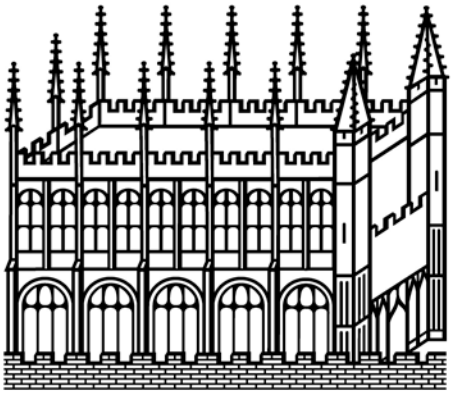


Bodleian Libraries

UNIVERSITY OF OXFORD

This book is part of the collection held by the Bodleian Libraries and scanned by Google, Inc. for the Google Books Library Project.

For more information see:

http://www.bodleian.ox.ac.uk/dbooks

This work is licensed under a Creative Commons Attribution-NonCommercial-ShareAlike 2.0 UK: England & Wales (CC BY-NC-SA 2.0) licence.

ANGINA PECTORIS

HARLOW BROOKS

C

1524 f. 8

HARPER'S
MEDICAL MONOGRAPHS

ANGINA PECTORIS

Harper's Medical Monographs

PRACTICAL MONOGRAPHS FOR THE PRACTITIONER

ADVISORY EDITORS

W. C. ALVAREZ, Rochester, Minn.
G. E. BREWER, New York
G. W. CRILE, Cleveland
N. B. FOSTER, New York
L. HAMMAN, Baltimore
A. T. OSGOOD, New York
E. A. PARK, Baltimore
H. C. TAYLOR, New York
G. B. WEBB, Colorado Springs
U. J. WILE, Ann Arbor

ANGINA PECTORIS

By HARLOW BROOKS, M.D., of New York

There has previously appeared in this series:
SYPHILIS, *by* Charles C. Dennie, M.D., *Kansas City*

Volumes on the following subjects are now in the course of preparation and will be published in the near future. They will be uniform with this work.

TUBERCULOSIS *by* Allen K. Krause, *Baltimore*
TUMORS OF THE BREAST *by* William C. White, *New York*
CARE OF TRAUMATIC AND INDUSTRIAL CONDITIONS *by* Clay Ray Murray, *New York*
ASTHMA AND HAY FEVER *by* Robert A. Cooke, *New York*
DIABETES MELLITUS *by* W. D. Sansum, *Santa Barbara*
THE PSYCHONEUROSES *by* Austin F. Riggs, *Stockbridge*

RALPH G. STILLMAN
Managing Editor
8 West 16th Street, New York, N. Y.

Harper's Medical Monographs

ANGINA PECTORIS

By

HARLOW BROOKS, M.D.

EMERITUS PROFESSOR OF CLINICAL MEDICINE
NEW YORK UNIVERSITY

VISITING PHYSICIAN, CITY HOSPITAL; CONSULTING PHYSICIAN, FIFTH AVENUE, FRENCH, POLYCLINIC, MONTEFIORE, NORWEGIAN AND BETH ISRAEL HOSPITALS, NEW YORK CITY; COLONEL, MEDICAL RESERVE, U. S. A.;
ETC.

HARPER & BROTHERS PUBLISHERS
New York *and* London
MCMXXIX

ANGINA PECTORIS
COPYRIGHT, 1929, BY HARPER & BROTHERS
PRINTED IN THE UNITED STATES OF AMERICA
FIRST EDITION
A-D

CONTENTS

Chapter		Page
	PREFACE	vii
I.	HISTORICAL	1
II.	OCCURRENCE AND GENERAL DESCRIPTION	5
III.	THE SYMPTOMS	15
IV.	DEATH AND COMPLICATIONS IN ANGINA PECTORIS	34
V.	PATHOLOGY	47
VI.	PATHOLOGICAL PHYSIOLOGY	63
VII.	PSEUDO ANGINA PECTORIS	67
VIII.	TOXIC ANGINA	81
IX.	DIAGNOSIS	91
X.	ELECTROCARDIOGRAPHY	94
XI.	PROPHYLACTIC TREATMENT	98
XII.	SPECIFIC TREATMENT	107
XIII.	GENERAL TREATMENT	120
XIV.	TREATMENT OF THE ATTACK	136
XV.	SURGICAL TREATMENT	149
XVI.	PROGNOSIS	160
	INDEX	163

PREFACE

There is probably a greater interest today in the subject of angina pectoris than ever before in the history of medicine. While in some respects very little advance has been made in its understanding and while today there remain several phases of its characteristics which are still as vague and debatable as when the condition was first recognized, the interest in the subject by the profession has tremendously increased, and beyond any doubt our present-day diagnosis and treatment have very much improved.

Either the condition occurs much more frequently here in America than formerly, perhaps as a result of the demands of modern life, or the increased interest and understanding of the profession has caused it to be more commonly recognized.

The great urge animated by modern cardiology has not resulted, in so far as this subject is concerned, in any striking contributions to this problem which mark great advance since the original communication of Heberden, except in one respect, and that is as regards treatment, but after all this

should be considered the most important field for all medical study.

Today the treatment of angina pectoris has advanced to such a stage that except in those instances which terminate soon after its recognition, life may be prolonged and comfort enhanced very materially. The economic saving now realized by eliminating the complete withdrawal of subjects of the disorder from productive life is another advance of real value.

Many physicians are still, however, unaware of what may be done for the sufferer from this syndrome. To some, as to most of the laity, the diagnosis of angina pectoris is tantamount to a death sentence.

The chief excuse for this little volume is that it is an attempt to present the practical phases of the complex in a sufficiently clear manner to be understood by all, and particularly to stress the subject of treatment. It is not the treatment of the condition, but rather that of the individuals who suffer from it which chiefly interests the writer.

HARLOW BROOKS.

New York, December, 1928

ANGINA PECTORIS

"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 here at length. The seat of it and sense of strangling and anxiety with which it is attended, may make it to not improperly be called Angina Pectoris."

—HEBERDEN, 1768

CHAPTER I

HISTORICAL

ANGINA PECTORIS presents a most striking and dramatic clinical picture. It is highly improbable that so impressive and serious a complex could have escaped the observation and study of many physicians long before the time of Heberden. In reviewing old medical literature as far back as we may go one finds frequent references to conditions which must have been instances of angina pectoris, but up to the remarkable study of Heberden, published in 1768, no observer had been able so to collate, classify and identify the clinical aspects of the syndrome as to make it possible to group these cases under a single head.

There are few instances in medical literature which equal in clarity, in detail of keen observation and in practical accuracy of application and clear-cut conclusions the original publication of Heberden. It is one of the outstanding pillars of historical medical literature. It stands even today as an article which requires but little modification or elaboration in so far as it pretended to go. It was published at a time when the observation of cases

was at its highest peak of development, and the writer knows of no recent publication concerning disease which more clearly portrays its purpose. At that time comparatively little attention was paid to the subject of pathological anatomy, except by the Hunters and this article is in no way different in this respect from most others of the period. It is almost humiliating, however, to confess that we have done very little even with our modern methods of research to add materially even in this respect. As a clinical description of the condition, Heberden's article today stands as the most satisfactory and graphic delineation of the clinical picture which has yet been written. The student of the subject today, irrespective of the length or character of his training, can probably derive no more clear concept of the syndrome than is detailed in this remarkable paper. Another astonishing feature of this communication lies in its completeness and in its accuracy of observation. There is little to be found in any modern article except as dealing with the etiology, pathology and physiology and treatment of the subject which is so complete and accurate as this study. There is also little said by Heberden which is not today recognized as equally true as it may have been at the time it was written. Almost nothing symptomatically can be added now which Heberden had not recognized and stressed.

I know of no other article on the subject which is today so complete in so far as clinical observation goes, nor so valuable for the student of the condition, no matter how extensive his experience or observation may have been.

It properly remained for Edward Jenner to point out in 1799 the frequent association of the syndrome with disease of the coronary arteries. Jenner's observations, subsequently confirmed by Parry, have also stood the test of time and no student of the condition today can controvert much that this shrewd student stated, though naturally our knowledge of the pathology has been greatly broadened since the report of this great master of research. As in Jenner's discovery of the elemental principles of immunity, in his monumental article on vaccination, his work on the pathology of angina pectoris remains largely unshaken and entirely modern in its applications and deductions. This article stands as the second pillar or epoch in our knowledge of the syndrome.

In 1821 Reeder first drew attention to the distinction and differentiation of the angina pectoris of Heberden and Jenner from certain closely simulating conditions, then named pseudo or "false" angina. In spite of this and many subsequent studies the differentiation between true, that is, dangerous angina and that form which simulates it,

but in the notable exception that it is rarely if ever fatal, remains as difficult a clinical problem today as then.

The subject still persists as one of the most fascinating problems of medicine and the interest in it is indicated by the fact that most of the great names in medicine subsequent to the time of Heberden have at one time or another engaged themselves in this study. Outstanding among these are the names of Charcot, Lancereaux, Huchard, Virchow, Eulenberg, Vaquez, Von Neusser, Cohnheim, Ortner, Krehl, Nothnagel, Osler and nearly all the modern leaders of internal medicine and pathology. The students of pure physiology in all ages since Heberden have likewise often concerned themselves with the problem in an intensive way.

The third and final outstanding contribution to our knowledge of the condition was furnished by Lauder Brunton in his discovery of the value of the nitrites in this as in other conditions. The three great and epoch marking contributions to our knowledge of the condition are, then, those of Heberden, Jenner and Lauder Brunton.

CHAPTER II

OCCURRENCE AND GENERAL DESCRIPTION

ANGINA PECTORIS has been largely classed in the past as a disease of relative rarity. This no longer holds true, in the opinion of the writer, certainly not in urban communities. There is much to suggest that the relative increase in the occurrence of the disease as it now appears is due to the increased stress of modern life. Formerly, as stated by Huchard, the disease was more or less limited to those occupations and professions which entailed mental and nervous stress. The complex was then more or less limited, as one may say, to the "intelligensia." It was of infrequent appearance in the hospital classes. In connection with this fact it must be recalled, however, that angina pectoris is a condition in which there are often long periods of immunity from attack, and there are long spaces of time in which patients would object to confinement to the hospital. It is not a hospital disease, in other words, but as a rule one in which ambulatory invalidism is the characteristic.

During the past ten years, however, I find it frequently present on my service at the City Hospital,

the clientele of which is entirely derived from the dependent classes of New York City.

The analysis of the writer's series of 350 consecutive cases shows 75 were engaged in domestic occupations, such as housekeeping, gardening and the like. Nineteen cases only were employed in work entailing physical stress, chiefly such as farmers, longshoremen, soldiers, sailors and the like. Thirty-one men belonged to the learned professions, medicine, law, the clergy, teachers and the like. Fifteen cases occurred among persons engaged in the artistic callings, painters, sculptors, jewelers, decorators, architects and designers. But 8 were engaged in financial endeavors, 3 were engineers and 50 patients were engaged in executive positions. Ninety-three were business men.

Naturally such statistics are very largely influenced by the environment in which a physician lives and by the character of his practice, but it appears to the writer that a relatively high percentage of cases are appearing among business men who are now as a class living lives of much greater stress than formerly, while the demands of life in general entail without exception much more nervous and emotional stress than was formerly the case and the obligatory demand presented to every person, physical or brain worker, is for more speed, greater production and greater

stress in every relation. Conditions of life, at least in America, have recently become more healthful in every class of society, and relatively particularly so in the so-called laborious occupations, yet the death rate from cardio-vascular disease is constantly on the increase, and this is nowhere more evident than in the occurrence of angina pectoris. Alexander Lambert (Sudden Death, Cardiac Pain and Angina Pectoris, p. 1) states that in New York City, in 1924, there was a total of 71,252 deaths. Of these 15,826, or 22%, were due to some form of cardiac disease; of the cardiac deaths 662, or 4%, were recorded as due to acute endocarditis, acute myocarditis and angina pectoris; 410, or 2.5% were due to angina pectoris alone.

Heretofore there has been a marked disparity of occurrence of this complex among women as compared to men. Thus Osler found but 44 women in his analysis of 200 cases. All writers on the subject, from Heberden down, have pointed out that women have possessed a definite immunity against the syndrome. This difference in sex occurrence is, however, less striking in my series. Though my practice is about equally divided among women and men, of my group of 350 cases, 88 occurred in women. It is the writer's impression that the complex is of growing appearance among women in this country because of the increased stress of life

to which they are obligated by the modern vocations and avocations of women. Paulin (Trs. Assn. Am. Phys., Vol. XXXIX, 1927) points out the rarity of the condition in the negro, a statement confirmed by Thayer and by Longcope.

The writer wishes particularly to stress the relation which emotional tension bears to the evolution of the complex. It is probable that this is due not only to the effects of stress of this character on the development of arterial disease, but also because a life of this character sensitizes the nervous mechanism. Certainly the nervous elements have much to do with the development of the symptomatic evidences of the complex, and they also determine, probably, the appearance in many instances. It must not be forgotten that all the lesions of the complex may and often do exist without symptomatology, and that the symptomatology in itself is probably brought about through a quite distinct mechanism. It is the lesion which kills, but the nervous defects which develop most of the symptoms. Albutt (London Lancet, May 5, 1923) says that the coronary arteries and the myocardium have nothing to do with the pain, but much to do with the mortality.

