The Syndrome of Relief of Angina Pectoris Following Myocardial Infarction

JACOB D. MATIS, M.D., F.C.C.P. and HARRY A. SOLOMON, M.D.
New York, New York

Although heart disease continues to increase in frequency among the causes of death in the population of the country, and although coronary heart disease constitutes the majority of cardiac disabilities, the prognostic outlook of coronary artery disease continues to improve steadily with time. This has recently been stressed in the medical literature and has even permeated into the lay press. It has been steadily emphasized that modern diagnosis and modern therapy have lessened the mortality of each individual episode of myocardial infarction and that following myocardial infarction, many individuals return to a useful life. It has been the experience of the authors that myocardial infarction occasionally is a blessing in disguise in that it causes the cessation of anginal chest pain which had existed previously. Such a medical paradox can be explained by recently observed patho-physiological mechanisms. This knowledge has been shared by other physicians and cardiologists, but the basis for the syndrome has never been satisfactorily described in the literature.

Anatomic Considerations

The subjective manifestations of acute coronary occlusion indicate extensive sensory receptor innervations of the heart with emerging afferent nerve fibers making widespread communications with a system of efferent neurons.1 The afferent nerve impulses come from the heart through the inferior cardiac nerves, the stellate ganglion, the cardiothoracic nerve, the thoracic sympathetic chain and the rami communicantes from the eighth cervical to the fifth thoracic segment.2 It has also been suggested that afferent impulses from the heart are carried by the vagus nerve.3 Many afferent impulses reach the local segmental areas of the spinal cord and produce reflex muscular spasms of the thorax. Most impulses apparently reach the hypothalamic region where they communicate with all the vegetative nuclei, especially the vagal system, which causes visceral spasms, sweating and other symptoms.

Pathology

The clinical manifestations of acute myocardial infarctions are not solely the result of acute closure of a coronary artery. In some cases, acute myocardial infarction occurs without new narrowing or occlusion, while in other cases, the acute occlusion is neutralized by collateral circulation which had formed as a result of the prior narrowing of the vessel. Thus, acute myocardial infarction is to be regarded as the resultant of two opposing processes; namely, the obstruction to blood flow by oclus-
sive processes and the compensatory development of collateral circulation. Such collateral circulation serves to offset the dire effects of coronary artery narrowing and occlusion.4

At examination of the heart post mortem, only the end result of the obstructive process and compensatory mechanism can be observed. The classical experiments of Blumgart et al 5, 6, 7 have shown that collateral channels are tailored specifically to the narrowing process which evokes them. They do not develop indiscriminately, and time is required for
their development. Of the types of coronary arterial anastomoses which these workers have described, intercoronary anastomoses seem to be most definite and most important in man. At post mortem study, only 16 per cent of normal hearts were found to show such interarterial communications, while such communications were found in 98 per cent of hearts which had suffered coronary occlusion. In addition to these anastomoses, there are extrinsic coronary anastomoses between coronary arteries and vasa vasorum, vessels of precordium, diaphragm, mediastinum and pleura. These anastomoses are of lesser importance.

The collateral channels in diseased hearts are of great functional significance. Despite complete occlusion in some hearts, the potentially infarcted myocardium has been free of fibrosis or other structural abnormality when coronary arterial narrowing progresses gradually. Complete occlusion of one or even three main coronary arteries may be compatible with continued life. The slow development of a rich anastomotic circulation may be responsible for the occasional clinical improvement of patients with angina pectoris. The collateral anastomoses act to lessen the effects of arterial narrowing with arterial occlusion when such occurs. The syndrome to be described as illustrated by the following cases evidently represents the clinical counterpart of this pathological condition.

Case Reports

Case 1: M. K. presented himself on January 3, 1950 when he was 54 years of age. Since December, 1949 he had experienced substernal pain associated with heaviness and aching of the left arm occurring with effort. This sensation was relieved by rest. He noticed that the substernal distress was worse when walking rapidly and also after a large meal. Physical examination was not remarkable and blood pressure was 124/88. X-ray film showed the heart to be of normal size. Wassermann, blood sugar, blood

![ECG images](image)
cholesterol, calcium and phosphorus were normal. Blood urea nitrogen was 17.6 mgm. per cent. Blood count and urinalysis were normal. The electrocardiogram was within normal limits (Figure 1). He was treated with vasodilators to which there was no response. There was an excellent response to nitroglycerin.

