

NON-FATAL STAB WOUNDS OF THE VENTRICLES WITH  
ELECTROCARDIOGRAPHIC SIGNS OF CORONARY  
THROMBOSIS AND ABSENCE OF ANGINAL PAIN.

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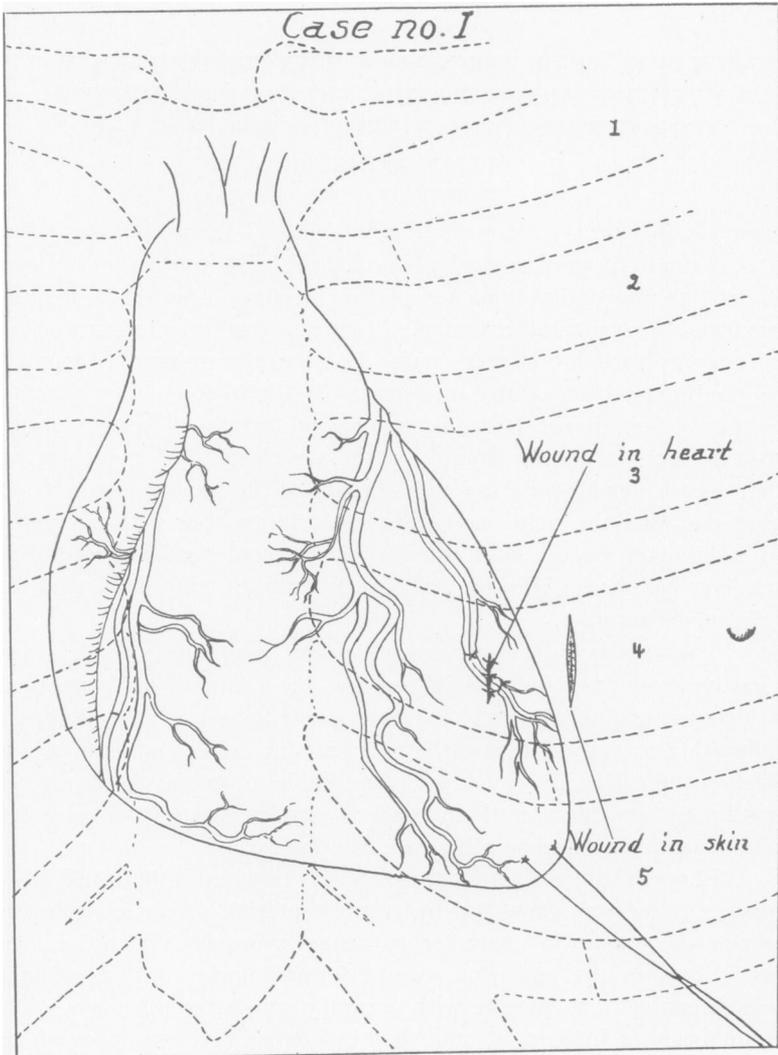
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Wounds of the ventricular musculature with and without injury to one of the major branches of the coronary arteries have been reported in considerable numbers in current medical literature. The electrocardiographic changes noted by observers using this method of study have been similar in essential details to those seen in acute coronary thrombosis. It has been our good fortune to have for study two patients with stab wounds of the ventricles; one involving the left ventricular musculature and a branch of the left coronary artery, and the other the right ventricular musculature, but with no injury to a coronary vessel. Both patients were treated surgically and each patient has been followed sufficiently long to note his complete clinical recovery.

The electrocardiograms are of interest in that the changes are positive and are produced by lesions, the location and extent of which are definitely known. It is reasonable to assume that the heart muscles were normal before the accident, for neither patient gave a history indicating any previous disease liable to produce heart muscle pathology, and their youth and general physical survey precluded the existence of any degenerative vascular disease.

