosis were negative, as were L. E.-cell preparations and measurement of blood urea nitrogen.

Her course was extremely stormy. Initial hospitalization lasted 66 days, and was characterized by almost daily episodes of severe chest pain, incapacitating the patient and necessitating frequent doses of opiates. A transient friction rub was noted on the fourth day of hospitalization. All attempts to increase the patient's activity resulted in a recrudescence of pain, inevitably persisting until large doses of narcotics were given. Eventually she began to experience improvement, and was continued on modified bedrest as an outpatient.

Two weeks after discharge she was suddenly seized by excruciating pain which was then located higher in the chest, and radiated to the neck and posterior scapular region on the left. Again the relationship to position and respirations was apparent. As with previous episodes of pain, the electrocardiograms showed generalized T-wave inversion (fig. 3). The "paradoxical pulse," which had been present on the first admission, was not evident. High doses of narcotics, which had not been necessary during her convalescence at home, were again required but afforded only partial relief.

The patient became extremely depressed and emotionally labile, in marked contrast to her cheerful, optimistic personality prior to the illness. In desperation, 15 ml. of 1 per cent lidocaine were injected about the left stellate ganglion. Within 15 minutes she was completely pain free. A typical Horner's syndrome was observed on the
Figure 1

Top. Electrocardiogram, taken 4 hours after admission of patient 1, showing symmetrical inversion of T waves across the precordium. Middle. After 8 days of hospitalization; T-wave inversion is more pronounced. Bottom. Taken on sixty-fourth day; there is marked improvement, with T-wave inversion manifest only in $V_2$ and $V_3$. 

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left, as well as increased warmth and flushing of the left upper extremity. To our gratification, she remained asymptomatic for 6 days. Again, however, pain recurred, which was located primarily in the precordial region, and was of only moderate intensity. To test the possible placebo effect of the previous treatment, the stellate block maneuver was repeated, with the injection of 3 ml of saline solution.

The patient noted some lessening of her discomfort, but this was by no means complete, and the pain recurred the following morning. She continued to require large doses of opiates, and 4 days later, when the pain increased in severity, a third stellate block was performed. On this occasion, as had been done originally, 15 ml of 1 per cent Lidocaine were administered, and again within 10 minutes the pain was completely relieved.

Following the first and third injections, movements of the left hemidiaphragm were observed under fluoroscopy, establishing that the phrenic nerve had not been inadvertently blocked. The patient has resumed full activity, and has been observed for 3 months without recurrence of pain.

Case 2

In October 1960, a 30-year-old soldier awoke with crushing, precordial chest pain that was markedly increased by deep respirations or lying supine. He obtained relief only by sitting up and leaning forward.

The patient was extremely restless and in severe discomfort. The physical examination otherwise was within normal limits except for a “paradoxical pulse” of 15 mm. Hg.

Serial electrocardiograms (fig. 4) showed the typical evolution of acute pericarditis. The cardiac silhouette was not enlarged on x-ray. Serum glutamic pyruvic transaminase was 3 units and serum glutamic oxalacetic transaminase was 2 units. Laboratory tests for other causes of pericarditis were negative.

The patient received narcotics during the first several days of hospitalization with considerable diminution of pain. It was noted, however, that in the evenings he would suffer a severe recurrence, located primarily in the left side of the neck and left scapular region. Moreover, he stated that he was actually never free of pain. Because of our experience with the first patient, a left stellate ganglion block was performed on the seventh hospital day. Within 10 minutes the patient developed both a Horner’s syndrome and erythema and warmth of the left arm. In 30 minutes his pain had completely disappeared. The remainder of his hospital course was uncomplicated, although a friction rub was heard from the tenth to the twenty-fifth day of his hospitalization. He has experienced no
recurrence of pain during a 2-month follow-up period.

Discussion

The cause of the pain in pericarditis is obscure. In Gray's Anatomy it is stated that the nerve supply to the pericardium involves the vagus and phrenic nerves, and also sympathetic trunks. In other basic texts afferent fibers from the pericardium are said to be carried through sympathetic and parasympathetic thoracic ganglia. White, and also Bonica, stated that the pericardium is pain-sensitive only on its diaphragmatic aspect, the result of fibers supplied from the left phrenic nerve as it courses toward the diaphragm. They explain the radiation of the pain to the left shoulder and its relation to respiration on this basis. Roberg, while accepting this explanation, believed that the answer may lie in the distortion or compression of mediastinal structures, including the heart itself, by stretching of the pericardium during the inflammatory stage of the disease. Capps et al. believed that the pericardium is insensitive to pain. Carmichael stated frankly that the cause of the recurrent pain in pericarditis is not understood.