The older writers on the subject, those who wrote and observed before the days when it was customary or possible to resort to accurate methods for

the determination of arterial tension have in the main emphasized the influence of hypertension in the evolution and appearance of the complex. Now that we are able to determine blood pressure accurately, we find that the condition is not definitely associated with hypertension. So generally has the profession accepted the dictum that hypertension is a factor in angina pectoris that the writer finds it at times very difficult to persuade colleagues that the absence of hypertension is no argument against the positive diagnosis of angina pectoris. In the author's series of 350 cases, however, it is found that 200 of the cases showed normal tension, 54 showed hypotension, and only 96 cases showed hypertension. As a rule during the paroxysm the blood pressure is distinctly depressed relatively to the ordinary tension of that individual. Incidentally it may also be remarked that the blood pressure in arteriosclerosis is quite as frequently depressed as elevated. Most commonly it is relatively normal.

All observers have agreed that age is an important factor in the occurrence of angina pectoris. The disease, very rarely, does occur in infancy and youth, notably as has been already remarked, after the infectious diseases, scarlet fever, influenza and rheumatic fever. Heberden reports in his original paper the case of a boy of two years. In these conditions as a rule it will be found that the

lesion present in the heart is a myocarditis or myocardial degeneration, but there are exceptions.

S. C. Jamison and C. H. Hauser (Jour. A. M. A., Oct. 31, 1925) report a case in a boy of eighteen. Post-mortem examination showed in brief the following: The coronaries were tortuous and rigid, healed infarcts were found in the intraventricular septum, anterior wall of left ventricle and the capillary muscles. Organized thrombosis of the right coronary, near its origin, was present. All the blood vessels showed obliterative endarteritis. A small liver abscess was found but no microscopic evidence of syphilis or rheumatism. The cause was supposed by the authors to be due to "A diffusable toxin acting directly through the circulation." Marked proliferation of the endothelium was present in the blood vessels.

In the author's series but one case occurred between the ages of twenty and thirty; 72 cases between forty and fifty; 130 between fifty and sixty; 101 between sixty and seventy; 24 between seventy and eighty; and but 1 between eighty and ninety. In this relation the student must recall that the latter periods of life are as a rule accompanied by lessened physical and emotional stress while in the years of greatest appearance the emotional stresses of life are at their maximum, and physical stresses are decidedly on the wane; nonetheless, as

every physician knows, when physical stresses are imposed during these unpropitious years the complex in some of its gravest types appears with shocking frequency. The old man must learn that he may not with impunity continue the physical activities, normal and beneficial to youth.

Mention has already been made of the occurrence of angina in certain disease conditions. This is notably seen in nephritis, gout, diabetes, and in arteriosclerotic conditions in general. Given age plus the acute or chronic infections and we have also a groundwork highly provocative of the complex.

Heredity plays a very important rôle in the occurrence of angina pectoris. Every practitioner of wide experience is certain to have on his lists families in which this fact is manifest. There are several factors probably concerned in this feature of occurrence. First to be considered is the well-known hereditary tendency toward cardiovascular disease in general and toward arteriosclerosis in particular. Next we must recognize the definite familial inheritance of emotional tendencies and of nervous temperament. The author considers it important that the physician should recognize this fact, for thereby he may early adopt measures in young persons born in anginal families and so direct their habits of diet, emotional control and occupational and recreational selection as to minimize the tend-

ency toward angina pectoris. Thus should preventive medicine also enter into our consideration of angina pectoris.

Another factor which is apparently highly determinative of the occurrence of angina pectoris includes nervous and emotional elements. These have already been mentioned and it is certain that they will again be frequently referred to in the course of this text because of the dominant rôle which they undoubtedly play in the occurrence of the disease. Not only is the individual attack in very considerable part precipitated by emotional states but also it is almost exclusively in persons of an emotional type that the disease most frequently develops. Prolonged emotional stress is frequently a forerunner of the initial paroxysm as well as a certain predisposing factor. The early students of the disease almost without exception mention this as a fact and more often than present writers they pointed out the fact that it was in the nerve-straining and emotional occupations that the syndrome developed with greatest frequency. Lack of emotional control is a part of this predisposing element. It is customarily noted by intelligent sufferers from the condition that when self-control is practiced the attacks may be diminished and minimized. It is said of Sir Astly Cooper that he, knowing himself to be a subject of the angina, stated that

his life was at the mercy of any rascal who called him a fool. Disputatious and opinionated persons are more commonly seized by the disease than those of bland and placid temperament. That this tendency is nonetheless associated with the tensive changes which are supposed to have to do with explosions of temper does not as we have shown specify the factor which either develops the condition or determines the immediate seizure. It is in all probability due to vaso-motor factors concerning vascular supply of a far more intricate mechanism.

Not only do recurring disagreeable emotional stresses act as excitors of angina pectoris but also pleasurable and agreeable experiences as well. The author knows of several instances in intensely appreciative musicians who are unable to listen to favorite compositions because of the precipitation of an attack on such occasions. Fortunately, however, music and other artistic experiences have a quite contrary effect on most sufferers from angina, so definitely so that agreeable music may be utilized as an avocation or means of treatment in suitable instances.

Sudden surprise, emotional shocks of either type precipitate attacks and frequent recurrences undoubtedly determine the development of the chronic condition. Sex excitement, whether satisfied or merely stimulative, is a highly productive factor

and most physicians can recall instances in which occurrences of this type have resulted in immediate death in anginoid patients. This fact must obviously be recognized in the treatment of cases.

The author has attempted to determine in so far as is possible whether diet plays any specific rôle in the occurrence of angina. Of course in established cases known to be caused by gout, by fatty changes in the heart and in nephritis, diet may play an important rôle, but in the evolution of its pathology rather than in the precipitation of the attacks. The increased effort necessary in fat individuals subject to anginal attacks may be also a factor of this sort, but except as diet may contribute to the development of the pathology of the condition, the writer does not feel that it in itself is a determinative factor in occurrence. Familial tendencies toward overweight or toward endocrine states may also possibly be influenced by diet.

CHAPTER III

THE SYMPTOMS

ANGINA PECTORIS is essentially a symptomatic picture, not a pathological or even a clinical entity, hence the importance of the symptomatic evidences of the complex, and while the basis of symptom production is varied to a considerable extent the symptoms of angina are of the most dramatic and striking character possible.

The most constant and most important symptom of angina pectoris is *Pain.* The pain in angina is indeed more or less characteristic of the syndrome and it and its variants are the most constant diagnostic phase of the picture.

The author here wishes to quote extensively from the original description of Heberden which he feels should be properly received as the standard description of the complex:

"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 con-

tinue; but the moment they stand still all this uneasiness vanishes.

"In all other respects the patients are, at the beginning of the 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 extends very frequently from the breast to the middle of the arm. The pulse is, at least sometimes, not disturbed by the pain, as I have had opportunities of observing by feeling the pulse during the paroxysm. Males are more liable to this disease, especially such as have passed their fiftieth year. After it has continued for a year or more, it will not cease as instantaneously upon standing still, and it will come on not only when persons are walking, but when they are lying down, especially if they lie on the left side, and oblige them to rise out of their beds. In some inveterate cases it has been brought on by the motion of a horse or carriage and even by swallowing, coughing, going to stool, speaking, or any disturbance of mind.

"Such is the 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 some-

times reaches down the right arm as well as the left and even down to the hands, but this is uncommon; in a very few persons the arm has at the same time been numbed and swelled. In one or two persons the pain lasted some hours or even days, but this has happened when the complaint has been of long standing and thoroughly rooted in the constitution; once only, the very first attack continued the whole night."

The pain in angina is ordinarily severe in degree, but the intensity varies very much, not only in different cases, but also in different attacks of the same case. The frequency of the attacks bears no relationship to the severity of pain. Attacks are ordinarily graded by the patient by the severity of the pain suffered.

There are no precise location points for the pain, except that in the same case the pain ordinarily at its outset originates in about the same spot. The most frequent point of origin of the pain is posterior to the manubrium sterni. It is almost invariably described by the patient as deeply located and as clutching or squeezing in character. Frequently the symptom of pain passes through a definite cycle, beginning as a dull heavy ache deep behind the manubrium or at the ensiform, or at times located in the gladiolus passing upward, increasing in severity to an agonizing clutching pain which is cus-

tomarily reflected in some usual pathway and subsiding in the same gradual manner as it usually develops. Occasionally the onset is sudden and is described as like a severe blow in the mid-chest. At times the pain is very slight and is little more than a mere uneasiness in the sternal region. Occasionally the pain appears to originate in the epigastrium and this is particularly true in those instances in which it seems to be induced by gastric stimuli.

Nausea frequently occurs with the pain, but actual vomiting, even in these instances is not common, unless it be induced as it occasionally is by an effort on the part of the patient, not infrequently successfully relieving the agony. Very commonly during the pain periods there is a local hyperesthesia of the skin covering the painful area and this hyperesthesia may persist for some time after the severity of the pain has subsided. Percussion or even auscultation, if done with much force during the occurrence of the pain is very distressing for the patient. In some cases the patient defines the pain as of a bursting character or of a sense of swelling, sometimes with lancinating or tearing sensations. The duration of the pain varies tremendously, even in the same case. Marked salivation is a frequent accompaniment of the pain.

In more cases than not the pain is reflected and

as a rule the reflection in any individual case is along certain definite paths. Occasionally it definitely follows certain nerve and vascular trunks, but in other instances this is not so apparent. Undoubtedly the most frequent path of reflection is into the left shoulder and down the arm. Occasionally the reflection is into the right instead of the left arm and it is stated that in instances of dextrocardia this is the customary path. It is notable that in those instances in which the usual reflection is into the arm or shoulder the basic pathology is usually located in the coronary vessels. Cases which are apparently based chiefly on aortic disease are more likely to show a reflection into the neck and up into the jaw. Not infrequently the teeth pain in a most distressing way, and contractures of the neck muscles take place during the paroxysm.

In other cases the pain is described as of a fan-like distribution proceeding outward from the precordial area like a bursting bomb, sometimes reflected downward into the abdomen. These manners of reflection are rather frequently associated with a primary myocardial pathology. In certain cases and at certain times in any case the reflection may take on variant forms. Reflection into the ovary or testis is common, reflection into the post-occipital region is also not infrequent.

In some cases a reversal of reflection appears to

take place, and the attack may be introduced at the periphery and proceed toward the cardiac area. In one of the author's cases, in which a chronic appendicitis was long known to have existed the anginal attacks, up to the time of exitus after several years, invariably originated in the right lower abdominal quadrant, extending up into the right shoulder and thence into the precordium. Similarly in some patients the onset of the paroxysm is with a tingling and numb sensation in the fingers of the upper extremity, thence to the precordium.

Associated or concomitant spasms of pain in widely separated areas are also not infrequent. In 10 of the author's cases intermittent claudication was present and the associated occurrence of abdominal anginas is very frequent. In some instances the entire location of pain may be extracardiac, as in the abdomen, and yet the lesion as seen at post mortem is found located in the heart. In three cases studied by the writer temporary attacks of glycosuria occurred coincident with severe cramp-like pains in the pancreatic region; attacks almost impossible to distinguish from those of acute pancreatic disease or from the passage of pancreatic calculi. Another frequent location of aberrant anginal pain is in the region of the gall bladder. More than once the writer has known of cases being operated upon under the diagnosis of gall bladder or gall passage dis-

ease when the real lesion was in the heart. Within one recent week in a service adjoining that of the author, two cases entered with the diagnosis of acute gall bladder disease. Fortunately because of the desperate condition of the patients the surgeon in both instances refused to operate, and at autopsy, in both, coronary thrombosis was discovered. The history of previous similar attacks definitely fixed the condition in both instances as an angina pectoris, especially as in both instances the gall bladder and its ducts were found to be in perfectly normal condition. Eleven cases in which gall bladder disease was closely simulated appear in the author's series.

In a beautifully descriptive and carefully considered chapter on this subject, Alexander Lambert (Tice's Practice of Medicine, Vol. X) shows how it is possible for a stimulus originating in practically any part of the vegetative or the regular nervous tracts to be developed along recognized nerve tracts and either traced to the heart or from the heart to any of these areas. "It is notable that the transmission is along the course of the blood vessels in most instances, and it is notable also that irritable foci, as a chronic appendicitis, an inflamed gall bladder or any other focus similarly involved, may thus act as the focus of inspiration for the development of an anginal attack. The frequent association of anginal attacks with lesions or irritations of the

gastro-intestinal tract is particularly notable, and many patients have discovered for themselves the fact that distention of the gastro-intestinal tract is highly productive of the precipitation of attacks of angina." Von Neusser says that all abdominal types of angina must be looked upon as radiating along the course of the pneumogastric and the phrenic nerves.