In one instance a coronary artery was tied and in both instances muscle tissue was enmeshed by suture material, producing a lesion somewhat similar in character to acute myocardial infarction. In each instance the operation was performed under local anesthesia which enabled one to note with accuracy subjective phenomena. It is of peculiar interest to note that in neither case was pain of an anginal type noted during or following the procedure.

This report is presented not only to place on record patients with wounds of the heart, one of the right and the other of the left



**ILLUSTRATION No. 1.**  
Showing the approximate location and size of the wound in the left ventricle  
and the severed coronary vessel.

ventricle, who were treated surgically with recovery, but particularly to record the clinical behavior of the patients, noting the absence of pain in heart lesions with electrocardiographic changes similar to those observed in coronary occlusions and produced probably by myocardial changes of the same fundamental nature.

#### CASE REPORTS.\*

Case I. W. R., negro male, aged 23 years, was admitted to the St. Philip Hospital October 2, 1930, at 12 noon. He had been stabbed in the left side of the chest about thirty minutes before admission. He later stated that he walked one and a half blocks after being stabbed before he began to feel faint, and estimated that ten to fifteen minutes elapsed between the time of injury and the appearance of marked weakness. When first examined in the hospital he appeared to be in profound shock. The radial pulse was not detectable and his blood pressure was not obtainable. Examination of the chest showed a stab wound in the fourth intercostal space five cm. to the left of the sternum. The heart sounds were distant and muffled and the cervical veins were full. These signs were thought to indicate hemopericardium with cardiac tamponade. An operation was deemed urgent and was performed under local infiltration anesthesia. When the pericardium was incised 200 or more c.c. of blood was evacuated and bright red blood was found to be spurting from a wound in the left ventricle. A wound one cm. in length was found on the left border of the heart (Illustration 1), parallel to its long axis and about five cm. from the apex. The descending branch of the left circumflex coronary artery had been divided. The wound was closed with three interrupted sutures and the bleeding artery ligated. Incision of the pericardial sac produced a prompt improvement in the pulse rate and volume.

The patient was discharged from the hospital 16 days after the injury. He has been studied frequently since leaving the hospital and all methods of study indicate complete recovery. Chart 1 illustrates the electrocardiograms taken while in the hospital and since his release.

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\*A detailed report of the surgical aspects of these cases will appear in the proceedings of the Southern Medical Association for 1931.

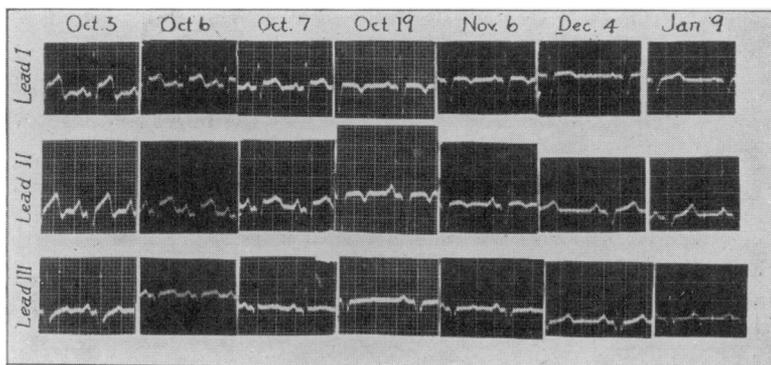


CHART NO. 1, CASE I.

The first electrocardiogram was made 14 hours after the operative repair of the wound. The changes noted are characteristic of those seen in coronary thrombosis. The electrocardiogram made on January 9 shows a complete return to normalcy.