Myocardial pain sensitivity, on the other hand, a much more important and prevalent clinical problem, has received a proportionally greater share of attention. Unfortunately, its mechanism is also far from clarified. Herrmann concluded that the autonomic nervous system, including the vagus nerve, superior, middle, and inferior cervical ganglia, the inferior, middle, and superior cardiac nerves, as well as the first through the fifth thoracic ganglia, including the posterior spinal roots, are involved in the mechanism of myocardial pain. He stated that various types of surgical procedures, including the division or resection of all three cervical ganglia, or of the stellate ganglion alone, have resulted in complete and sustained cessation of anginal pain. He makes no mention, however, of the pain pathways of the pericardium.

The stellate ganglion is derived from the last cervical and first thoracic sympathetic ganglia. In both of our patients, the pain was markedly affected by respiration, and was located in the shoulder area. This observation supports the contention of those authors who think that the nerve supply to the pericardium may course along the phrenic nerve. The phrenic is formed from cervical segments three, four, and five, and since both patients
experience complete relief from stellate ganglion block, it seems unlikely that the distribution of the pain is the result of phrenic nerve involvement.

The block was performed as recommended by Moore, who noted many conditions, including angina and “cardiac” pain, for which this procedure is indicated. He stressed that the development of a Horner’s syndrome does not necessarily indicate stellate ganglion block, as this syndrome may result from blocking any cervical sympathetic ganglion.

Increased warmth, vasodilatation, and increased dryness of the skin of the left arm must be present in addition, as it was in both of our patients.

Our rationale for the employment of this procedure is actually fairly obvious, since it has been used successfully for myocardial pain on many occasions. We planned to follow with a left phrenic block if the stellate block were unsuccessful. It is difficult, of course, to eliminate the nonspecific effect of suggestion, particularly when one injects in such an un-

Figure 4
Top. Electrocardiogram taken 6 hours after admission of patient 2. Early S-T elevation is evident in lead II and also in V5 and V6. Bottom. Four days after admission. T-wave inversion emerging in leads III and aVF as S-T segments return toward the baseline.
usual area. The dramatic and complete relief experienced by the patients when Lidocaine was used, as contrasted with the minimal effect of saline, seems good evidence that our objective was truly achieved.

It is realized that most patients with uncomplicated pericarditis do not require therapy of this sort to relieve discomfort. Nevertheless, it might fruitfully be employed in the early stages of the disorder when the pain is severe and, in addition, in the occasional patient who develops incapacitating recurrences. Rehabilitation may be hastened, and severe emotional setbacks minimized.

One might also speculate whether such therapy, if tactfully and reassuringly employed, might not be attempted in the much more common problem of acute myocardial infarction, particularly in those cases that do not respond satisfactorily to narcotics and other traditional methods of therapy.

Because of the source from which our patient material is drawn, primarily young soldiers and their families, we have not had occasion to test the latter hypothesis.

Summary

Two patients are presented, in whom dramatic, complete, and prolonged relief from the pain of acute pericarditis was afforded by left stellate ganglion block. In one, this method was successful after the patient had been virtually incapacitated by pain for 75 days. As far as we have been able to ascertain, this mode of therapy has not been previously employed. The distribution of the pain seemed to implicate the phrenic nerve; however, the relief secured by stellate ganglion block appears contrary to this traditional theory.

It is concluded that the mechanism of pericardial pain is not completely understood but that the current, generally accepted explanations are quite likely inaccurate. The inferior cervical and first thoracic sympathetic ganglia may play a large, if not complete role in the transmission of these pain impulses.

There are several practical aspects to our observations. Stellate ganglion block might be logically employed during the acute phase of pericarditis if the pain is severe, and also when the patient is incapacitated by recurrences. There remains also a virtually unexplored field, the relief of the pain of acute myocardial infarction by this procedure.

References

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