Although the course of pain reflection is usually chiefly along well recognized sympathetic tracts, as particularly mentioned by Lambert, this is by no means exclusively so. In association with Dr. Marcus Rothschild the writer had the opportunity of studying a prolonged case of coronary thrombosis which developed in a case of angina of long standing, in which a typical herpes zoster developed along the course of the musculospiral and the ulnar nerve trunks. Robert Wertheimer (Wein. klin. Wochenschr, *40*:623-624) reports a case of herpes zoster in a sufferer from angina pectoris of eight years standing. The patient was a male, seventy-five years of age. Herpes blisters encircled the left half of the thorax. Wertheimer assumes that the chronic irritation of the spinal segments, a result of the longstanding angina so reduced the tissue resistance that the infectious virus of the herpes zoster was able to act. Wertheimer cites in the same article a case reported by Ortner. It occurred

in the distribution of the left ulnar nerve. The autopsy showed intense sclerosis of the coronary vessels without aortic affection. While herpes zoster is apparently rare in angina pectoris, areas of hyperesthesia are very common in the areas of reflection and other trophic manifestations appear sufficiently frequently to indicate the profound effect which these attacks have upon the tissues. The author has seen true non-suppurative mastitis follow persistent attacks in women and both Osler and Huchard cite instances in which the pain was reflected to the testis and in which attacks were followed by tenderness, pain and swelling of the testis.

As a rule the pain in anginal seizures is so severe that the patient presents a typical clinical picture while the pain is in progress. This is so dramatic and startling in its appearance that it may be considered as highly diagnostic of the verity of the attack, a quite different condition than that which occurs in the numerous hysterical and other emotional attacks which but simulate true angina and which are quite properly classed as pseudo angina. The patient suffering from the paroxysm of a true angina is inarticulate. He stands fixed or transfixed in his agony, he rarely even moans, and he never seeks to express his agony in words or gestures. He is far too keenly occupied with his own agony to attempt any theatrics at such a time. What-

ever his position may be at the onset of the attack, he will maintain it. The muscles are fixed, even the effort of lying down is rarely attempted when the attack comes on with the patient standing. If seated he remains so and the muscles of the whole body are fixed in an apparent effort to limit the pain. The skin is grey in color, covered with cold sweat and the whole picture is one of tremendous inarticulate agony.

The degree of terror is so great in this disease that it appears as a distinctive feature of the symptomatology. There are cases of angina pectoris, so-called "Angina sine dolore" in which the element of actual pain is not in itself great, but in which the associated vaso-motor phenomena of collapse are still present. Several cases which have been successfully operated upon by section of the sympathetic or removal of the ganglia are reported as retaining this symptom, notwithstanding the fact that the pain itself is entirely gone. This anxiety appears to be a distinct phenomenon and it seems to be entirely independent of the volitional mentality of the patient. Cases in which anxiety and terror exceed and dominate the associate symptom of pain are relatively common. In 122 instances of the writer's series this peculiar symptom was a dominant factor. The symptom has been described as a fear of death. While in some cases this term

does properly express the symptom, in others it is a very inadequate phrase, for the agony of the patient may be so very great that he rather fears that death may not come to his relief. As in the painful cases the patient stands transfixed with agony. Even educated physicians who have angina may be unable to describe this symptom adequately, but there is a sense of impending disaster, of breathlessness, apprehension, melancholy and such emotional agony that the suffering is approximately as great as from the pain itself. The same reflections of disturbed sensation may be present with the same distribution as in the reflected pain. The skin becomes greyish blue in color, covered with cold sweat. Tingling, numbness and all sorts of sensory disturbances appear and at times a furious itching of the affected areas may develop.

No matter how accustomed the sufferer may be to these attacks; no matter how understanding he may be of them or how indifferent he may be to death, he is inevitably terrorized by them. The writer is at a loss to explain this symptom except as another evidence of grave sympathetic stimulation akin to that seen in true shock. Perhaps no word so well fits the condition as that of a severe anxiety. It is a very striking manifestation of the disease, probably allied to and not distinct from pain; once seen or experienced it is so different

in appearance from a hysterical or neurotic manifestation as to be unmistakably distinct from it. The patients who suffer the anxiety without the pain also make no complaint or outcries during the attack; they remain perfectly still and their appearance and behavior is precisely similar to that of patients who are suffering from the grilling pain just described.

These two symptoms, pain and anxiety, are the two outstanding and altogether characteristic symptoms of angina pectoris. Attacks may almost be said to be characterized by a lack of definite cardiac disturbances. After the acute attack has subsided the patient frequently voids large quantities of clear, light colored urine of low specific gravity.

Arrhythmia in the course of the attack is generally slight or entirely absent, unless the heart be arrhythmic also when no attacks are present. In some instances in which arrhythmia is customarily present, it ceases during the attacks. The author has observed attacks in cases of auricular fibrillation which during the attack ceased to fibrillate and returned to a normal rhythm. The pulse may be full and regular and the heart sounds may be apparently normal, but usually the pulse is rather small and the heart sounds distant and of poor quality. The writer has seen cases the verity of which was testified to eventually by death and au-

topsy in which auscultation showed absolutely nothing indicative of disease during even severe paroxysms. He has also on several occasions been enabled to watch the cardiac shadow for several minutes through the fluoroscope in mild attacks, when nothing of an independently suspicious nature was manifest. In but 31 cases of our series was an arrhythmia present.

Even normal electrocardiographic graphs may be taken not only between attacks but also in the midst of major paroxysms. In one instance the writer was enabled to cardiograph the patient during what proved to be a fatal attack and the graph read by several experts was pronounced to be that of a normal heart. A still more interesting case was demonstrated to the author by Meyer Rabinowitz of the Brooklyn Jewish Hospital. This patient gave a history of frequent clinically unmistakable attacks. He presented a history and the clinical findings indicative of an extensive coronary arteriosclerosis. The electrocardiogram presented indisputable evidence of serious myocardial disease and of presumable disease of the coronary system. He was electrocardiographed in the midst of an attack in which thrombosis of a coronary branch occurred. The record was taken by the same cardiographer who had taken the original records and on the identical instrument. It showed during the paroxysm

absolutely nothing indicative of cardiac disease, though the clinical aspects of the case were entirely unmistakable at the time. Thus the difficulty of recognition of non-characteristic attacks of the syndrome is illustrated and our diagnostic dependence on the symptoms of angina and anxiety emphasized. Of course in mild cases the valuation of these two symptoms is dependent also to a great degree on the credibility accorded the patient.

Murmurs or other abnormal sounds not present during the quiescent periods are rarely developed in the course of an attack. We must of course except here the pericarditis which appears with such great frequency in coronary thrombosis. Small wonder then that the tyro in the observation of the complex very frequently fails to recognize the disease as of cardiac origin or to realize its grave significance except in those instances where the pain is very severe.

With such profound evidences of sympathetic involvement and mechanism in the complex one would naturally expect to find great variations in blood pressure, particularly since the theory of vascular spasm in the coronary vessels seems to be so well authenticated, at least for some cases. The writer does not know why it is, unless it be the effect of presphygmographic tradition that so many physicians not well familiar with the condition still

assume that it is associated with hypertension. It is true that hypertension, or the lesions associated in its production, may be causative factors in the production of angina pectoris, a fact indicated by 91 instances of this apparent origin in my series, but hypertension is not an immediate cause of the paroxysms, even though, as said, in certain instances it may be a remote cause. During the actual paroxysm the blood pressure is commonly lower than the customary pressure of the individual. Angina pectoris in my experience is unassociated with hypertension in so far as the immediate attack is concerned.

In the immediate attack, as in all forms of shock, the blood pressure may be depressed somewhat, at least relatively, during the paroxysm, but much less than in shock. How the pressure may be in the heart itself or in the aortic arch or the coronary vessels, is only a matter of speculation as yet, but we do know that the use of such vaso-dilators as the nitrites for the relief of the spasm is not contraindicated by hypotension. Nor is the drug less efficient in such instances. The pulse rate is usually unchanged, but it is more frequently slowed than accelerated.

Believing, as the author entirely does, that angina pectoris is inevitably a result of grave disease of the heart or aorta, it is peculiar and noteworthy

that dyspnœa is so rarely a symptom of the complex. In the cases of the author's series this as a symptom was complained of prominently in but 61 instances. During the immediate attack also, true dyspnœa is rarely seen. The cases are breathless, the muscles of the thorax and neck are fixed, but the patient does not, as a rule, present such a dyspnœa as is seen in cardiac decompensation from any cause or in nephritic disease or in asthma. Similarly cyanosis is infrequently present in the individual attack. Though the patient has a ghastly appearance, it is not that of a cyanosis, but a pallid grey color in comparison to the blue tinge of true cyanosis.

The Precipitation of the Paroxysm: One of the most frequent immediate excitors of the paroxysm in susceptible cases of angina pectoris is physical exercise. The patient soon learns that attacks are precipitated by any exercise beyond the usual, by walking against the wind, up flights of stairs, by lifting objects, even by bending over as to tie a shoe lace; anything which demands a physical effort sometimes of very trivial character, may produce the attack. The patient must always be warned never to undertake any physical effort beyond that necessary for the requirements of his obligatory responsibilities.

After this, undoubtedly the most frequent causes

of attacks are emotional stresses or excesses. Anger, grief, even pleasurable emotional experiences, are among the most frequent excitors of attacks. Prolonged mental or emotional effort are serious matters for the sufferer from this condition.

Combined with these two chief factors are certain physiological processes which incite attacks with great frequency. Many seizures, for example, are developed as a result of straining at stool, in the course of coughing paroxysms, even the efforts of mastication and deglutition may develop an attack. Sufficient attention has never been paid in the literature of the subject to the dangers of coitus for subjects of this disease. Every physician of wide experience appreciates nonetheless the frequency with which attacks are developed in this effort, combining as it does both physical and emotional stress. Unsatisfied sexual stimulation is however almost as bad, and the astute physician must judge his patient wisely in his advice on this subject. Overloading of the stomach is a frequent exciting cause of attacks, singing, performing on wind instruments and even the emotional effects of musical and art appreciation, the reading of exciting books, the witnessing of thrilling plays, movies and the like entail real danger to the temperamental anginal patient. Disagreeable or harassing thoughts in susceptible persons may excite attacks, and frequently do so.

Exposure to cold air, to hot applications, needle baths of either extreme, and even vesication or any other form of skin stimulation, as from the unwise use of electricity, may cause attacks to appear.

Complete vasomotor relaxation such as occurs during sleep may be a cause. This factor should perhaps receive some elaboration because of its apparent inconsistency. A good many subjects of the disease develop paroxysms during sound sleep. Mackenzie particularly has studied these occurrences and believes them to be brought about by a slowed cardiac circulation and a cardiac ischæmia. This picture is of course not to be confused with those attacks which develop during sleep in which dreams of a violent character appear and thus precipitate the attack either through emotional or physical stress.

As a rule each individual, if he does not die too soon, learns the factors which are most likely to induce his personal attacks, and the physician will do well to listen to the prejudices of his patients in this respect, for the range of idiosyncrasy is large and extremely varied. There is no doubt but that complete recognition on the part of the patient of the factors which are most likely in him to induce attacks forearms him against them. But while it must be impressed on the patient that he is during the attack in greatest danger, he remains also in constant danger as long as the pathology of the

basic cardiac disease exists. Once a patient has had an anginal attack, he should always thereafter remain under medical supervision in order that the basic pathology may in so far as possible be remedied. Subsequent attacks of the syndrome must be prevented by the avoidance of the factors which induce the attack in so far as this is possible. Prompt means to abort or terminate an attack should be taken at its very onset if it develop, for there is no question but that prompt means, to be discussed under the heading of Treatment, may and commonly do prevent a following attack from becoming severe. The danger increases proportionately with the severity of the seizure.

[CHAPTER IV

DEATH AND COMPLICATIONS IN ANGINA PECTORIS

DEATH in angina pectoris is most frequently brought about by coronary embolism or thrombosis with resulting cardiac infarction. Coronary embolism or thrombosis, based on the existence of the cardiac pathology of a tentative angina pectoris may be, indeed, the very first symptom or sign, but it occurs as a rule after fairly definite evidences in the way of preliminary attacks of angina. Coronary thrombosis invariably results in a cardiac infarction the degree and extent of which is determined by the size and particular coronary radical which becomes involved and on whether the process subsequently extends or not. Post mortem evidence has shown definitely that many cases of coronary thrombosis recover. The writer recalls the case of a colleague who had suffered from typical angina pectoris for upwards of sixteen years. During this period of time he had unmistakable coronary thrombosis nine times, finally dying as a result of his last attack. The infarcted area in the cardiac muscle may become revascularized from ad-

jacent vessels, or in some instances by a canalization of the originally diseased vessel. Coronary embolism is exceedingly frequent in angina pectoris. Of the cases in the writer's series, 85 showed coronary thrombosis. Recovery takes place in a very considerable number of instances, it is true, but once the lesion has developed, the efficiency and possibilities of that heart are greatly minimized. In many instances, death takes place immediately when the coronary lesion appears; it may then be the first and also the last symptom of the disease, but usually several hours elapse before death eventuates. During this period the patient suffers severely from anginal pain; he becomes cyanosed in some instances, blanched in others, but his appearance marks the patient as unmistakably gravely ill, in all cases. The pain may last from a few moments to many hours, or even days before it becomes sufficiently mitigated so that the patient is able to go on without heavy doses of morphine or of some other powerful sedative.

When death does not take place soon after the lesion, a typical clinical picture, that of coronary thrombosis, is presented. The patient develops a temperature ranging from 100° to 103°. A pericarditis is usually present, and a polynuclear leucocytosis of from 10,000 to 20,000 appears. The pulse is very rapid as a rule, much more so than is pro-

portionate to the temperature, though in some instances, particularly where there has been interference with the bundle of His, a slowing of the pulse is present. In all instances the pulse at the wrist is soft and weak. The blood pressure drops very low and a state of collapse comparable to that seen in the acute attack is present. Physical examination shows as a rule hyperesthesia of the precordial area. The cardiac area of dullness is increased, and the weak distant heart sounds are further obscured by a pericardial friction rub. Arrhythmias of various sorts appear, the character and degree of which are largely determined by the extent and location of the area of infarction.