Case II. W. J., negro male, aged 36 years, entered St. Philip Hospital at 11 P.M., July 4, 1931. He stated that he had been stabbed in the chest with a pocket-knife about 7 P.M. After being stabbed he ran about 50 yards and then fell to the ground unconscious. He was put in a car and carried five miles to a physician, who noted that the patient was bleeding freely from the chest wound and was in profound shock. He was given morphine and atrophine and placed on a table with his feet elevated. In about fifteen minutes the pulse became palpable and the skin warm. The patient was brought 20 miles to the hospital and when admitted the systolic blood pressure was 112 mm. Hg., pulse 90 per minute. A stab wound was noted five cm. from the midsternal line in the left fifth intercostal space. The area of cardiac dullness was not definitely increased, but the heart sounds were distant and muffled. A diagnosis of stab wound of the heart was made from the history, suggesting a cardiac tamponade, which had probably been relieved by the free bleeding from the chest wound. The operation was performed under local infiltration anesthesia. Only a moderate amount of blood was found in the pericardial sac, but there was a constant flow of dark blood from a transverse wound 1.5 cm. in length in the midportion of the anterior wall of the right ventricle about five cm. above the

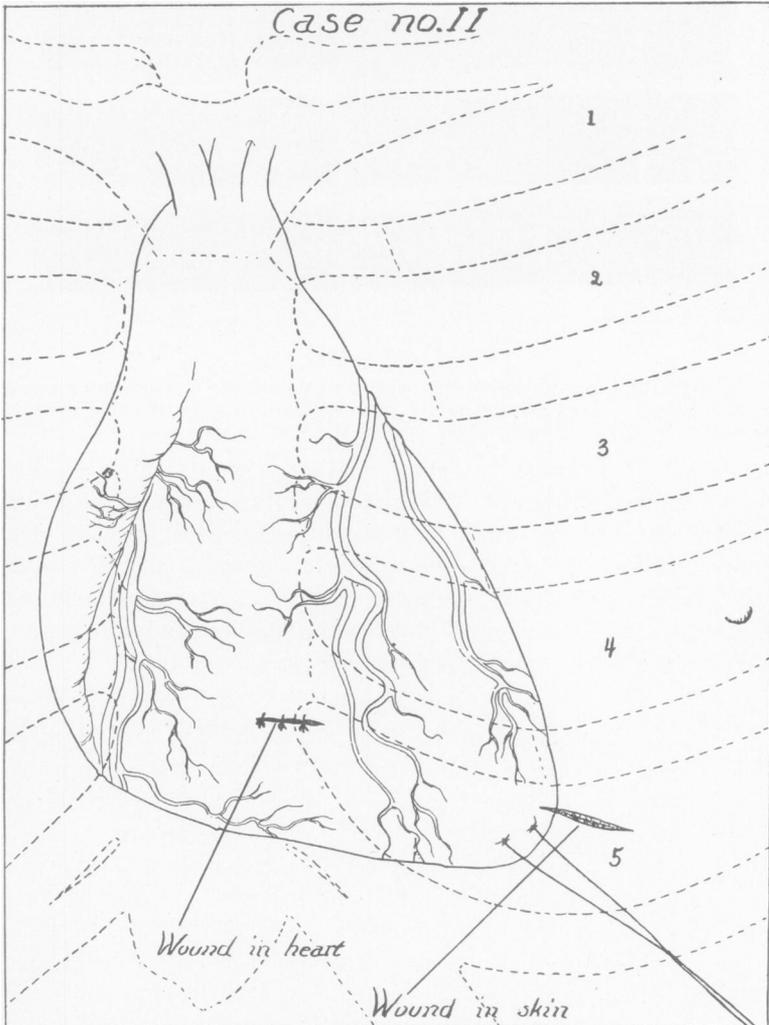


ILLUSTRATION No. 2.  
Showing the approximate location and size of the wound in the right ventricle.

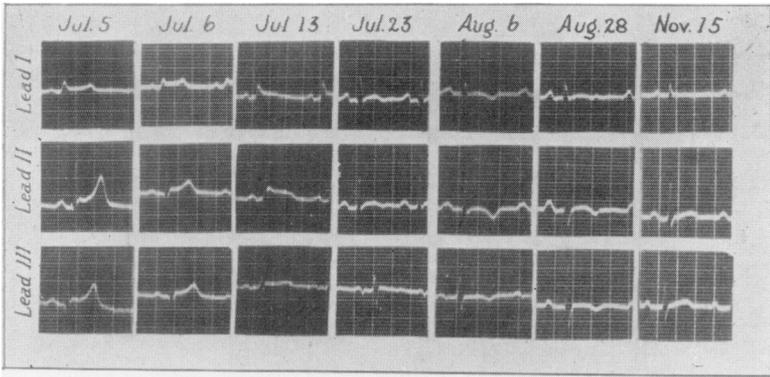


CHART 2, CASE II.