On June 15, 1942 he suffered an acute myocardial infarction for which he was treated with the usual conservative measures at home. Figure 2 shows changes of acute posterior-septal wall myocardial infarction. He recovered from the myocardial infarction but soon thereafter developed severe angina. This increased in frequency and intensity so that by the beginning of September, 1952 it occurred at rest as well as with effort. Blood pressure was 120/90 and physical examination was not remarkable.

On September 29, 1952 he suffered another episode of acute coronary thrombosis with myocardial infarction which necessitated hospitalization. His clinical condition was critical and was associated with marked shock as well as congestive heart failure. Figure 3 taken on September 29, 1952 indicates acute posterolateral wall myocardial infarction. Once he recovered from the critical phase, he did quite well, so that by December 31, 1952 he was able to work full-time. Then there was no anginal symptom and he felt quite well. Digitalis was continued. The x-ray film showed a slight increase in the transverse diameter of the heart. When last examined on September 29, 1954, when he was more than 58 years of age, he reported that he felt quite well and was able to work full time. There was no anginal symptom and the physical examination was normal. The blood pressure ranged about 110/60 mm. Hg.

Case 2: H. J. When 50 years of age, this man presented himself on January 10, 1940 for routine examination. This white executive had no cardiac symptom and examination was completely normal. The electrocardiogram was normal (Figure 4). He was next seen on June 18, 1943 when he complained of severe anginal pain which occurred both with effort and at rest. He stated that he had experienced frequent attacks of angina pectoris since using a lawn mower three months previously.

On July 1, 1943 he suffered acute myocardial infarction of the anterior wall. Figure 5 taken July 23, 1943 shows changes of anterior wall myocardial infarction. Following uneventful recovery from this episode, he remained symptomatically well and was able to perform all his work and to take part in all his social activities. He was examined yearly and remained well. The electrocardiogram showed persisting signs of old an-

---

terior wall myocardial infarction. When examined on October 10, 1952 he said that anginal pain had occurred during the four previous months. This pain occurred with effort and also at rest, so he was compelled to limit his physical activities.

On January 12, 1953 he suffered a second episode of acute myocardial infarction which was attended by severe shock, marked hypotension, and congestive heart failure. The electrocardiogram indicated acute antero-lateral wall myocardial infarction (Figure 6 taken January 15, 1953). Following a stormy course, he made a recovery from this episode, but mild congestive heart failure remained. When he was examined on April 29, 1953 he stated that he no longer experienced chest pain with effort or at any other time. He was completely asymptomatic, although examination showed signs of mild congestive heart failure. As of October 4, 1954 he was attending to all his


business and social functions without symptoms. The electrocardiogram taken on September 21, 1953 (Figure 7) showed the residual signs of old anterior wall myocardial infarction.

**Case 3:** A. S. When 52 years of age in 1949 this well developed adult white artist had been experiencing precordial pain on effort for several years. Physical examina-
tion as well as electrocardiographic study were reported as within normal limits (Figure 8). Because of the presence of gall stones, cholecystectomy was performed and it was hoped that this might help the angina pectoris syndrome. Following operation, however, there was no relief from the symptoms of angina pectoris which increased in severity and frequency. By September, 1953 he suffered from severe status anginosus so that he was confined to his room and had not attended to business in several months. Figure 9 is an electrocardiogram taken September 11, 1953. On September 20, 1953 he suffered an episode of acute myocardial infarction complicated by severe shock. He was hospitalized during this acute episode and was in critical condition during the major portion of the hospital stay. When examined on May 19, 1954 he reported that he no longer had any symptom of angina pectoris. He was able to walk without pain, drove his car and was able to attend to his business. His major difficulty at this time was a moderately severe cardiac neurosis which caused him to feel inadequate and insecure. Physical examination disclosed a rough systolic murmur at the apex and at the aortic area, blood pressure was 144/72, and x-ray film showed moderate enlargement of the left ventricle of the heart. The electrocardiogram (Figure 10) revealed evidences of old anterior wall myocardial infarction. The patient continued to be fairly well until the morning of July 2, 1955, when he was awakened from sleep by severe crushing anterior chest pain and died shortly thereafter. Death was thought to be due to a new episode of acute myocardial infarction.