If death is not immediate it may be delayed for long periods of time and no small number of cases of even extensive coronary thrombosis live for years after the injury has taken place. In some of these instances, curiously, the patient is relieved of his acute suffering with the onset of the lesion. Even though he may have been suffering from frequent attacks of angina previously, after the thrombosis he has complete relief. I have known nine instances of this kind in which complete relief from attacks was granted and the patients were enabled to live in tolerable comfort and a degree of efficiency nearly as great as that existing before the injury for as long as fifteen years. As a rule, how-

ever, subsequent to the attack there is a great limitation of muscle reserve and the patient's cardiac possibilities are greatly lowered.

One coronary embolism is almost always followed eventually by other attacks, if the patient survive the first.

Months, or years, after the coronary thrombosis has taken place, the area of infarction is marked out by a dense scar, the extent and location of which is determined by the tissue originally supplied by the diseased vessel. Post mortem studies show many instances in which canalization of the thrombosed vessel has taken place and scar tissue repair has been carried out so effectually that few symptoms may have followed. The writer has been particularly interested in the not infrequent cases of angina pectoris which receive after coronary thrombosis complete or nearly complete relief from attacks of angina. Von Neusser mentions the disappearance of angina after the heart decompensates, and he cites Curschman who found in such an instance the obliteration of a small branch of the left coronary artery. Similar instances are recorded by Langer and Toldt. Musser is also quoted as having noted a complete disappearance of the anginal manifestations after the heart has decompensated. Other writers have made the same observation. It seems highly probable that at least some of these instances

are like the cases cited by Curschman, Langer and Toldt. The mechanism of anginal relief may be the same as the writer has mentioned above, namely that a thrombosis of a diseased coronary necessarily precludes further spastic contractures on the part of the involved artery and thus gives relief from the attacks. Naturally the heart after such an incident can never regain its normal muscle reserve, and more or less evidence of decompensation or of limitation of reserve at least, is to be expected. Result must not however be considered as cause. From such instances it even seems possible to predicate that at some time, when the surgery of the heart becomes better understood, tieing of a diseased branch of the coronary may be attempted for the relief of angina caused by coronary disease.

In some cases of coronary embolism, death does not directly result from the coronary lesion, but from a secondary embolism, as from infarction of the lung, presumably caused by the dislodgement of an intracardiac clot. In other instances, a postural pneumonia appears as the final complication of the coronary thrombosis. Multiple coronary thromboses are not infrequently seen at necropsy.

Rupture of the heart is a frequent result of coronary thrombosis. The rupture may take place immediately coincident with the infarction of the lesion or it may be delayed hours, days or weeks.

It may result in the formation of aneurism of the heart, often diagnosable by means of the fluoroscope and X-ray plate in association with the history and other physical findings. Rupture of the fibrous sac of the cardiac aneurism may take place years after the primary coronary lesion was inflicted.

Acute rupture with hemorrhage into the pericardial sac is a quite frequent terminal lesion of coronary thrombosis. The patient may even show all the symptoms of severe internal hemorrhage before he eventually dies. The writer recalls a case in which the patient, a syphilitic man of sixty-five years, after an acute coronary thrombosis, was able after a period of two to three hours rest to walk a distance of five city blocks to his railway station. He took his train, riding thirty miles to his home town, and then walked, though with great suffering and much dyspnoea, nearly a thousand yards up a gentle slope to his house where he went into collapse, expiring eventually some twelve hours after the coronary embolism. Judging from the symptoms and the lesions found at autopsy, he died at least nine hours after a rupture of the heart through the infarcted area had taken place and hemorrhage from the left ventricular cavity had begun. At the autopsy, the pericardium was found enormously distended with laminated clot.

The author has seen at autopsy several specimens

which apparently indicate that pericardial adhesions after coronary thrombosis had resulted in a temporary repair of the rupture of the heart. From the mingled blood clot and pericardial exudate a sufficiently sound dam of tissue had developed to actually repair the leakage from the ventricular wall.

Another frequently fatal complicating lesion of angina pectoris is cardiac thrombosis. As a rule it occurs in the auricles though it may also be found in the ventricles. Secondary embolic processes may then take place in other organs, notably in the lung, the spleen, the brain, and the kidneys. Five such instances occurred in the writer's series. Obviously, cardiac thrombosis is most common, secondary to thrombosis of the coronary vessels.

Hypostatic pneumonia is still another frequently fatal complication of angina pectoris. Naturally it is particularly common in cases which become bedridden, or in which the heart manifests not only attacks of angina but also a general muscle defect.

Von Neusser (Authorized English Translation) calls attention to the interesting fact that angina pectoris is almost unknown in either mitral insufficiency or stenosis. He also states that in aortic insufficiency of rheumatic origin, angina is an occurrence of the greatest rarity. These statements are in entire accord with the author's experience. Myo-

cardial fibrosis and myocardial degeneration are frequent complications, or perhaps more properly, concomitants of angina. Renal defects are other frequent associations. Hypotension, and hypertension are other conditions which either cause angina, are associated with it, or are a result of the recurrent attacks.

Of particular importance in the course of an angina are the acute infections, tonsillitis, rheumatic fever, bronchitis and pneumonia. Perhaps most important of all this group is influenza, which is a particularly serious condition for the subjects of angina because of its grave degenerative effects on the heart muscle and the frequency with which dilatation of the heart follows.

Hyperthyroidism is one of the exhausting conditions which is especially serious in the subjects of this complex, not only because of the degenerative changes in the heart which the thyroid defect engenders, but also because of the cardiac exhaustion which appears perhaps from no more than the rapid action of the heart. Hypothyroid states are frequently associated with anginas, but the writer does not consider hypothyroidism either as a direct cause of angina nor, per se, as a result of it. Most hypothyroids seem to tolerate angina with a fair degree of resistance.

Naturally a progressive arteriosclerosis of any

type is a particularly unfortunate associate of angina pectoris. Cases which manifest it are particularly poor risks when they show also the symptoms of angina. The same is true of nephritis of any form, and particularly of either the acute or the chronic glomerular types.

Obesity is a serious complication for the anginal subject. Twelve such instances occur in the writer's series. Every experienced clinician realizes that family types and tendencies have much more to do with the determination of obesity than any dietetic factor, but the influence of a fat rich diet is very real in obese persons who are also the subject of angina. It is as a rule unsafe to attempt any severe dietetic measures in anginal patients who are also obese, and the best effects are commonly produced by reducing the amount of food intake, but taking care to preserve a well balanced relationship between the various food elements. As Chittenden has well said, it is not so much starvation which is dangerous but rather the taking of an unbalanced diet. Many of the diets in use for fat reduction especially on lay authority are very dangerous undertakings for the subject of angina. Another reason why obesity once established is a particularly serious matter in angina pectoris is that, because of the syndrome, patients are unable to exercise adequately and thus to reduce in a normal way.

Joslin in his text book has in particular called attention to the association of angina pectoris with diabetes. It is unlikely that the hyperglycemia is responsible for the production of the angina, but rather that arteriosclerosis of the pancreas is productive of diabetes, and that it is frequently associated with a similar change in the coronary system which is in turn a cause of angina. He states that of 4676 cases of diabetes seen since 1895, a diagnosis of angina pectoris was made in 122 instances. Twenty-six cases of probable angina or coronary occlusion should probably be added in which sudden cardiac death took place. Joslin says that among diabetics it is not profitable to attempt to distinguish between coronary occlusion and angina pectoris. He finds that angina trebles in the second ten years of diabetes as compared with the first ten years of the disease. From the nature of Joslin's chief material it is natural to suppose that most of his cases were originally diabetics and that the angina appears as an incidental occurrence. Kahn (Am. Jour. Med. Sci. 1926, 172, 195) found 10 cases of diabetes in 82 instances of angina pectoris. Wilder has also remarked on the high occurrence of coronary disease and myocarditis in diabetes, and notes particularly advanced coronary disease in cases which had gangrene of the extremities. In the writer's series of anginal cases 15 were diabetics.

There is no doubt but that diabetes affects the course of angina pectoris most unfavorably.

Though angina pectoris is a condition as shown before in this text, associated with age, in actual senile states it is not of very frequent occurrence. But 8 such instances are found in this group. The complex is not then a hall mark of senility.

The association between nephritis and angina has already been remarked, but it is so frequent a matter that it may be again mentioned in this regard. It is particularly those forms of renal diseases which are accompanied by hypertension that develop angina, though as has been already stated hypertension is not in itself a cause of the syndrome. The association of a hypertension in angina is nonetheless a particularly unfavorable factor in cases once developed, not only because of its arterial, tensive, and presumably also its toxic conditions, but particularly because of the emotional and nervous irritability which mark hypertension and the forms of nephritic disease which go with it. The cardiovascular renal complex is a frequent association in angina, and a particularly unfortunate one from the standpoint of treatment, especially because of the vascular changes in this condition and the cardiac stress which is an invariable accompaniment, in addition to which we must also add the mental and emotional irritability which is a part of the

symptomatology of the cardio-vascular-renal complex.

Still another complication or unhappy association in angina pectoris is nervous and emotional hyperexcitability, hysteroid states in which constant apprehension and emotional instability render the patient all the more likely to suffer from nervous or mental states which not only induce attacks but render them the more severe when they occur. All intelligent sufferers from the complex recognize this important element and find that the cultivation of a sense of philosophy and of toleration materially lessens both the severity and the frequency of attacks. The opposite mental characteristics inevitably lead to the opposite result.

Digestive disorders in general are frequent complications of angina pectoris. Among the most frequent immediate excitors of attacks are gastric inflation, pylorospasm, gastric retention, gastroptosis, and enterocolitis. Many persons subject to angina suffer attacks only when the stomach or intestinal tube is distended either under pathological or physiological states. The appearance of almost any sort of gastrointestinal or gall bladder disease induces and aggravates the angina. In some instances, notably when the basic pathology is a defective heart muscle, gastrointestinal symptoms appear as a result of the angina or because of its etiologic

pathology which in instances of cardiac defect may bring about a congestion of the liver and of the gastrointestinal tube. There are no cases of angina in which the study of the gastrointestinal phases does not form an important subject, and disturbances of this tract are particularly frequent and always important.

CHAPTER V

PATHOLOGY

ALTHOUGH the clinical picture of angina pectoris is consistent and dramatic in its character, even the earliest pathological studies soon demonstrated that there was no inflexible, constant, and certain pathology back of the symptom complex. No student of the disease can feel otherwise than that we are not in truth dealing with a definite and specific disease in angina pectoris. Irrespective of its symptomatological identity we are certainly not dealing with a specific disease process, but rather with a symptom complex or syndrome which may be brought about by a highly diversified and quite variable pathology. Angina pectoris is not then a disease but merely a symptom complex in so far as our present knowledge goes. It is important for the therapeutist to recognize this fact if he is to correctly treat cases of the syndrome. Treatment is by all odds the most important subject for the physician to comprehend, and it must in each instance be directed primarily to a correction of pathology, and an attempt at return to a normal physiological state.

Whether or not there exists a physiological basis

or disturbance which is universally present in all cases of the syndrome remains also to be shown. Many studies designed to establish such a basis have been, and are being made. Even should such a basis be discovered by means of which a common physiological deviation could be established, it would still not materially affect treatment of the condition at least in most instances, for curative treatment must almost inevitably be directed toward the correction of primary pathology. Mere symptomatic treatment in this complex is now so well established as to admit of but little hope of improvement. Our larger problem certainly from the standpoint of therapeutics should be aimed at the pathology. If this is successful and is inaugurated before permanent tissue changes or tissue habits have been established there is hope of a complete and radical cure of the condition with the restitution of a normal degree of life expectancy.

Heberden was apparently of the opinion that the complex was produced and was dependent on a true muscle cramp exactly comparable to the cramp which occurs in striated voluntary muscle, or in the spastic contractures of smooth muscle. Traube supported this theory, and even today it has its advocates. Judging from the symptoms chiefly manifested in the disease, Heberden's original assumption is quite natural, and it is well sustained by the

character of the pain present; but clinical observation shows that almost invariably there is no pause or intermission of the pulse during the attacks, for it is usually maintained at a regular rhythm and rate throughout the paroxysm. That cramp or spasm does not take place in any portion of the heart muscle during the attacks is also shown by electrocardiographic studies. Furthermore, in so far as we know, the striated involuntary muscle of the heart never does go into a condition of cramp or spasm.

Jenner's and Parry's observation that the symptom complex is usually associated with disease of the coronary arteries is founded on sound pathological observation which stands today as then. On the other hand, numerous studies have shown that disease of the coronary system is not invariably associated with this syndrome. Every student of gross pathology knows furthermore that extensive disease of the coronary arteries may be, and often is present in numerous instances entirely without the production of the syndrome of angina pectoris. It is also known that complete blocking of a coronary vessel while it temporarily may produce a clinical picture similar to that of ordinary though prolonged anginal seizure, is often followed by a complete or temporary cessation of the anginal attacks. It is then evident that disease, slight or

severe, permanent or transitory, resulting in strictures of coronary lumen, is not the entire pathology of angina pectoris. It is however undeniably the most frequent basic pathology.

