Editorial

The Recognition of Angina Pectoris

IN 1772 William Heberden’s classic description of angina pectoris was first published. Heberden, one of England’s outstanding physicians, was also a scholar who possessed "the art of clear and accurate description, so that his vivid portrayal of the symptoms of angina pectoris, recorded in remarkable clarity, stand unchallenged today and is without a doubt one of the most cherished and brilliant masterpieces of the past."1

The following is a verbatim quotation from Heberden’s Commentaries on the History and Care of Diseases as published posthumously in London in 1802 by one of his sons.1

But there is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned more at length. The seat of it, and sense of strangling, and anxiety with which it is attended, may make it not improperly be called angina pectoris.

They who are afflicted with it, are seized while they are walking, (more especially if it be up hill, and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or to continue; but the moment they stand still, all this uneasiness vanishes.

In all other respects, the patients are, at the beginning of this disorder, perfectly well, and in particular have no shortness of breath, from which it is totally different. The pain is sometimes situated in the upper part, sometimes in the middle, sometimes at the bottom of the os sterni, and often more inclined to the left than to the right side. It likewise very frequently extends from the breast to the middle of the left arm. The pulse is, at least sometimes, not disturbed by this pain, as I have had opportunities of observing by feeling the pulse during the paroxysm. Males are most liable to this disease, especially such as have past their fiftieth year.

After it has continued a year or more, it will not cease so instantaneously upon standing still; and it will come on not only when the persons are walking, but when they are lying down, especially if they lie on the left side, and oblige them to rise up out of their beds. In some inveterate cases it has been brought on by the motion of a horse, or a carriage, and even by swallowing, coughing, going to stool, or speaking, or any disturbance of mind.

Such is the most usual appearance of this disease; but some varieties may be met with. Some have been seized while they were standing still, or sitting, also upon first waking out of sleep; and the pain sometimes reaches to the right arm, as well as to the left, and even down to the hands, but this is uncommon: in a very few instances the arm has at the same time been numbed and swelled.

It is of some interest that among Heberden’s friends and colleagues were the brothers John and William Hunter, both physicians of note. The former, a celebrated surgeon, had angina pectoris and contributed to the knowledge of this entity by keeping personal records of his own illness. Apparently he suffered many severe attacks of angina, and his observations and descriptions of the symptoms and signs of the disorder were the first recorded by a physician suffering from this disease. As so frequently quoted he said that his “life was in the hands of any rascal who chose to annoy and tease him.” It was generally agreed that “the violent disagreements he had with his colleagues at St. George’s Hospital hastened his death, which occurred on October 16, 1793, following a meeting of the board of governors.
of the hospital, at which a colleague had directed some disparaging remarks to him."1

According to Willis2 the postmortem examination of Hunter furnished one of the early accounts demonstrating the coexistence of angina pectoris and coronary sclerosis.

Let us now turn to a more recent description of angina pectoris as published in 1953 in the *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Blood Vessels.*2 We find the following remarks about the "anginal syndrome," more commonly known as angina pectoris:

In this syndrome the major symptom is thoracic pain, which is precipitated usually by effort but sometimes by excitement, a heavy meal or exposure to cold. The pain is usually substernal or just to the left of the sternum. Occasionally the pain is epigastric and in rare instances it may be localized in the neck or the left arm or shoulder.

There is a tendency for the pain to radiate, most frequently to the left shoulder and arm and occasionally to the fingers. Less frequently it may radiate to the neck, jaw and teeth, to the back, upper abdomen, or to the right shoulder and arm. At times the pain will start at one of these points before focusing on the anterior surface of the chest.

The intensity varies from a slight sense of heaviness to a severe crushing pain. Since the precipitating cause is commonly physical exertion, rest usually causes the pain to subside. The length of the episode, therefore, is relatively short. Occasionally an attack may come on while the patient is at rest or even when asleep.