Case 4: M. C. In 1951 this 43 year old salesman complained of precordial pain with effort. The pain necessitated limitation of his physical activities and of the amount of traveling he would normally do in association with his occupation. In the past, he had been treated for gall bladder disease and peptic ulcer. His brother and father had suffered coronary thrombosis early in life. Physical examination, laboratory studies, and study of heart size all were found to be within normal limits. The resting electrocardiogram (Figure 11) as well as the Master two step exercise test were found to be normal. Because of increasing frequency of chest pain with effort, he was well on his way to becoming a complete cardiac invalid when he suddenly suffered an episode of acute coronary thrombosis on August 1, 1951. Figure 12, the electrocardiogram taken on August 2, 1951, shows changes of acute posterior wall myocardial infarction. He was treated with anticoagulants at home and made an uneventful recovery. Within two months, he was able to return to his occupation as a salesman and he reported that he no longer experienced any chest pain with effort. He follows a low-fat, low-cholesterol diet with little change in his weight. He is now able to pursue his full activities and travels freely by rail, automobile, and airplane. Because of increased
endeavor, his present earning capacity is greater than that of any prior year. When examined on September 20, 1954 he appeared well, was adjusted mentally, and was happy to report that he no longer suffered chest pain. The electrocardiogram showed persisting evidence of old posterior wall myocardial infarction (Figure 13).

He neglected to return for a scheduled check-up examination, and upon inquiry it was learned that he had placed himself in the hands of a naturopath. The latter has informed him that he never suffered any cardiac injury and evidently the patient feels well enough to accept this judgment.

Case 5: A. E. This real estate executive was first observed in 1942 when he was 43 years of age. He complained of mild precordial chest pain and slight dyspnea associated with effort. Complete physical and laboratory examinations were normal. The electrocardiogram (Figure 14) taken on May 26, 1942 was normal. While the family history was positive for diabetes, studies of the patient for this disease were negative. He was observed at frequent intervals. His angina became more severe so by the end of 1948 he was forced to curtail some of his business and social activities. Throughout this interval, however, the electrocardiogram remained normal. On May 2, 1949 he suffered acute coronary thrombosis for which he was hospitalized and treated with anticoagulants. Figure 15 is the electrocardiogram taken June 14, 1949 showing changes of acute anterior wall myocardial infarction. On July 12, 1949 he reported that he was completely free of symptoms and that he was able to walk with no chest pain. With the consent of the attending physician he was permitted to return to busi-

![Figure 16](image1)

**FIGURE 16 (Case 6): M. S. December 18, 1951.**

![Figure 17](image2)

**FIGURE 17 (Case 6): M. S. Feb. 20, 1953. FIGURE 18 (Case 6): M. S. Apr. 8, 1953.**
ness and within a month was more active than he had been in the previous seven years. He reported regularly for examination and always stated that he was completely free of chest pain although he had undertaken additional business obligations. He continued with full business activities and was frequently observed. The electrocardiogram continued to show evidence of the old anterior wall infarction and no cardiac enlargement was recognized on fluoroscopy or X-ray film of the chest. On April 3, 1952 while walking, he suddenly collapsed and was found dead upon arrival of medical aid. He had been completely active and without symptoms until this time, some two and one half years following the acute myocardial infarction.

Case 6: S. M. This 44 year old white man presented himself on December 18, 1951 complaining of angina pectoris on effort for two and one half months. The pain had increased steadily in severity. Physical examination as well as determinations of the uric acid, cholesterol, basal metabolic rate, sedimentation rate and complete blood count, were normal. The electrocardiogram was normal (Figure 16). When seen on July 23, 1952 he was suffering from status anginosis so he was unable to attend to his business. Physical examination remained normal and the blood pressure was 125/80. The electrocardiogram remained unchanged. Examination on February 20, 1953 showed no essential change in his condition. He was virtually confined to his home. The electrocardiogram remained normal (Figure 17).

On March 21, 1953 he suffered acute coronary thrombosis for which he was treated at home. Figure 18 is the electrocardiogram taken April 8, 1953 showing evidence of posterior wall myocardial infarction. When examined on May 11, 1953 he was happy to report that he was completely without chest pain and that he had returned to his regular occupation. This entailed using the subway system and he was able to

FIGURE 19 (Case 7): P. W. November 9, 1945.

walk subway stairs without discomfort. He was now more active physically than he had been in the previous two and one half years. He continues to remain well, attends to his business and requires no active therapy.

Case 7: P. W. This 46 year old business man was observed on November 9, 1945 as part of a routine physical examination which as well as the electrocardiogram were reported as normal. Figure 19 is the electrocardiogram taken November 9, 1945. He again presented himself on September 14, 1951 stating that he had recently begun to experience substernal pain with effort and after his large meal. Walking was particularly likely to produce substernal pain. The electrocardiogram taken at this time remained normal. He was seen periodically thereafter and reported that the anginal pain increased in frequency and intensity.