The majority of physicians adhere to the theory that the anginal paroxysms are brought about in diseased coronary arteries by a spasm of the lumen as advocated originally apparently, by Allen Burns in 1869, who compares the process to that known to take place in intermittent claudication. This theory was also advanced by Potain in 1870. This theory has stood the test of time in the main, and it receives strong corroboration also from the fact that in most instances the paroxysm may be aborted or terminated by the administration of the nitrites as shown originally by Lauder Brunton. To this day, this is the commonly accepted theory of most physicians, but it is not invariably true, for we have also instances of angina apparently authentic in every way which probably do not show coronary spasm.

Mackenzie and many others before him, assumed that the ischæmia, temporary or permanent, which followed this spasm with its limitation of muscle nourishment induced the attack. The negative electrocardiographic findings which are not infrequently obtained during the immediate paroxysm tend to refute this theory, but it furnishes in my opinion the most satisfactory and helpful single

explanatory basis for the most striking symptoms of the disease. It is that theory which is as yet best borne out by therapeutic experiment and result.

The syndrome is found in many instances in which, at post mortem, extensive disease of the aorta is present. As a rule those cases of aortitis which develop angina pectoris are such as show the disease changes most extensive in the first part of the ascending aorta, in or about the sinuses of Valsalva. It will be recalled that it is from this point that the coronary arteries take their origin, and in many such instances the author has observed the degenerative or inflammatory process to be very marked at the openings into the coronaries. Here a small tumulus of tissue may in part obstruct the lumen of the coronaries. It is easy to assume that lesions similar to these may produce at times circulatory conditions precisely similar to those mentioned above as appearing in coronary endarteritis or coronary spasm, and the assumed mechanism may be precisely identical. This is however not invariably the case for in some instances no apparent lesions of this character exist. At least there can be no question whatever in the mind of students of the complex but that there is a frequent association of aortitis, occasionally without associated coronary disease with angina. It is not however an invariable association nor is angina pec-

toris always present even in very advanced instances of aortitis. Brown and Coffey (Trs. Assn. Am. Phys., Vol. XXXIX, p. 451) evidently believe that the substernal pain is caused by a spasm of the first part of the aorta which develops as a result of a vasoconstrictor impulse transmitted through the superior cardiac nerve. How such a profound change could take place without producing alterations in the pulse and other definite indications in the circulation, is very difficult to understand, nor can the collapse and frequent death be thus explained.

Myocardial disease, either degenerative or inflammatory is found in association with angina pectoris in many instances. This fact has been cited by many observers and many types of myocardial lesions have been found so associated. Albutt notes the occurrence of angina after influenza in particular, and its appearance after other infections, and notably in rheumatic myocarditis, admits of a similar assumption of production. Anginas of this type of origin in all probability number a good many instances in which spontaneous cure follows full convalescence from the infections. The lesions may probably be compared to those in which localized ischæmias of the heart muscle appear as the probable exciting lesion. It is possible also that some of the instances of angina which may appear in early syphilis may be of similar origin, based on the char-

acter of early syphilitic myocarditis described by Warthin and his students.

Hirschfelder and others suggest that the syndrome may be produced by acute cardiac dilatations: "It is possible that under these conditions acute dilatations of the heart, due to momentary diminution in the tone of the heart muscle, might be the cause of the heart pain, which would thus be of primary cardiac rather than vascular origin." Hirschfelder goes on to point out that dilatation of the heart may produce diminution in the coronary flow which would throw this classification again into a possible coronary group. Such an explanation might account for the influence of physical stress on the immediate precipitation of attacks of angina. The author's experience with the syndrome has not emphasized the probability of this theory though it doubtless remains as a possibility. The notable absence of dyspnœa, and the usual absence of cyanosis, arrhythmias, and œdema in true angina is against this theory.

Von Neusser cites the case of a patient who two years after an attack of rheumatism showed typical signs and symptoms of an angina pectoris. Necropsy showed only adhesions of the heart to the pericardium. Other authors also have stressed the association of pericardial adhesions in angina pectoris. In most of these instances, it is notable that the

chief process has been about the base of the heart and the root of the aorta which is supplied mostly by branches of the superior cardiac nerve. It must also be remembered in this relation that a pericarditis almost always follows coronary embolism, and if life persists adhesions are to be expected. This might well explain Von Neusser's case. Also it must be admitted that pericarditis is almost inevitably associated with more or less myocarditis. The observation of the author does not incline him to give too much credence to this theory, but where the adhesions are about the base of the heart in particular, it is readily conceivable that serious interference with the coronary circulation may be brought about and so induce an angina. Were this explanation a frequent cause, one should find angina in acute pericarditis which is rarely seen except when the pericarditis is caused by coronary thrombosis.

Very naturally the symptomatology of angina has been variously ascribed to neuralgic conditions. This theory has received some corroboration from the work of Lancereaux, Grocco and Penati, who have described lesions in the cardiac plexus, but the theory lacks at least general acceptance, and it is not in accord with what we know of the well established pathology of the complex, nor does it accord well with what we know of the nervous control of the heart in other respects.

Further light appears to be thrown on the probable pathology of angina pectoris by the association of the syndrome with certain definite disease conditions.

Though angina occasionally appears in children particularly after grave infections, and especially after rheumatic fever, it is preeminently a disease of late adult life, occurring mostly after fifty years. This certainly suggests the influence of ageing processes which are, it is well recognized, for the most part identified with arterial disease.

All modern students of the condition recognize the frequency with which angina pectoris occurs in association with myocardial disease and its appearance after scarlet fever, influenza and particularly after rheumatic fever has been almost universally remarked.

It occurs frequently with diabetes, as has been particularly pointed out by Joslin and by Kahn. Again in diabetes an association with arterial disease appears to be present, as will be emphasized later on in this book. That the glycosuria in itself is not a causative factor is suggested by the fact that high blood sugars are not apparently associated with the condition, irrespective of the other factors which are dependent on vascular ageing.

The frequency of angina in syphilis has long been noted, and the statement is attributed to Osler that

angina appearing under forty years of age should be considered as luetic until the contrary has been proven. The author reported on a study of the relation of syphilis with angina, in 1926 (Transactions, Assn. Am. Phys., Vol. XLII, p. 31). A total of but nine instances of angina pectoris caused by syphilis was discovered in a list of 200 consecutive instances of angina. Paul White, before the same society in the previous year, gave similar figures. The average age was 52.6 years; the oldest patient was 63 years of age; the youngest 51. This corresponded very closely to the average age of cases of angina pectoris from other causes. It also coincided closely with the average age in which serious cardiac symptoms appeared in his series of cases of cardiac syphilis. But one instance occurred in a woman. In four cases a definite history of syphilis was obtained. In no instance had early or proper treatment been given. In none did angina appear less than twenty years after the probable initial infection. This corresponds also very closely to the average time of occurrence of cardiac symptoms of a disqualifying character found in his analysis of cases of cardiac syphilis. In five instances the angina was associated with a history and physical findings indicative of associated syphilitic disease of the central nervous system. Again this agrees closely with the analysis of cardio-vascular syphilis. In

three instances the angina was associated with marked generalized arterial disease, probably of luetic origin. As to the anatomical structure probably more seriously involved, two cases gave evidence only of dilation and sclerosis of the aortic arch with aortic endocarditis; one gave positive evidence only of myocardial disease (electrocardiographic study). In two instances the dominant change seemed to be in the coronary vessels, in both of which coronary thrombosis took place. In the remaining four instances the aorta and the cardiac muscle showed associated lesions of definite character; three cases which showed marked improvement under treatment belonged to this associated group. Both cases of coronary embolism died promptly. The Wassermann reaction was negative in two instances, one of these was subsequently found to be congenital in origin. This case, nonetheless, showed very marked improvement under specific treatment. Typical syphilitic stigmata were present not only in her, but in her brothers, two of whom had died of aortitis. Two cases showed intermittent claudication with generalized arterial disease, probably syphilitic. All instances comprised in this study gave a story of stressful life, of emotional excesses, and of an occupation implying long continued nervous strain. Two instances only showed a vascular hypertension, 175/120 mm. of mercury in one, and

115/100 mm. in the other. The only instances of hypotension were those of coronary thrombosis which should not be considered as basically hypotensive. In the remaining cases, the blood pressure was well within normal limits. In so far as this list of cases goes, the writer was surprised at the relative infrequency of angina pectoris as compared with the number of cases of cardiac syphilis. It seems very probable that in this respect the occurrence of angina pectoris caused by syphilis will be found in about the same proportion to the occurrence of angina in other types of cardio-vascular disease. It seems reasonably certain that angina pectoris is not a very frequent symptomatic manifestation of syphilitic cardiac disease, probably no more so than it is in the forms of cardiac disease caused by other factors. It has impressed the writer also that anginal symptoms do not as a rule occur as an early manifestation of syphilitic heart disease. In all instances the time of occurrence has been at about the usual age of appearance of grave heart defect, in cardio-vascular syphilis without angina. Evidently early syphilis rarely develops the symptomatic picture of angina pectoris.

Dr. James E. Paullin, in a study of syphilitic infection in the negro (Trans. Assn. of Am. Phys., Vol. XLII, p. 46), states that in 662 cases of syphilis the cardio-vascular system was found in-

volved in 176 instances. In 46 of these instances aneurism of the thoracic or abdominal aorta was present, 75% occurred in the male. 0.45% only of this large group of cases developed a coronary thrombosis, and in none of the cases was angina pectoris mentioned as having been present. In the discussion of this paper, both Dr. Paullin and Dr. W. S. Thayer, called attention to the difficulty of eliciting a history of anginal pain in the negro. Dr. F. T. Lord in discussing the writer's paper, states that of 140 of his patients with angina, having a Wassermann test, it was positive in but 6. Dr. Scott in discussion of the same paper reported that anginal attacks were rarely seen in 75 hospital cases of syphilitic aortitis. Warfield Longcope believes that in estimating the importance of angina as a symptom of syphilitic aortitis, we must take into account the question of racial differences. Dr. C. M. Pincoffs differed from the writer and finds that the average case of cardio-vascular syphilis might show angina at 40.6 years. This average was obtained from a series of 17 cases. In contrast to this he found that the average age of non-syphilitic angina in the same hospital was 60 years. Albutt states (Lancet, May 5, 1923) that in persons under middle age, it is for the most part due to syphilitic origin. He does not, however, quote his statistics. It must be recalled that syphilis is a disease which

pathologically is identified with vascular changes, and statistics not to the contrary there is an undoubted dependent relationship between angina pectoris and syphilis. The frequent relationship between syphilis, aortitis and coronary disease has long been recognized as well as the frequency with which syphilis induces also myocardial lesions, usually extending out from the coronary trunks.

Constitutional diseases, as gout and chronic diseases of the kidney, show also an association with the development of angina which is so frequent as almost to bear a causal relationship in its etiology. These conditions also are notable because of the profound alterations which they induce in the circulatory organs, especially in the production of arteriosclerosis of all forms.

Notwithstanding the unquestioned influence of nervous and emotional factors in the induction of the symptomatology of angina pectoris, the writer is very anxious to impress his view that practically all instances of true angina pectoris are associated with and are probably caused by grave lesions in the heart quite sufficient in themselves to constitute a most serious menace to life and quite capable of causing under conditions of stress, the most dramatic feature of angina pectoris, sudden death. Later on in this book the writer will attempt to make a sharp distinction between true angina pec-

toris and pseudo angina, and he will hope to show that the so-called toxic anginas are also distinct from true angina in that in them, as in false angina, no grave pathologic basis is present. There is thus, in the opinion of the writer, a very definite pathologic basis of distinction between true angina pectoris which is based on a pathology always of grave nature, one in which death is likely to occur at any time and the other forms of angina which resemble the true form only symptomatically. Furthermore the writer believes that the differential diagnosis between true and false angina is commonly possible.

The writer also believes that the pathology of this disease is sufficiently definite for us to assume that one may safely believe that true angina pectoris is based on a pathology of quite definite character which may be subclassified as

1. Cases caused by disease or spasm of the coronary arteries.
2. Cases caused by aortitis, chiefly involving the first portion of the aorta.
3. Cases caused by disease of the myocardium.

In many instances these three factors are associated, as aortitis with coronary disease or all three together as is frequently found, for example, in syphilis or rheumatism.

It is obvious that all these lesions may exist, and

they frequently do without causing angina pectoris, but they never exist without bringing about the menace of sudden death. In those instances in which the symptomatology of angina appears there is an additional factor present which acting through the nervous system brings about the typical clinical picture of angina pectoris. These nervous elements may exist independently of the pathological lesions and in such instances they may produce the syndrome of a false angina, which may be distinguishable from the true syndrome only by the recognition of a basic pathology which may be commonly detected or strongly suspected, certainly in most instances by the methods now available to any well equipped physician.

The angina pectoris described by Heberden cannot be accounted for on a purely nervous basis. It is exceedingly doubtful if purely nervous stimulation or disease can ever cause death independent from primary and independent disease of the heart muscle, coronary vessels or of the aorta.

CHAPTER VI

PATHOLOGICAL PHYSIOLOGY

HUCHARD is said to have collected forty-seven different explanations of the mechanism of symptom production in angina pectoris. It is neither profitable nor practical to consider all of these, nor is it possible to consider all or even many of the newer explanations of the syndrome which authors since the time of Huchard have presented for they are very numerous. Nor from the standpoint of a clinician does the writer consider it necessary to discuss elaborately these various theories. In presenting therefore, his own explanation of the mechanism of symptom production of angina pectoris, the writer at the same time wishes to offer an apology for still further encumbering the literature of the subject already too rich in explanations and too poor in therapeutic suggestions for the relief of the condition.