The pain is often accompanied by a sense of choking or inability to breathe which is also relieved by rest. The patient will often complain of flatulence as well. If the attack is not relieved by rest or a nitrite and lasts for an hour or more, and especially if it is accompanied by circulatory collapse, myocardial infarction should be strongly suspected. Occasionally the pain of myocardial infarction may be identical with the pain of the anginal syndrome. The associated symptomatology and the subsequent course will determine the diagnosis.

It seems quite obvious after reviewing these quotations written about 200 years apart that the clinical features of angina pectoris as first described by Heberden are essentially the same as those mentioned in the "Criteria." In other words there has been general agreement for many years as to the nature of this symptom-complex. There is still considerable difference of opinion, however, as to the mechanism that initiates the syndrome. It is thought by many that angina pectoris represents an abnormal physiologic, and possibly chemical, mechanism rather than a disease entity. It therefore is not interpreted by them as being synonymous with coronary arteriosclerosis. The underlying disturbance is probably one of anoxia of the myocardium caused by transient inadequate blood flow to the heart muscle fibers. Needless to say other explanations are also entertained.

As based on the clinical and pathologic findings in many cases of angina pectoris, the majority of patients with this disorder have some underlying cardiac ailment, most often disease of the coronary arteries. It also occurs, however, in patients with syphilitic aortitis complicated by aortic insufficiency or by coronary ostial stenosis, as well as in patients with rheumatic heart disease, especially in the presence of aortic valvular deformities, i.e., stenosis or insufficiency. Congenital anomalies such as patent ductus arteriosus may occasionally be associated with angina pectoris. It is also known to occur in hyperthyroidism, anemia, and in a variety of other disorders, some of organic nature, others of functional origin.

The recognition of angina pectoris is dependent primarily on the correct interpretation of a patient’s symptomatology. The latter in turn is obtained by a careful and detailed history, taken not by a nurse or secretary, but by the examining physician. Physical signs may be completely normal, including normal heart size, normal heart sounds, a normal sinus rhythm, and normal blood pressure determinations. White3 stated that in about one fourth of all cases of angina pectoris examination fails to reveal abnormality of the heart. Of most importance and frequently overlooked is the fact that a 12-lead electrocardiogram may be normal. This is so in a high proportion, some 20 to 30 per cent, of patients with angina pectoris. In a series of over 400 patients with angina pectoris due to coronary sclerosis, electrocardiograms were normal in some 58 per cent.4 Mainly because of this fact many physicians are misled and therefore in con-
sequence many patients with angina pectoris are given erroneous negative diagnoses. Some of these patients are informed that they are in normal health or that they are neurotic or are given a variety of other explanations for their complaints.

The failure of some physicians to obtain a thorough history in patients with angina pectoris is of great concern. They seem to rely mainly on electrocardiographic data recorded after exercise tests of varying degrees of exertion in order to determine whether or not a patient has the anginal syndrome. Occasionally myocardial infarction is precipitated by exercise electrocardiogram tests, although the number of such reports in the literature is small.\(^5\)\(^6\) This may be due to a natural reluctance to report a deleterious effect following a test of any kind. There are also known instances of fatal attacks of myocardial infarction that developed during or immediately after exercise in patients who were known to have had angina pectoris but were nevertheless submitted to such a test.\(^7\)

Patients with so-called atypical angina pectoris and known to have normal resting electrocardiograms have developed signs of myocardial injury including infarction in the electrocardiogram following an exercise test. This has occurred even in instances in which the exercise was discontinued promptly after the patient experienced chest pain. The author recently saw 2 such cases in consultation.

Master,\(^8\) whose exercise tests are employed very frequently, has never had an accident during the performance of many thousands of "two-step" exercise tests. He has stated that accidents following tests have been reported in the literature but that "in these cases either the test undertaken was not the Master test, or it was contraindicated because the resting electrocardiogram was abnormal and/or the patient was obviously sick." He states that if it is insisted that the resting electrocardiogram be normal before exertion is attempted, added assurance is thus obtained.

