Early Relief of Chest Pain by Ethyl Chloride Spray in Acute Coronary Thrombosis

Case Report

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Within a few minutes after its onset, intense pain of acute myocardial infarction was abolished by briefly spraying the precordium with ethyl chloride. Prompt relief of pain did not prevent tissue necrosis, but may have ameliorated the clinical course. We infer that the stimulus for pain in acute infarction is of extremely short duration but gives rise to a secondary, self-sustaining pain cycle which may be terminated by altering the flow of nerve impulses from the skin. The case also illustrates the increased susceptibility to digitalis which develops some days after myocardial infarction.

Since we emphasized the forgotten fact that blocking trigger mechanisms in the chest muscles is of value in controlling pain due to a fresh myocardial infarct, we have often been asked how soon after its onset the pain of acute coronary thrombosis can be relieved by local block therapy. We refer to procaine infiltration of trigger areas in the skeletal muscles and spraying of the overlying skin with ethyl chloride. The following case report provides an answer to this question, since the patient was a physician and was able to apply this method of treatment (ethyl chloride spray) very early in the course of infarction.

Case Report

W. T., aged 79 years, a physician. On Oct. 5 and 6, 1949, on three occasions, the patient noted transient mild substernal pain on walking. He had been entirely free of chest pain since his second coronary thrombosis in May, 1945.

At about 10 p.m. on Oct. 6 (termed the second day of illness), while driving in his car, he had severe substernal pain which radiated across both sides of the chest and to the inner aspect of both elbows. He took glyceryl trinitrate (nitroglycerin) at once, and again at home 10 or 15 minutes later, without influencing the pain. He then sprayed his chest with ethyl chloride in interrupted sweeps in a hit-or-miss fashion. After one minute of spraying the pain stopped completely.

When seen by Dr. Harry Gold soon afterward, the patient was comfortable. There were no signs of heart failure. Blood pressure was 120/60, and heart rate, 60 per minute and regular. Rectal temperature was normal. A presumptive diagnosis of myocardial infarction was made. No morphine was given. The patient had been taking digitoxin, 0.2 mg. daily by mouth, for about one year and this was continued.

The patient slept soundly all night. He was awakened at about 8 a.m. by a return of the severe substernal pain with radiation to the elbows. He relieved the pain again in about one minute by spraying the front of his chest as before. Later in the morning an electrocardiogram was taken which, except for the T wave in Lead IV, resembled the last previous one, a year and a half earlier. Both tracings (fig. 1, C and 1) showed some depression of the S-T segments attributable to the digitoxin. The P-R interval was 0.24 second in both. There was no leukocytosis (white blood cells 8,200, polymorphonuclear leukocytes, 65 per cent).

When I first saw the patient (about noon of the third day of illness), moderate substernal and precordial discomfort had just reappeared. Pain was relieved promptly by ethyl chloride spray. Two exquisitely tender spots were then found in the outer part of the left pectoralis major muscle at the level of the second costochondral junction; each of these was infiltrated with about 1 cc. of 0.5 per cent procaine hydrochloride in saline. Diffuse radiation of pain throughout the precordium was produced momentarily by these injections. Another tender spot at the same level just lateral to the left sternal border was similarly infiltrated, with radiation to the parasternal region. A fourth trigger area was located on the front of the sternum just to the left of the midline, in what was judged to be the vari-

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able sternalis muscle. When the needle was introduced into the myofascial tissues at this site, the pain reference matched the distribution of pain experienced at the onset of the attack. The patient had no further chest pain until about 8 a.m. on the following day (fourth day of illness), when a continuous but mild ache developed across the upper precordium. When I saw him about an hour later, he had not bothered to use the spray. Another trigger area was found at a different site in the left pectoralis major muscle than had been injected the day before. This was similarly blocked by infiltration. There was no more chest pain at any time.