The explanation which the writer finds most helpful from the standpoint of the practicing physician is necessarily an adaptation of many theories. In the mind of the author the most important concept for the physician to bear in mind is that this

complex has a definite though varied basis in real cardiac pathology. As already stated, the writer feels that all true cases of angina pectoris have a distinct pathology located either in the first portion of the aorta, in the myocardium or in the coronary arteries. In point of frequency the last, that is, the vessels of the coronary system appear to be the most important. It is also conceivable that pericardial disease through extension to the root of the aorta on the one hand or involvement of the myocardium on the other may also present a basic pathology sufficient to explain angina pectoris. The writer feels definitely that any explanation which does not involve lesions of the heart and aorta quite capable of causing death is insufficient and dangerous for the physician to entertain.

On the other hand it is obvious that these changes alone are incapable of causing the symptom complex characteristic of this condition. All of the lesions mentioned are found frequently postmortem in patients who during life present absolutely no indications of angina pectoris. These are the cases of sudden cardiac death which occur so frequently without any preliminary history of circulatory disease and without any history of angina, though, as before stated, a certain considerable number of cases do so terminate with the symptoms and signs of a

coronary thrombosis or of an angina terminal in character.

Why do some of these cases develop angina and why do some of them with precisely similar lesions not develop angina? These are the questions which the profession has been trying to answer since the time of Heberden, Jenner and their contemporaries. They still remain unanswered, though many of us are still amusing ourselves in the attempt.

Angina pectoris is curiously a disease of the more highly organized persons. It occurs almost exclusively in highly emotional and nervously sensitive persons. The attacks are precipitated not only by physical stress, but notably by emotional and nervous factors. In other words then, in addition to a basic cardiac pathology it is necessary that a sensitive nervous arc which must include the higher psychic centers be also present for the development of the symptomatology. The heart is itself largely or entirely insensitive to pain but through its sympathetic connection to the spinal cord and its pain sensitive cells the other phase of the chain is supplied. Both are necessary to the development of an angina pectoris, a basic cardiac pathology which is in itself dangerous to life and with this a resulting and probably protective sensitized nervous arc which makes the patient aware of his circulatory

defect and warns him against those conditions or states which augment the danger.

If this theory has something of truth in it, then we should find that a break in this nervous arc, as a result of which the body is made aware of its infirmity should be followed by an increased danger to life, on the basis of the theory that knowledge of the lesion, gained through pain forearms the patient as it forewarns him. This is a point which should be well appreciated in determining the treatment which patients with this complex should receive.

CHAPTER VII

PSEUDO ANGINA PECTORIS

THE term pseudo angina pectoris has been severely criticized by many authors of the highest possible standing. This is particularly true of such standard clinicians as Albutt, Mackenzie and Lambert. In most of these criticisms the basis of the complaint is founded on the fact that true angina pectoris is not a definite disease condition, nor in any sense a true disease entity. This is a fact from which no physician of any experience would for a moment dissent. It remains however that the clinical picture of angina is characteristic and important, whatever its mechanism of production or basic pathology may be. The very great gravity of the complex, whenever it occurs is so apparent to every experienced physician that the recognition of the picture as a clinical entity, probably due to a highly varied pathology simply demands that the condition be identified and characterized as certainly as possible. The other chief argument against the recognition of this group of symptoms, distinct from true angina, in that it is not a menace to life, is the great difficulty of differentiation from true an-

gina. This is unfortunately also true of many other symptomatic pictures.

In pseudo angina the entire clinical picture is subjective whereas in true angina while the chief symptoms are also of subjective nature in well pronounced or recurrent attacks almost always a certain degree of observable symptoms, the fascies typical of true angina appear. There is no other differential sign so certain between true and false angina as the appearance and reactions of the patient during the attack. A very interesting and distinctive differential point between the two conditions is that in true angina, when the attack is fully developed, the patient is almost invariably speechless, he holds himself rigid, he neither wishes nor asks for pity or solicitude, while the subject of false angina is almost always voluble, he groans, cries, desires sympathy, describes at length his miseries and almost always betrays thereby the hysterical and neurotic nature of his complaint.

While true angina pectoris may attack a neurotic and hysterical person, they rarely manifest the evidences of these tendencies while actually suffering in the attack. On the other hand all instances of pseudo angina occur in neurotic or hysterical persons and the acme of this tendency is manifested during the so-called attacks of pseudo angina.

Subjects of pseudo angina are as a rule sophis-

ticated and educated persons, at least in so far as the subject of angina is concerned. Adroit questioning along suggestive lines will in most instances suffice to obtain from most of these patients a mass of detail which either mirrors the picture suggested by the physician's leading questions or conforms so altogether with the layman's concept of angina that the verity of the attacks falls at once into question. On the other hand the subject of the true complex when not in the attack is able to give a clear concise and very positive account of his sufferings and no amount of suggestive questions will cause him to deviate from its simplicity and directness. When in the paroxysm he will ordinarily not answer at all.

Unfortunately in both conditions absolutely no evidence, physical, electrocardiographic, or by the X-ray plate may appear of real cardiac disease but in the true condition some such evidence is usually available, and repeated careful examination or careful questioning will usually demonstrate the fact that some demonstrable defect of the heart is present in true angina. The electrocardiogram is often of crucial importance in this differentiation. A very detailed and repeated physical study of the circulation is often necessary before a differential diagnosis can be made between true and false angina and in the experience of the writer in any case

of doubt the physician should not content himself with but a single observation and study of the case, but especially in supposed false cases the wise physician should insist on a period of observation before a final and conclusive diagnosis is made.

Treatment also should be of more or less marked assistance in the differentiation between true and false angina. Once the tentative diagnosis of a probable pseudo angina has been made the course of treatment outlined should in itself serve definitely to a conclusive differentiation. In true angina the patient is advised against physical stress and exercise, whereas in suspected pseudo instances he is encouraged in these directions. If an error has been made in diagnosis under such conditions almost certainly definite attacks of a bonafide type will develop. Suggestive therapeutics are splendidly efficient in false angina, but are without any effect in the true condition.

Finally the differentiation between the two conditions depends not only on a study of the circulatory signs and symptoms alone, but also to a very large extent on a study of the personality and psychology of the patient. This demands on the part of the physician far more than a mere scientific knowledge of disease, it requires also an intimate knowledge of human nature and of personality.

Because the differential diagnosis is a difficult matter is no reason why it should not be made.

Pseudo angina is much more frequent in women than in men, it is rarely seen independent of associated and allied neuroses, phobias and psychoses. This, then, is also a means of differentiation in a general sort of way. In the experience of the writer a very interesting differential point frequently arises when the subjects of this disturbance are persuaded by the physician that they are free from true cardiac disease by the fact that many of these subjects immediately substitute some other phobia or neurosis, if possible of equally sinister import. Every teacher of medicine knows how frequently such mental mechanisms appear particularly among nurses and medical students and very much of the experience of the writer has been derived from a study of such examples. In these special instances at least it is probably the result of a fear complex.

An argument frequently advanced by those who feel that there is no essential difference except in degree between true and false angina is that both the grave and the non-dangerous forms originate through the same mechanisms. This is based on the assumption that true, that is, grave angina originates not from primary cardiac disease, but from spinal or neural defects which, acting through the reflex sympathetic arcs, produce a symptomatology

which is identical in both conditions. It should in this relation be pointed out, however, that in most of these non-cardiac anginas the origin of the impulse is in the emotional and cerebral, and not in the spinal centers, otherwise it is impossible to explain the tremendous influence which suggestion plays in the eradication of the symptomatic picture.

It is certainly a very dangerous and pernicious teaching particularly for the young clinician to group these disorders together for it blocks at once anything constructive in the way of treatment in the case in which a true cardiac defect of serious import exists.

That a reverse pathological mechanism is concerned and that sympathetic or spinal disturbances react on the heart and induce in it eventually actual lesions, is against all that we know of such mechanisms. It has no parallel in any other form of disease with which we are acquainted.

The reader has, no doubt, been impressed with the writer's entire belief that pseudo angina pectoris is a neurotic or hysterical chimera devoid of danger to life, while true angina pectoris is founded on a definite, though variable, pathological basis of damage to the heart and aorta; it alone is always fraught with great danger to the life of the patient. The ground of differentiation has been founded on years of careful observation, and in the opinion of

the author this differential assumption is critically important, so that the life of the subject of true angina may be conserved and his suffering and distress mitigated.

The great point in the treatment of pseudo angina is to persuade the patient that he is not the subject of a grave and extremely dangerous circulatory disease and that he must attempt to divest his mind entirely of any such concept.

Pseudo angina is unquestionably the condition which is differentiated with most difficulty from the true condition. Beyond any doubt it also is numerically the most frequent condition confused with true angina pectoris, but there are other conditions most of them associated with or caused by true circulatory pathology which are frequently differentiated from true angina with difficulty.

One of the most important and frequent of these is the pain which appears in the precordium in association with myocardial disease of the degenerative types, especially such as follow many of the acute infections as pneumonia, typhoid fever, and notably influenza. In these conditions as a rule we find a precordial pain present which is less intense than that of true angina. It is located usually in the precordium at the ensiform or in the epigastrium. It is rarely reflected and unlike pure angina is almost always associated with other and very definite

evidences of cardiac inadequacy. Dyspnœa is the rule in these cases, it is an infrequent symptom in true angina. Cyanosis is also the rule, and is a very different condition from the grey-like pallor of true angina which more closely resembles the fascies of shock than that of a mere circulatory defect. Exercise in these conditions greatly intensifies the symptoms, notably the dyspnœa and cyanosis and as a rule after exercise swelling and tenderness of the liver is to be made out. Oedema is also frequent in these conditions, it is rare in true angina pectoris.

Not only in the conditions mentioned may angina vera be differentiated with difficulty, but also in the early stages of decompensation after old valvular disease. The recognition is exceedingly difficult at times in rheumatic disease, whether it involves the valves alone or is limited to the myocardium or when both conditions are associated, as is so frequently the case.

A precordial pain confused with true angina is also seen in patients after prolonged physical strain. It is very commonly found in the instance of improperly trained athletes, especially such as have been required to put out prolonged and unfamiliar effort of a degree to which they are unaccustomed. The writer has observed this type of cardiac pain particularly in mountain climbers, hunt-

ers and explorers. The pain in these instances is not paroxysmal as in true angina, but is dull and heavy, rarely reflected and it passes very much more slowly than is the case in true anginal attacks. It is almost always associated with digestive disturbances, with nausea and tenderness over the liver, or in the region of the gall bladder. A more or less profound general exhaustion is also frequent and insomnia appears. Rest is effective in the relief of these conditions, but its effects are less sudden than in the case of true angina. Bradycardia is the rule in these cases, though tachycardia and arrhythmia may be shown.

A very important means of differentiation of these cases from true angina is that in the types of real cardiac distress which we have just described, digitalis gives prompt and striking relief, as a rule, much more certainly and much more efficiently than in those cases even of true angina in which the basic pathology appears to be myocardial disease. We must also never forget the frequency with which the pain caused by a pericarditis may simulate that of angina.

There is another very interesting condition, characterized by true cardiac pain which may be readily confused with that of angina pectoris. This is in those frequent instances of precordial distress which appear after prolonged mental stress, as in grief,

or other prolonged emotional stresses of severe character. Students frequently complain of such symptoms after a series of difficult and feared examinations. Relatives suffer from them after long attendance at the sick beds of beloved ones, and physicians know them well as occurring after long and difficult cases, particularly such as impose an extraordinary degree of anxiety, or in which little rest is had. Unhappy love affairs develop these pains in sensitive young people.

There is also a considerable group of cases of vaso-motor origin which may be easily confused with true angina pectoris. Indeed it is questionable if there be any sharp line of distinction between these cases and those of true angina, for one condition apparently merges into the other imperceptibly. The chief characteristics of this group of cases is that the attack is ushered in by symptoms of vaso-motor constriction similar to those seen in Reynaud's disease and suggestively like those which appear in some cases of intermittent claudication. Symptoms of this character are frequent in true angina pectoris and in the writer's series of cases of angina, vaso-motor spasm elsewhere than in the heart was present in 100 cases.

Hirschfelder in his book asserts that these cases are not associated with organic changes. With this statement the writer begs to differ, for he would

make the statement that while these phenomena may occur without marked morphological changes in the blood vessels, in most of the instances which have come to his observation there have been pronounced changes in the blood vessels.

The attacks in this type of angina are introduced usually after some emotional or physical stress by the appearance of coldness of one or more extremities; there is numbness, tingling and formication of the chiefly involved areas with weakness and a wax-like pallor, succeeded by deep congestion and cyanosis. The pulse may for a time entirely disappear in the involved extremity. Shock may be simulated and anginoid pains appear not only in the precordium, but also in the involved extremities. This condition appears exclusively in highly emotional and neurotic persons, and it is usually associated with other signs indicative of disturbance in the endocrine glands. During these attacks the patients may suffer considerable agony, but the writer has never seen death take place in them.