On September 20, 1954, while walking up-grade, he developed intense substernal pain which was not relieved after 30 hours of rest. The diagnosis of acute coronary thrombosis was made clinically and he was hospitalized for a period of four weeks. Figure 20 is the electrocardiogram taken October 4, 1954 showing changes of postero-lateral wall myocardial infarction.

On October 30, 1954 he returned to business and has since been working regularly full-time. When examined on March 21, 1955 he felt perfectly well, did not experience symptoms of angina with effort or with other activity. The electrocardiogram (Figure 21) had returned towards normal.

Comment

While it has been frequently observed that angina pectoris is experienced for the first time after an episode of myocardial infarction, the syndrome which is described illustrates that angina pectoris may be relieved following myocardial infarction. In Cases 1 and 2, the angina pectoris became more intense after the first episode of myocardial infarction, but was relieved after the second. In Case 3, the myocardial infarction afforded relief from angina pectoris which had been present for four years. There also was evidence of increased coronary reserve in that the patient was able to increase his activities greatly. However, death occurred due to a new coronary thrombosis two years later, in spite of his apparent well being. In Case 5 there was relief of chest pain after myocardial infarction. Death occurred some three years later.

Those who experience relief of angina after myocardial infarction evidently have increased functional capacity. Whether this relationship indicates a better life span remains to be studied. It would seem that one could draw a parallel between the cases described which indicate compensatory collateral circulation and the recently proposed surgical procedures which attempt to increase and promote circulation through revascularization of the heart.9

SUMMARY

Cases are reported to illustrate that there may be relief from severe and often intractable angina pectoris following acute myocardial infarction. While the cases described showed varying clinical manifestations and varying localization of cardiac pathology, there was the common experience of relief from chest pain following acute coronary artery occlusion. The likely explanation for the relief is that compensatory collateral circulation had developed during the period when angina pectoris was present.

In addition to the theory of increased collateral circulation following coronary artery narrowing, it has been theorized that transformation of an ischemic myocardial area into scar tissue might prevent the origin of
painful stimuli. The latter theory seems less likely in view of the relatively good functional capacity after myocardial infarction in the patients described.

RESUMEN

Re relatan casos que ilustran el hecho de que puede haber alivio de una angina de pecho a veces intratable y grave al parecer un infarto del miocardio. Si bien los casos descritos mostraron variables manifestaciones clínicas y diversas localizaciones de la patología cardiaca hubo la experiencia común de alivio del dolor del pecho después de la oclusión coronaria. La explicación verosímil es que se desarrolló circulación compensadora colateral durante el periodo en que había angina.

Además de la teoría del aumento de la circulación colateral después del estrechamiento coronario, se ha teorizado sobre la posibilidad de que la transformación de una área isquémica en cicatriz evite la existencia de una zona de estímulo doloroso. Esta teoría parece menos plausible en vista de la capacidad funcional relativamente buena después de infarto miocárdico de los enfermos descritos.

RESUME

L'auteur rapporte des cas montrant qu'un infarctus du myocarde peut apporter un soulagement dans l'angine de poitrine sèvère et souvent incurable. Alors que les cas décrits étaient caractérisés par des manifestations cliniques diverses et des localisations variées d'atteinte cardiaque, on a pu noter généralement, après l'occlusion coronarienne aiguë, un soulagement des douleurs thoraciques antérieures. L'explication vraisemblable en est donnée par le fait que la circulation collatérale compensatrice se développe pendant la période d'angine de poitrine.

Outre la théorie de la circulation collatérale augmentée après l'étranglement de l'artère coronaire, on a pu émettre l'hypothèse que la transformation d'une zone myocardique ischémique en tissu cicatriciel pourrait empêcher l'origine des stimulis douloreux. Cette dernière théorie semble moins vraisemblable si l'on considère la capacité fonctionnelle relativement bonne après un infarctus myocardique chez les malades étudiés.

ZUSAMMENFASSUNG


Zusätzlich zu der Theorie des erhöhten Colateral-Kreislaufes im Anschluss an eine Verengung der Coronar-Arterien wurde theoretisch erwogen,
dass die Umwandlung eines ischaemischen Myocard-Bezirkes in ein Narbengewebe das Auftreten schmerzhafter Reize verhindern könnte. Die letzgenannte Theorie scheint wenig wahrscheinlich im Hinblick auf die relativ gute funktionelle Leistung der beschriebenen Fälle mit Herzmuskelinfarkt.

REFERENCES