An electrocardiogram (fig. 1, 2) taken on the fifth day of illness, compared with that of the second day, showed that S-T1 had become further depressed and a depression of S-T2 and S-T4 had appeared with flattening of T1 and T4 and inversion of T2; the P-R interval had lengthened, from 0.24 to 0.27 second (lead II). These serial changes, although masked to some extent by the digitalis effects, together with the rise in rectal temperature to 100.5 F. and fall in blood pressure to 100/70 on the fourth and fifth days, respectively, confirmed the initial diagnosis of myocardial infarction. The blood sedimentation rate (Westergren) which was 24 mm. in one hour on the third day, was 70 mm. on the ninth day. Subsequently it showed a gradual return to normal; 50 mm. on the seventeenth, 27 mm. on the twenty-ninth, and 21 mm. on the forty-third day. Low-grade fever lasted eight days.

At no time did the patient appear critically ill. He was permitted to get up once a day and walk a short distance into the bathroom. However, on the eighth day, without any concomitant symptoms, the heart rate suddenly dropped from about 60 to

![Fig. 1. Serial changes of acute myocardial infarction, on which are superimposed changes in S-T segments due to digitoxin administration and withdrawal. (C), control taken a year and a half earlier; daily maintenance dose of digitoxin, 0.2 mg. (1), (2), and (3), same daily dose of digitoxin (0.2 mg.). Note progressive intensification of digitoxin effects with complete A-V dissociation in (3) on eighth day after first appearance of chest pain. (4) and (5), no digitoxin for 2 and 15 days, respectively. Note gradual return of S-T1, S-T3 and S-T4 to normal coincident with digitoxin elimination, and simultaneous deepening of S2, inversion of T3, and increased amplitude of T4, as in posterior wall infarction. (6), taken 16 days after resuming digitoxin; daily maintenance dose, 0.1 mg. Some depression of S-T3 is again evident.](attachment://image.png)
It did not return to its previous level of 60 to 70 until he had rested in bed for six to seven hours. Because of this cardiac acceleration, digitoxin was resumed. A daily dose of 0.2 mg. was given for one week, and 0.1 mg. thereafter.

An electrocardiogram (fig. 1, 5) was taken on the thirty-third day, after four weeks without digitoxin. The digitalis effects had regressed; the P-R interval was still 0.24 second; notching of QRS; and deepening of S2 had appeared. Another electrocardiogram (fig. 1, 6) was taken on the forty-ninth day, after 17 days of digitoxin. The flattening of T4 in this tracing may represent a digitalis effect, but the deepening of Q3, inversion of T1, and increased amplitude of T4, as compared with tracings taken during digitalization (fig. 1, C, 1 and 2) are suggestive of recent infarction of the posterior wall.

This was the patient’s third attack of acute coronary thrombosis. The first occurred in April, 1943, and the second in May, 1945. Each time, unremitting chest pain had been relieved at once by procaine infiltration of trigger areas in the precordial muscles, as described under case 1 in a previous report. Recovery from each attack was apparently complete, and the patient had played tennis with fair regularity, summer and winter, up to a few weeks before the third infarction. There were never any signs or symptoms of congestive failure; slight edema of the ankles was attributed to varicose veins. Arterial circulation in the extremities was excellent, although the vessel walls were of “pipestem” quality with some calcification on x-ray examination.

**DISCUSSION**

In this case of acute myocardial infarction, intense substernal oppression with pain radiation to both elbows had been present for only 10 or 15 minutes when brief application of ethyl chloride spray to the front of the chest immediately stopped all pain. When pain recurred in 12 hours, and then 4 hours later, it again ceased at once after the chest had been sprayed intermittently during only a minute.

As a physician, the patient was familiar with the technique that we had previously described for applying ethyl chloride spray in rhythmic interrupted sweeps, which avoids frosting of the skin or aching due to excessive cold, and which often relieves skeletal muscle pain dramatically. It is difficult for most patients themselves to apply this material properly, but in this case the area to be treated, the front of the chest, was readily accessible and the patient well versed in handling the spray.