The precordial pain following paroxysmal tachycardia is probably also to be grouped with these conditions for in the opinion of the writer the pain in all these conditions just considered is an evidence of cardiac exhaustion, perhaps to be classed with the exhaustion pains which appear in

voluntary muscle following prolonged and particularly unwonted exercise.

Hirschfelder in his book, "Diseases of the Heart and Aorta," (p. 294) calls attention to the pain described as almost anginoid which occurs in soldiers' hearts as described by Da Costa. This condition of neuro-circulatory asthenia, described by Lewis, and by other British authorities mostly under the heading of the "Effort Syndrome," is a condition frequently confused with angina pectoris. Neuro-circulatory asthenia, however, occurs almost exclusively in young persons, more in males than in females as a rule. It appears almost exclusively in highly neurotic individuals who have been submitted to unwonted emotional or physical stress. It is one of the most frequent forms of pseudo angina. Death never occurs in the condition. The heart is, as a rule, small, frequently centrally placed and the condition is commonly associated with thyroid hyperactivity which is probably a part of the syndrome which is definitely to be classed as an endocrine disorder and not in any way circulatory in its basic pathology.

The cardiac pain in hyperthyroidism is on the contrary a quite different and much more serious matter, for in hyperthyroidism a true myocardial degeneration, usually a brown atrophy with ulti-

mate dilatation and decompensation appears. The pain produced, while it may under some circumstances simulate that in angina, is definitely that of a cardiac exhaustion and myocardial degeneration. The pain is rarely paroxysmal in occurrence, it is rarely transmitted in the characteristic manner seen in angina, it is associated with tachycardia and other evidences of hyperthyroidism.

Though in hyperthyroidism there is a very considerable degree of cardiac danger, especially in the latter stages when degenerations of the myocardium and muscle exhaustion with dilatation occur, there are other endocrine conditions which produce cardiac pain, independent of cardiac or aortic disease and entirely free from circulatory danger. The most frequent of these conditions is the cardiac pain which appears in the menopause. In this condition the concomitant association of emotional instability and of all manner of neurotic and hysterical phenomena is shown and error in diagnosis is rare indeed. It is sometimes difficult, though important for the physician to realize that such persons may occasionally also have a true cardiac lesion and a true angina superimposed upon their endocrine disturbances.

In practically all these disturbances which we have been considering the nitrites are entirely with-

out effect in so far as the relief of the pain is concerned. In fact, in most of them the nitrites augment the distress and the annoyance of the patient. This is in itself a suggestive diagnostic point.

CHAPTER VIII

TOXIC ANGINA

DISTINCT on the one hand from pseudo angina and on the other from true angina must be arranged a series of cases in which symptoms closely simulating those of a true angina appear, but which are brought about by definite chemical substances, and not from morphologic disease of the heart. Such are the conditions which are brought about by the abuse of tobacco, coffee, and tea. These forms of toxic angina differ in degree as well as in origin from cases of true angina pectoris. The writer has never seen any case of toxic angina which in any way approached in severity of suffering that which usually occurs in cases of true angina. Another very important differentiation exists between true and toxic angina. Cases of toxic angina rarely or never die, unless they are complicated by anatomical disease of the heart muscle, aorta, or coronary vessels which are quite incapable of having been caused by the toxins which produce toxic angina.

Not only are the toxic anginas produced by the excessive use of tobacco, tea, or coffee, but they also

appear in certain persons who are very susceptible to such substances, though never using them to excess. This sensitization is most acutely evident in tobacco angina, and it may occur spontaneously as the result of a natural sensitization toward that particular substance or as a result of prolonged over dosage.

The toxic anginas are also differentiated from the true in that in the toxic forms of the condition the symptomatic picture is produced solely by the one substance concerned. The writer has never seen a case of toxic angina in which the patient was sensitive to more than one of the three substances mentioned; nor do emotional or mild physical stresses induce the attacks in toxic angina.

The symptoms of toxic angina also are considerably different from those of true angina. As a rule they occur soon after the exposure to the specific material and they usually quickly subside when this substance has been eliminated, even temporarily. As a rule the pain is much less severe. It is usually limited to a sense of dull heavy pain in the precordium or epigastrium. It is rarely squeezing or boring in character and reflection, if present at all is much less wide and much less severe than in true angina pectoris. It rarely originates in aberrant areas as is so frequently the case in true angina pectoris.

Toxic angina is more frequently brought about

through the action of tobacco than from any other agent. Tobacco angina of greater or lesser grade is extremely frequent, and most chronic users of tobacco sooner or later experience it to some degree if they become excessive in the use of the weed. Albutt states however, (Lancet, May 5th, 1923) that tobacco angina in the intenser degrees is a rare condition.

The pain in tobacco angina is usually dull and aching more than sharp or lancinating. It is usually located in the precordium or in the epigastrium, and is not ordinarily reflected but if reflected it goes more frequently to the neck than to the arms or shoulders. Differing from the attacks of true angina pectoris, it commonly occurs not in paroxysms or seizures, but as a dull ache not only during smoking but particularly an hour or so after it. The attacks rarely are of such severity that the patient is required to give up his occupation, though he may be made very miserable because of the more or less constant weight-like pain in the precordium. This pain may last hours at a time, but during the pain the patient is rarely incapacitated and still more rarely is he obliged to go to bed, or to take powerful sedatives. When the pain is present, exposure to the fumes of tobacco either by smoking or by mere inhalation of smoke produced by another ordinarily much intensifies the suffering. Usually

even a few days' abstention from tobacco will give tremendous relief from the pain.

When the pain is present it is not commonly accompanied by any more intense anxiety than that of a natural fear as to the real nature of the pain, associated with a natural but not extravagant anxiety. The pain is usually moderately intensified by exercise and marked cardiac arrhythmia is frequently present, much more frequently than in true angina. Either a bradycardia or sometimes a tachycardia may be present; the arrhythmias may simulate almost any possible sort. As a rule the electrocardiogram shows a normal mechanism, and very frequently slight exercise such as walking "double-time," standing in place and similar tests for muscle reserve cause a resumption of normal rate and rhythm. Attacks of tobacco angina are very rarely precipitated by emotional or nervous stresses.

The aspect of the patient suffering from an attack of tobacco angina is rarely impressive or alarming. The writer has never known a patient afflicted with this condition to die except when there were found at autopsy lesions which tobacco is quite incapable of causing. There can be no question but that the use of tobacco by patients with true angina as a rule greatly increases the frequency of attacks, but in the opinion of the author, there is no similar basic pathology in the two conditions.

Tobacco angina usually promptly subsides when tobacco is withdrawn. Within a few days or weeks after tobacco has been discontinued the patient ordinarily will be entirely free from the symptoms and will be able to undertake physical stresses which certainly indicate a normal muscle reserve. The writer has previously reported the case of an eminent medical man, who had been an excessive smoker for years. After a period of some weeks during which the chief symptom was gradually increasing precordial pain which incapacitated him for his work, the diagnosis of a true angina pectoris was made. Serious disease of the heart muscle was assumed because of quite marked dyspnœa and marked arrhythmia especially on exertion. He was advised to give up tobacco and did so, suddenly ceasing from almost constant smoking during his waking hours to none at all, certainly the best way to stop tobacco in cases of this kind. Within a month the pain, together with the arrhythmia, had entirely disappeared, and he was able to undertake heavy work and the exercise of a hunting trip during which he carried his own pack and canoe, doing quite as heavy work as his guide. There has been no return either of the angina or of any other indications of cardiac disease, but after several years' abstention, he is still very sensitive to tobacco.

There is only one method of treatment effectual

in tobacco angina; that is the absolute cessation of the use of the drug. It is inadvisable to attempt to cut down on its use; once tobacco angina has developed an increasing sensitization appears to follow, as a result of which even small doses of the weed produce more or less distress. When patients are unwilling to give up entirely the use of tobacco, it is best to cease smoking entirely for a time, and then if so desired to resume it after a period of complete abstinence in a much more temperate way.

The writer has never seen tobacco angina from the use of chewing tobacco, nor from the use of snuff, though he knows no reason why it may not be so caused. Curiously, there is a marked difference in some cases as to the form of the tobacco employed. Certain patients get the effect only when smoking cigarettes, others smoke cigarettes with apparent impunity, but are unable to tolerate a pipe or even a mild cigar.

In the opinion of the author, little benefit is obtained from the smoking of denicotinized tobaccos in any form in so far as the relief of tobacco angina is concerned; the one thing to do is to give up tobacco at least for the time being, or to very greatly reduce its usage.

While the experience of the writer justifies him in feeling that the condition of tobacco angina is practically devoid of danger to life, he is positive

in the feeling that the use of tobacco in cases of true angina pectoris is very harmful, since it not only increases the number of attacks, but apparently also increases their severity.

Coffee is usually named as the second most frequent cause of toxic angina. The experience of the author has led him to feel that coffee is much less frequently concerned in this condition than is popularly supposed. He has known physicians to make the diagnosis of a coffee angina, and having refused the patient further use of coffee, has administered caffeine, theophyllin, and euphallin to counteract the supposed symptoms. Such treatment seems incoherent and inconsistent at least. It is however quite possible that other bodies than these chief ingredients of coffee are responsible for the symptoms of this form of toxic angina.

There can be no question but that certain individuals are very sensitive to coffee, and that they may develop as a result of the taking of the drink, precordial pain, but the author has never seen an instance in which coffee angina has sufficiently resembled true angina pectoris as to cause difficulty in differential diagnosis. Coffee is much more likely to produce symptoms of gastric distress, of "indigestion," in which naturally the gastric distress may be assumed to be of cardiac origin. Neither has the author ever seen cases of true angina directly

unfavorably affected by the temperate use of caffeine or of coffee except that the decoction, like the caffeine itself, may cause over-excitability of the nervous system, and so badly influence the complex. Occasional cases of true angina are apparently benefited by the temperate and proper use of coffee, just as pharmacologists have shown that drugs of this class have a beneficial action on coronary circulation. The writer feels, however, that the over stimulation of the nervous system produced by many of the drugs or preparations of this character renders their employment unwise in many instances.

When any doubt exists as to the relationship between the taking of coffee and precordial pain, it is a very simple matter to discontinue the coffee. No harm is certainly done by the restriction, and if the pain is brought about by the use or the abuse of coffee, or if on the other hand, the distress of a true angina is intensified, it should promptly decrease when the coffee is discontinued, for these substances are all very quickly excreted.

The writer has seen no case in which the use or abuse of tea has produced symptoms similar to those of a true angina pectoris. Such cases are not however very infrequent in the literature, and he has received personal reports of such cases from entirely credible sources which leave no doubt in

his mind but that the condition may occur. It can not however, be very frequent, nor can the attacks approach those of the true angina either in gravity or frequency. The writer has in the past frequently recommended the use of tea or coffee as a temperately taken beverage in angina pectoris as he believes with symptomatic benefit, just as he has used caffeine, euphallin, and theocin in other circulatory disorders. He feels that in those instances in which the patient is very susceptible to the stimulative effects of these substances, they frequently work harm rather than good, particularly if these agents are taken at night.

Many drugs, among them strychnine, if given to unusually susceptible persons or in too large doses, may cause cardiac pain, which can be easily confused with that of a true angina. Certain articles of diet which cause gastric distress, such as the more active condiments may cause distress of gastric origin which may be reflected upward and so give rise to symptoms which are readily mistaken for angina. It is to such conditions that the writer is persuaded to attribute most instances of so-called toxic angina. The writer does not desire to be dogmatic or narrow minded on this question, but he can only reason from his own experience and observation. Conversation with many medical authors

has led him to feel that too frequently in such matters, one writer copies from the statements of other well-known men who in turn may have resorted to similar authorities rather than to their own personal observation.

CHAPTER IX

DIAGNOSIS

THE diagnosis of angina pectoris in characteristic instances is relatively easy. To a very considerable degree, it has already been discussed in a much more detailed way than is called for under this heading for the chief difficulties lie in the way of differentiation from the conditions which have just been discussed under the heading of pseudo angina. Because of the serious prognosis which must necessarily go with the diagnosis, the writer feels that snap diagnoses are unwise in the presence of this pain, but he feels also that the patient should be given in most instances the benefit of the doubt and that a tentative diagnosis of angina should be made in most of the simulating conditions until exclusion is possible by differential study and particularly by observation and study of the individual instance.

The chief points of an absolute diagnosis are as follows:

The character and appearance of the attack. It is paroxysmal, the pain is severe, substernal or precordial in location, and as a rule reflection is

present. This reflection is most commonly into the left shoulder, arm, forearm, and to the fingers. The reflection may involve the right upper extremity instead of the left, or both may be invaded, or the pain reflected into the neck, lower jaw, or to widely distributed areas. The pain is severe in degree and is accompanied in even relatively mild instances by a sense of fear or apprehension out of proportion to the calm judgment of the patient in his uninvolved moments.

Except when coronary thrombosis has taken place, partial to complete relief is afforded by the giving of the nitrites. Purely sedative or soporific drugs are required in very large amounts to still the agony of the true attack. Associated with the pain are symptoms of shock of varying degree.

During the immediate attack, other symptomatic evidence of true cardiac lesions may be largely wanting; notably is this true of dyspnœa, cyanosis, œdema, and swollen or tender liver.

The personality and credibility of the patient must be well weighed in arriving at the final diagnosis.

Electrocardiographic evidences of coronary disease or of lesions of the heart muscle may be found. Failure to discover such changes does not contraindicate the diagnosis, but when found in typical form, the electrocardiogram in association with

characteristic symptoms may be considered as absolute.