The first electrocardiogram was made nine hours after the operative repair of the wound. The changes noted are characteristic of those observed in coronary thrombosis involving this area of the heart muscle.

apex level (Illustration 2). The wound was closed with three interrupted sutures of fine silk. The patient's course in the hospital was uneventful, except for a mild local staphylococcal wound infection. The patient has been followed since leaving the hospital and all studies fail to reveal pathologic aftermath. Chart 2 illustrates the electrocardiographic changes noted while in the hospital and two months after his discharge from the surgical service.

#### DISCUSSION.

The lesion produced in the heart muscle in each case consisted of a puncture wound from a sharp-edged weapon which segmented muscle fibers and in Case I sectioned a branch of the left coronary artery. The end result following surgical repair was an area  $\frac{1}{2}$  cm. in length with the muscle fibers enmeshed in suture material and drawn sufficiently tight to control bleeding, and in Case I the injured coronary vessel was ligated. It is reasonable to assume that local ischemia existed at the point of the repaired wound and in Case I in the area supplied by the artery, which was tied. In each instance collateral circulation might conceivably overcome the induced ischemia, but this would not occur for several hours and probably not for a much longer period.

The electrocardiograms made following the operations are similar in every respect to those noted in coronary occlusion, and proba-

bly have the same pathologic significance. The only other logical explanation for significant changes in the electrocardiograms would be the effect of an accumulation of fluid in the pericardial sac. This factor was excluded by Roentgen ray studies at frequent intervals. For the first few days following the operation these studies were made daily. Fluid did accumulate in Case I to the extent of embarrassing the venous return to the heart eighteen hours after operation. It is interesting to note that changes previously observed in the electrocardiograms remained constant after the fluid was evacuated by opening the wound, yet all phenomena of cardiac tamponade were promptly relieved.

The infrequent association of cardiac pain with wounds of the heart muscle is of peculiar significance. In the two cases here presented pain of anginal type was entirely absent at any period before, during or after operative repair and the surgery was done with the use of a local anesthetic injected only in the chest wall. Neither patient was sufficiently ill at the time of the operation or afterwards to modify the perception or consciousness of pain. It may be profitable therefore to consider the significance of the absence of pain in heart muscle wounds, especially those involving the coronary vessels as related to the pathogenesis of cardiac pain in angina pectoris.

Keefer and Resnick<sup>1</sup> have recently reviewed completely the accumulated evidence dealing with the various factors concerned in the pathogenesis of angina pectoris. They contend that the most tenable hypothesis is that this syndrome is related directly to conditions which induce an ischemia of the heart muscle, and that the pain of coronary thrombosis is of the same fundamental nature. They comment upon the interesting clinical fact that the pain of myocardial infarction tends to subside with the development of heart muscle degeneration and eventually disappears entirely.

Sutton and Lueth<sup>2</sup> have recently presented experimental evidence, which they feel proves conclusively that myocardial ischemia is fundamental in the production of cardiac pain.

The studies of MacWilliam and Webster<sup>3</sup> on the sensory phenomena associated with defective blood supply to the working muscle indicate that the pain of ischemia occurs only when the muscle is exercised under conditions including inadequate oxygen supply.

Lewis<sup>4</sup> and his co-workers in their studies in intermittent claudication have shown that the pain in this syndrome is due to a substance generated during muscle contraction which accumulated to a concentration sufficient to induce pain and remain stable in the tissue spaces during circulatory arrest.