The onset of severe pain on the second day of illness probably marked the time of sudden myocardial infarction. However, the preliminary episodes of mild substernal pain suggest that thrombosis in the coronary tree and narrowing of the lumen began about 36 hours prior to closure of one of its branches. Further extension of the thrombus with additional closures may have occurred with each recurrence of severe pain on the third and fourth days of illness. It is possible that the development of complete A-V dissociation on the eighth day marked a further extension of the thrombus, but it seems more likely that this event represented increased susceptibility to digitalis action, which is known to develop, not immediately, but some time after a myocardial infarction.

It should be noted in this case that the early relief of pain, almost coincident with the major closure, did not prevent signs of tissue necrosis from appearing subsequently. However, the mildness of the clinical course in this 79 year old man with a history of two previous myocardial infaracts leads one to speculate as to whether the early blocking of referred pain from the heart may not have played a beneficial role.

There is evidence to show that the discharge of high intensity impulses from a trigger area in the skeletal muscles may cause localized vasoconstriction in regions specifically related to the trigger mechanism. The regions subject to such reflex vasospasm include not only the somatic reference zone of pain, which is relatively constant from person to person for a given trigger area, but probably include also specific regions of the brain, spinal cord and viscera. Furthermore, with respect to the heart, Lindgren has demonstrated that neural impulses from the superficial structures of the chest may contribute to the pain of angina pectoris. In patients with this effort syndrome, precordial local anesthesia (procaine infiltration) increased the capacity for work of cardiac...
musclemuscle as measured by exercise tolerance and anoxia tests, and hence, one may conclude, improved the coronary flow. In this patient, therefore, it seems highly possible that blocking impulses from trigger areas in the chest muscles by ethyl chloride spray contributed toward the release of vasospasm in the coronary tree. This in turn would lead to diminution in reflex ischemia surrounding the infarct and to reduction in the ultimate size of the necrotic area.

One may well ask why, when ethyl chloride spray had already relieved pain, the tender spots in the precordial were injected with procaine. This procedure was based on the concept of the latent trigger area, namely, that silent trigger mechanisms may exist with thresholds of excitability just below the critical level necessary to produce spontaneous pain. For example, at the time when this patient had no chest pain, the introduction of a needle into the latent trigger area in the sternalis muscle set off referred pain exactly like that which had attended the initiating event of cardiac infarction. Whether the discharge of subthreshold stimuli from such silent trigger mechanisms is of clinical importance cannot be stated at the present time. However, our experience with many types of painful muscle syndromes indicates that a latent trigger area may be readily activated by minor strains and stresses to produce periodically its full-blown pattern of referred pain and reflex vasomotor and other autonomic concomitants. On the basis of these observations, the logic of blocking all latent trigger areas in the chest muscles as a prophylactic measure against further coronary vasospasm is clear.

If one grants the wisdom of eradicating all possible sources of noxious stimuli under these conditions, the next question is: Could this not be accomplished equally well by repeated applications of ethyl chloride spray to the appropriate areas? The therapeutic effect of ethyl chloride spray indicates that afferent impulses from the skin play an important role in the mechanism of deep pain, and that under suitable conditions altering cutaneous stimuli may relieve myofascial and visceral pain in an extraordinary manner. Nevertheless, it is our impression that the latent trigger mechanism may be permanently abolished with greater certainty by direct infiltration of the trigger area itself.

**Summary**

Momentarily spraying the front of the chest with ethyl chloride at the onset of myocardial infarction abolished the substernal and radiating arm pain at once. When pain recurred several hours later, the procedure was again immediately effective. The latent trigger areas in the chest muscles were blocked by local procaine infiltration at a time when the patient was free of pain.

The mildness of the clinical course of myocardial infarction in this 79 year old man suggests that these procedures may have had a beneficial effect on the compensatory coronary circulation. This conclusion is in harmony with the known effects of somatic trigger mechanisms on visceral function (motorovisceral reflexes).

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