A recent study\(^9\) of 250 patients with definite coronary disease that included the use of the "two-step exercise test," made by Master and his associates, revealed that there were those who had a "classic anginal syndrome" as well as patients who had had a previous coronary occlusion and presented abnormal electrocardiograms indicative of previous infarction. Nevertheless, Master\(^9\) is of the opinion that "if his simple directions are followed, no accidents will occur except possibly by sheer coincidence.''

An exercise electrocardiogram test is indicated in only a few patients, since an accurate diagnosis of angina pectoris can practically always be made by means of a careful interrogation. Sometimes a patient needs to be seen on 2 or more visits if the initial history is vague or atypical for an anginal syndrome. Such a patient should be instructed to observe more closely the characteristics of his complaint and the circumstances under which it appears as well as its response to rest and to nitroglycerin. In those few instances in which, despite careful questioning, a diagnostic history cannot be obtained or if objective evidence of underlying coronary disease is essential because of insurance or medicolegal reasons, then an exercise electrocardiogram test may be of some help. An electrocardiogram taken at rest, however, should always be recorded and recognized to be within normal limits before submitting the patient to exercise.

A great variety of ailments associated with chest discomfort are not infrequently listed as closely resembling the anginal syndrome and, therefore, in need of being differentiated from it. If the distinct clinical features of angina pectoris are kept in mind, however, this need of differentiation from other conditions will rarely be indicated. In practically every instance the typical history is sufficient to distinguish the anginal syndrome from those diseases simulating it. Unfortunately, it is very time-consuming for the average practicing physician to obtain a careful clinical history.

In closing this editorial I think it most appropriate to quote the introduction to an address on Cardiac Symptoms presented at the 1951 meeting of the American College of
Physicians by Sir John Parkinson\textsuperscript{10} of England.

The subject of symptomatology will always retain its importance in medicine because symptoms form the first contact between patient and doctor. It is the voice of nature, and when a patient complains he enters our world and we recognize a human need. For convenience I shall apply the term “symptoms” to subjective sensations of which the patient complains. While everyone agrees on their value in approaching a diagnosis, symptoms have been moved to the background by current interest in signs and in scientific technic. But if we apportion too little time for eliciting symptoms, we shall suffer in our diagnosis. Besides, knowledge acquired by the art of listening and of questioning has value far beyond a lead towards the diagnosis. A doctor learns what kind of human being faces him, and what reaction he is making to his malady.

These remarks, by another outstanding physician in England’s great medical heritage, are a fitting counterpart to those of Heberden as quoted verbatim at the beginning of this editorial.

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\section*{Neuroses of the Heart}

\underline{Angina Pectoris}

In fatal cases of angina the coronary arteries are almost invariably diseased, either in their main division, or there is chronic endarteritis with great narrowing of the oriﬁces at the root of the aorta. Experimentally, occlusion of the coronary arteries produces slowing of the heart’s action, gradual dilatation, and death within a very few minutes. Cohnheim has shown that in the dog ligation of one of the large coronary branches produces within a minute a condition of arrhythmia, and within two minutes the heart ceases in diastole. These experiments, however, do not throw much light upon the etiology of angina pectoris. Extreme sclerosis of the coronary arteries is common, and a large majority of the cases present no symptoms of angina. Even in the cases of sudden death due to blocking of an artery, particularly the anterior branch of the coronary artery, there is usually no great pain either before or during the attack. The lesions of the nerves described by Lanceleroux, Hadden, and others cannot yet be correlated satisfactorily with the symptoms of true angina. Various forms of true angina have been recognized, but the differences, in the majority of instances, are not sufficiently marked to permit a separation.—\textbf{William Osler, M.D.} \textit{The Principles and Practice of Medicine}. New York, D. Appleton & Company, 1893, p. 656.

\section*{References}

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Circulation. 1960;21:1061-1064
doi: 10.1161/01.CIR.21.6.1061

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/21/6/1061.citation

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