Angina pectoris occurs in those patients in whom contractile power of the heart muscle is well preserved and frequently disappears when congestive heart failure supervenes. This would indicate that the pain is caused by a substance dependent upon muscle contraction under special conditions, for those factors which induce pain do so by increasing heart muscle work. If one transfers the recent observations made on the mechanism of pain in skeletal muscle to the wounds of heart muscle here studied, one finds a reasonable explanation for the absence of pain, although heart muscle ischemia existed.

The wound in either of the cases presented consisted primarily of a severance of the muscle fibers extending in depth the entire thickness of the heart wall. The area of ischemia resulting from repair of the wound, which in one involved the placing of a ligature on a coronary artery, embraced an area of heart muscle which had been so injured by the penetrating weapon that normal contraction could not occur. The pain causing metabolites which form during muscle contraction and accumulate in the tissue spaces to a concentration sufficient to induce pain were therefore not produced, though absolute or relative circulatory arrest existed. That the absence of pain was due to a rapid relief of ischemia by collateral circulation is not tenable. The nature of the lesion and subsequent changes in the electrocardiogram indicate that an injury to the heart muscle similar in nature to that occurring in coronary thrombosis was induced by the wound and operative repair.

A case reported by Burian<sup>7</sup> of a wound of the heart which severed a branch of the left coronary artery, but involved only superficial layers of the heart muscle has a direct bearing on the cases under consideration. His patient, following the repair of the wound, which consisted mainly in tying the bleeding vessel, had postoperative anginal pain which, he stated, was a troublesome symptom. The anginal attacks were attributed by him to an area of heart muscle

anemia. In the light of recent data and the cases here studied it would appear logical to assume that the origin of pain was due to the contraction of heart muscle under ischemic conditions, the superficial nature of the lesion not interfering with this function in the area supplied by the severed vessel.

This case is contradictory of the observations of Sutton<sup>2</sup> and Lueth, for they noted that pain was not produced in dogs if the continuity of the vessel or the adventitia was broken.

If the absence of pain in patients with penetrating wounds of the heart muscle, who have as a result of the wound and operative repair an area of myocardial ischemia, be here given the correct explanation, it logically follows that certain aspects of angina pectoris are hereby accounted for. The angina pectoris of effort is relieved by cessation of exercise, for the degree of myocardial ischemia is insufficient to allow the pain producing substance to remain in the tissue spaces in sufficient concentration, under the conditions of reduced rate of formation consequent upon body rest. The pain of coronary occlusion with acute ischemia in the infarcted area of heart muscle gradually lessens with the development of muscle degeneration and finally ceases when this has developed to a degree incompatible with muscular contraction.

These observations emphasize the importance of myocardial ischemia as a fundamental factor in the mechanism of angina pectoris, but at the same time suggest that heart pain will not occur if the ischemic area involves muscle tissue degenerated sufficiently to impair its contractile power.

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## DISCUSSION.

DR. CHARLES C. WOLFERTH: I have been very much interested in Dr. Porter's paper, which I have had the privilege of going over, because for years my associate, Dr. Wood, and I have been trying to study in animals conditions somewhat analogous to those which Dr. Porter has observed clinically.

There are several points of interest and great importance, I think, brought out by Dr. Porter's presentation this morning. In the first place it is remarkable how much trauma the heart can endure sometimes. It is equally remarkable—a point not illustrated by these cases—how little trauma will sometimes set the heart into ventricular fibrillation.

Dr. Wood and I have found that by manipulation of the thread of the heart one can do a great deal to it frequently without disturbing the heart very much. However, when the posterior surface of the heart is manipulated with even a small branch of the posterior circumflex of the left coronary artery occluded, one may very quickly find the animal developing ventricular fibrillation.

A second point, Dr. Porter's cases add to the ever growing list of cases in which successful surgery of the heart has been accomplished. His electrocardiograms show very clearly, I think, the injury of the heart.

With regard to the mechanism of pain production, there have been many hypotheses proposed, as well we all know. We remember the controversy of the coronarians versus those who believed that the pain of angina pectoris was produced in the aorta, the latter view being particularly upheld by Albot and Rickenbaugh. The development of our knowledge of coronary occlusion has made the position of Albot and Rickenbaugh one of considerable isolation, and we know that pain can arise from aortic lesions, but the type of pain which occurs with angina pectoris does not seem to be the aortic type of pain, so at the present time I think most observers believe that the pain in angina pectoris and in coronary occlusion is produced either in the artery itself or is produced in the ischemic part or area of muscle.

Dr. Porter's hypothesis is a very attractive one. It is worth remembering, however, that the blood supply of the heart is unique in that one has the coronary arteries, the coronary veins, and the sinusoidal vessels, so that it is possible that X substance might be carried off fairly readily from an area of ischemic muscle.

Furthermore if one occludes a coronary artery in a dog, the area occluded very quickly becomes extremely cyanotic, begins to dilate, and in a very short time one does not see any visible pulsation or contraction in this area, so that I would be a little dubious about a continuation of contraction in the area involved by a lesion of the coronary artery being responsible for continuance of pain over long periods of time.

The work of Sutton and Lueth interested me very much because these observers believe that the nerves carrying pain must go along arteries. They found that if a coronary artery was cut in dogs, pain no longer occurred; that is, if one is willing to accept the criteria for pain in dogs used by these workers.

They believe, therefore, the nerves carrying pain impulses were cut and from those in the case of Dr. Porter's, in which there was a section of the artery, one would not expect pain if this work was valid.

Furthermore, these observers found if they injured heart muscle away from a coronary artery, no evidences of pain appeared in the dog. Of course, the point which was made was that one hesitates to accept *in toto* this work as there are no accurate criteria for pain-production in dogs.

My own position is this: I am willing to concede with Dr. Porter that ischemia of the heart muscle may cause pain. Perhaps, the pain is carried up through the sheath of coronary arteries, perhaps not, but I am unwilling at the present time to throw aside the possibility that pain might be produced in coronary arteries themselves.

I think we owe a great debt to Dr. Porter for the presentation of this beautifully worked case, or these cases rather, and for the first time the very clear and unhesitating presentation of an hypothesis which, if it proves eventually to be correct or not, cannot help crystallizing thought in this matter and thus advancing our knowledge of the subject. (Applause.)

DR. L. WHITTINGTON GORHAM: Since Smith in 1918 first ligated the coronary arteries in dogs and produced these peculiar changes in the T wave, a number of further investigations in a variety of conditions have produced similar changes.

In 1920, Pardee, noting these changes in a clinical case, believed that they were in their progressive form more or less characteristic of cardiac infarction.

I would like to add today another instance where such waves were obtained in experimental work. During the past year in association with Drs. Dresbock and Randall, of our Department of Physiology in Albany, we have been producing experimental rupture of the auricle in dogs, operating upon the dog, clipping off a piece of the auricle, after having clamped it, and in back of the clamp placing a slip node, closing up the node, and four days later pulling the string and causing spontaneous rupture of the auricle to take place, and producing a marked hemopericardium.

In the first slide you see an inverted T wave in the first lead of the dog, but in other respects the electrocardiogram we believe is normal. In the next slide you will see a marked inversion of the T wave in all of these.

We were also able by means of injecting serum and oil into the pericardium in dogs to produce similar changes. You perhaps have noted that in 1929 Rupert and Kuntz reported the production of similar curves in dogs in whom a general anoxemia had been produced by rebreathing of CO<sub>2</sub> until their oxygen tension of the blood was reduced to 50 per cent below normal, also by the use of the drug pertussin, producing a coronary restriction. Similar curves were obtained by the use of the combined method of anoxemia and the drug pertussin, and much more marked curves were obtained.

I feel, therefore, that these changes in the T wave may not be taken in every instance as pathognomonic of coronary infarction, and I suggest that

perhaps in Dr. Porter's case one factor, which was common to both, was the production of hemopericardium. Am I correct?

DR. PORTER: Yes.

DR. GORHAM: In one vessel—in one instance the vessel ligated and in the other the heart muscle sutured.

DR. JAMES JOHNSTON WARING: I would like to report a little incident which has a little bearing on this discussion of the pain in angina.

It happens occasionally to a man who is giving artificial pneumothorax after a long interval to a case on the left side that a needle is put into the heart. This accident happened a few years ago to my associate who was giving a refill to a patient after a very long interval. I think the interval was about four months in a woman who had a very good collapse. Knowing from previous experience that her pleura was quite thick, and after putting in the anesthetic needle, and getting air and feeling he had space, he anticipated he would have plenty of room. He went in rather carelessly, I am afraid, and went down into the ventricle with the result—it was a small needle—and of course the blood rushed up into the needle, and out through into the rubber tube for about three or four inches, as far as the cotton filter that was used in the pneumothorax tube.

The interesting thing was that immediately the needle went into her heart he realized, of course, what he had done, and she realized it. She appreciated it by having an agonizing pain radiating up into the neck and down the left arm, with quite a typical ulnar distribution. Of course, he drew the needle out right away, and the pain passed off very shortly. He gave her a half grain of codeine by hypodermic, and when she was comfortable (he kept her at the office for an hour or more) sent her home, and the next day she returned to the office, got her pneumothorax again, and it has been continued at proper intervals ever since. She has apparently suffered no permanent damage, we are all very glad to say.

I have looked the matter up a little bit since then, and I find, after doing this and talking yesterday with two or three of the men here who give pneumothorax, some of them had the same experience, and in the literature there are quite frequent reports on a ventricular puncture in giving pneumothorax, but I have seen no instance of a description for this typical of anginal pain as occurred in this case. Usually when the needle goes into the heart there is no complaint of very severe pain.

DR. WILLIAM H. ROBESY: Dr. Stone did about 300 cases of pericarditis with effusion during the War, and he told me that he always ran the needle into the heart muscle because one can't tell always whether you are in the pericardial sac or not. If you have a window in the tube naturally you can see when the fluid starts to come out, but almost invariably the heart muscle is punctured.

There was a case at the Brookline Hospital in London where a fatal hemorrhage occurred because a needle was put into the coronary artery.

The reason I speak of this is that in speaking on this subject at a meeting

of the A.M.A. a few years ago Dr. Henry Soule objected very much to my statement that the needle could be put into the heart muscle without harm. I have seen it put in a number of times myself, and as far as I can remember, I have never known of an attack of angina pectoris to occur as a result of that procedure.

DR. PORTER: I want to thank each and every one who participated in the discussion. I will not attempt to prolong it, except to comment upon some of the phases.

I referred to the work of Sutton and Lueth, and I agree with Dr. C. C. Wolferth, if their work is correct, it is rather confusing as far as my hypothesis is concerned. However, there is one rather interesting case I ran into, published by a man in one of the Scandinavian countries, in which an army officer got into a combat with one of his fellow army officers and was stabbed over the heart, and the writer refers to the case as being one in which the knife grazed the surface of the heart and cut the coronary artery with little or no injury to the heart muscle. This patient, as a result of ischemia in the heart muscle, mainly of an anginal character, was a very troublesome case as to symptoms for several days.

It occurred to me, therefore, that this case might be at variance with the case reported by Sutton and Lueth, and that my interpretation of that would be that the heart muscle was not injured and contracted normally in the ischemic area.

In reference to the pericardial cases, that is discussed in the manuscript. We know that pericardial effusions will at times produce these changes. Just from what phenomenon we do not know.

I would like to say, myself, though, that the puncture of the heart in the aspiration of pericardial effusion, I don't believe is one per cent. I believe, myself, that if I punctured the heart muscle in pericardial effusion it is the exception and not the rule. I should like to think so at any rate.