The physician should not hasten to an absolute diagnosis of angina pectoris; rather he should not hasten to make a diagnosis excluding this possibility. The more intimate the knowledge of the patient which the physician possesses, the more able he is to make a final diagnosis of this condition which is in so large a part based on purely subjective findings. An absolute diagnosis should not be made without the physician seeing the patient in a paroxysm of the disease, for of all the diagnostic findings, the most conclusive is the appearance of the patient during an attack of the syndrome. Careful as the physician should be in the diagnosis of this condition, it is even more important that he exert the greatest possible caution and care in the exclusion of the diagnosis.

No case should be diagnosed as an instance of angina pectoris which is not supposed or shown to have lesions of the aorta, coronary arteries, or heart muscle which are in themselves dangerous to life. Diagnosis should not develop therapeutic pessimism on the part of the physician.

CHAPTER X

ELECTROCARDIOGRAPHY

IN A condition such as angina pectoris in which the pathology is so discursive, it is to be expected that reports as to diagnostic electrocardiographic findings should be also very indefinite, and to some extent this is true. All physical signs of angina pectoris may be wanting and yet the diagnosis must be made in those instances entirely on symptomatology. It is therefore to be expected that in not so very infrequent instances the electrocardiographic findings may be also entirely negative and may indicate absolutely no disease whatever. The writer saw a case of suspected angina in which repeated electrocardiographic records had been made, none of them showing anything which could be considered as definitely abnormal. While this patient was in the office under examination, he developed a seizure during which a cardiogram was made. It was again entirely normal, yet this seizure went on to a coronary thrombosis from which the patient died within a few hours.

The negativity of electrocardiographic findings in cases of angina may then signify nothing. It

certainly does not exclude angina, though in the opinion of the writer it lessens the probability of this diagnosis; for in the vast majority of instances the electrocardiograph does show changes suggestive of a pathology capable of causing the angina. It is certainly a very valuable method of examination in at least all disputed instances. Positive findings are highly confirmatory when present, and when associated with a symptomatology or physical findings indicative of angina. Negative findings are not positive in either direction, but at the same time they must be accepted as lessening the probability of real cardiac pathology.

In the observation of the writer, cases depending on coronary lesions are more likely to give electrocardiographic evidences than those instances in which the basic pathology seems lodged in the aorta.

According to Pardee about 70% of coronary cases will show one or more abnormalities, even though the heart is quite normal in size and free from murmurs. Half of these will have the special T wave peculiarly associated with coronary narrowing. Pardee states that in coronary occlusion some patients show a peculiarity of the T wave; the characteristic appears to be the upward convexity of the S-T interval. The T wave in lead III, or more rarely in lead I is found to be large and turns

very sharply downward after a brief upward curve. Lead II also has a downward T, and may or may not show this typical upward convexity in the S-T interval.

In cases in which the fault appears to lie in the myocardium, though perhaps remotely dependent on coronary changes, the cardiogram may take on very divergent pictures, any of which may be indicative of myocardial disease; and the same is more or less true of instances in which the chief defect appears to be lodged in the aorta.

Various cardiographers naturally differ greatly in their diagnostic estimate of the findings indicative of coronary or muscle defect in angina. F. A. Willius (Arch. Int. Med., Feb., 1921) states that there are no pathognomonic electrocardiographic findings. Significant T wave negativity occurred in one-third of his electrocardiograms, and it is an important abnormality. Abnormalities of the T wave contour affecting both the positive and the negative waves are significant. Abnormal QRS complexes in all the derivations of the graphs occurred in 14.2% of the records.

From the clinicians standpoint the writer feels that almost any electrocardiographic finding which indicates in so far as the cardiogram may, disease of the heart muscle or of the coronary vessels is to be considered as highly confirmatory evidence

when the clinical aspects of the case suggest angina. In the absence of the clinical evidence of angina, also the writer has been forced to feel that serious consideration should be given to positive cardiographic findings even in the absence of clinical evidence, if these findings are of sufficiently definite and constant character. Negative findings are to be considered clinically as merely negative findings, and much clinical weight must not be attached to them.

BODLEIANA BIBLIOTHECA

CHAPTER XI

PROPHYLACTIC TREATMENT

"Quiet, warmth, and spirituous liquors help to restore patients who are nearly exhausted, and to dispel the effects of a fit which does not soon go off. Opium taken at bedtime will prevent the attacks at night."

—HEBERDEN.

ALL students of the condition recognize the fact that angina pectoris is definitely a family disease. Every physician has or has had among his clients, families in which angina pectoris is the characteristic disease, and in which at a certain age, members, especially the males, develop the syndrome. Sometimes, it is true, members of these families die of other cardio-vascular conditions, of hypertension, arteriosclerosis, of the cardio-vascular-renal complex or of fibroid, or other degenerative types of cardiac or vascular disease.

It is extremely important that this definite familial tendency be recognized. It is quite possible to delay, mitigate or to eradicate entirely this tendency by careful supervision of the early and adult

life of individuals with this definite family trait. Those families which show a tendency in this direction associated with obesity, for example, may largely rid themselves of it by careful study and control of the life of its young members. The diet may be so devised as to eliminate in early life those foods, such as the starches, fats and sweets which tend to the abnormal deposit of fat. While we recognize that we may not entirely eliminate family inclination in the direction of obesity, much can be done to hold this tendency within bounds. Tendencies toward endocrine imbalance as in hypothyroidism, dyspituitarism and the like may receive early medication tending to lessen this factor also. Thyroid may be given from time to time in small amounts. Iodine has a definite deterrant effect, but most important of all, family tendencies in the direction of selection of foods particularly rich in fat building elements may be corrected. It must always be remembered that the most important thing in order that defects in diet may not occur is to give a well balanced diet, sufficient in all its elements to support not only life, but also adequate efficiency and well being, reducing in quantity rather than in quality, for the latter is often attended with dangerous results on the body economy. Iodine given in sufficient amount either as iodized salt, or iodine in the form of occasional courses

of Lugol's solution, or the sodium or potassium salts may help in this direction materially.

Adequate physical exercise, always short of excess, which leads in later life to an accentuation of the tendency toward fat deposit, should be insisted upon. It must never be carried to such an excess that an abnormal craving for food develops, which always works disaster in latter life when heavy physical work is ordinarily not possible and the dietetic desires and habits of early life as a rule persist. On the other hand it is very important that habits of adequate physical activity be encouraged, but never to the point of excess and the children of such families should never be allowed to enter competitive athletics in particular. Walking, golf, swimming, horseback riding, fishing, hunting and the like should be encouraged. It will be found in adult and latter life that these outdoor pleasures do much to fend off the very tendencies which lead on in the forties, fifties and sixties to the development of angina.

Such habits of vacation, of soul diverting pleasures, which induce the aggressive young business or professional man to desert the stress of professional and business life for mild diversion in the open will materially decrease his tendencies toward angina, and afford for his emotional and nervous tension an outlet which not only will last through-

out lifetime but which will not at the same time overstress his physical capacities as he grows older.

These young persons must also be particularly protected from the infections. Early evidences of rheumatism should in particular be combatted by the prompt removal of probable sources of this infection. The nasal condition should be put in as normal a state as possible, the tonsils should be removed when any suspicion of their being pathologic arises. Obviously adenoid tissue anywhere when it shows evidences of pathologic change should receive proper treatment. Slight evidences of chorea must be early treated. Attacks of bronchitis and influenza in particular should be promptly recognized and treated. Following any of the infections, especially after measles, scarlet fever and the like, a period of convalescent rest should be enforced more insistently, if possible, than in children with a perfectly normal heredity. The child should be immunized against diphtheria and against typhoid fever. Habits of healthful sleep should be developed. Emotional control should be educationally enforced and the child should from his early youth be impressed with the advisability of temperance in all things, especially in the use of tobacco and of condiments. Pyelitis, if it develops, should receive more than ordinary attention.

By measures of this character there can be no

question but that we are able to materially lessen the familial tendency toward cardiovascular degenerative diseases and a far more than ordinary resistance against the usual infections, which also lead to cardiovascular degeneration can be developed.

In the college years the child of anginal parentage should not take up competitive athletics of such character as football. He may safely play baseball. He should not try for the crew, but he may go in for canoeing. Running, tennis, the high jump or pole vault and the hammer throw are not for him, but he may be permitted to excel in golf, in trap shooting, fly casting and the like. He may achieve his college social standing through education in music, in acting or writing and in general while he must not be permitted the stressful athletic competitions he should not be denied the privilege of acting the part of a man among young men. The writer feels that to be a "mollycoddle" is worse than to have angina and his entire sympathy is with the young man who at this virile period of his life is denied the opportunity of playing his full part in the preparation for life as youth sees it.

The selection of life occupation is another matter of crucial importance. In the opinion of the writer the family doctor has not in the past suffi-

ciently entered into this most important matter. Our government has emphasized this desirable supervision in a most definite way in the selection and training of its candidates for the army and navy, and even in the selection of young men for the diplomatic service. In all of these occupations the candidates are not only selected after careful and repeated physical examinations but their training throughout is so carefully managed, notably at West Point and at Annapolis, that very few men in these services break from cardiovascular lesions, at least at the period of life when they are of greatest value to their country and to themselves.

It is a most common experience to find, especially in our technical schools, young men preparing themselves for work which from the nature of their family inheritance and physical equipment they will never be able to continue successfully for the expected period. We of the medical profession are as blameworthy in this respect as schools of engineering, and every teacher of medicine finds all too frequently young men preparing for medicine who are physically and emotionally unfitted for the well recognized requirements of this work. Many splendid young men are ruined for their life calling by this negligence in selection.

Such restrictions apply with particular pertinence to young persons with a family tendency toward

cardiovascular disease in their selection of an occupation. Young medical men anxious for surgery, for obstetrics or general practice, for military surgery and the like, for example, should be wisely diverted into those branches of the work which require less physical stress and less prolonged emotional tension, such as research medicine, ophthalmology, pathology, hospital service and the like. In the engineering professions, men with a cardiovascular family history should be directed similarly into those branches of the work which do not entail exposure, great physical stresses and similar routine which is particularly serious for the sufferer from cardiovascular defects and especially from angina pectoris.

Once a selection and preparation for life work has been made, the physician should in instances in which cardiovascular defects are becoming manifest, or in those individuals born to a heredity in these directions, be prepared so to advise his client, not yet a patient, how he may still carry on his work in an adequate way to justify success, and yet without jeopardizing his future welfare from angina or other cardiovascular defects of like character. The person must be advised to secure adequate rest, to sequester time if necessary so that adequate sleep may be obtained and at the same time and of equal importance, habits of beneficial

diversion and relaxation should be contracted. This may mitigate the stresses of his calling to such an extent that he may be able to evade, if fortunate, the familial disease and to live out in usefulness and pleasure a fully successful life.

The growing habit in America of taking vacations is a step in the right direction, but in many instances some of these patients and men select for their vacation and diversion things which impose more strain than their habitual occupation. The physician will find on any golf course men the subject of angina or other cardiac disease who, under the delusion that they are benefiting their health, all untrained and unprepared from a physical standpoint, indulge in stresses and strains fitted for the young college athlete, but not for the mature, and frequently overly plump business or professional man who, six or seven days out of each week, sits at the desk, stands on the turbulent floor of the stock exchange, or under the tremendous stress of the operating room. Old men try to play tennis with their sons, to defeat them at handball, to outdistance them in swimming, to outclimb them in the mountains: many of them develop angina.

The experience of the writer has persuaded him that such practices as he has just mentioned are among the most frequent causes of angina in men who have passed the forties. Prophylaxis on their

part on medical advice would be a very simple matter, if we were courageous enough to assert our professional knowledge.

Only physicians and coroners know how very commonly angina and coronary thrombosis occur in middle aged men as a result of intemperate sexual indulgence. It is a common joke with the laity to paraphrase Solomon: "The way of a (old) man with a (young) woman." One of the most frequent immediate causes of death from angina is from over sex indulgence, and until we discuss this subject more courageously with our patients and more frequently among ourselves, we shall continue to neglect one of the most important prophylactic methods of treatment in angina pectoris.

CHAPTER XII

SPECIFIC TREATMENT

ONCE angina pectoris has developed in any patient the physician should make the most careful study possible of that patient and of his habits, obligations, ancestry, and of his past history. The great object to be obtained if possible is to find the cause and if possible to eliminate it before irremediable lesions may have developed. The early recognition of a syphilis should be tantamount to vigorous treatment along specific lines. Weil, Stokes and the author among others have long emphasized the importance of initiating such treatment of this and of other forms of cardiovascular syphilis with the iodides and mercury or bismuth as an initial treatment. Arsphenamin and similar intravenous arsenicals are dangerous agents to employ in active cardiovascular syphilis until the patient has at least once been brought well under the effects of the iodides, mercury or bismuth. Many early cases of angina pectoris caused by syphilis may be absolutely and entirely cured by prompt and appropriate treatment. Most cases of angina pectoris caused by syphilis may be benefited at least

symptomatically by specific medication. The writer has, through the kindness of his colleague, Dr. Joseph Conley, recently studied a characteristic instance of syphilitic angina pectoris. The patient was a traveling man in the early thirties. He had been suffering for the previous three or four months from increasingly severe attacks of precordial pain reflected into the left shoulder, down the arm, forearm and into the fingers of the left hand. The attacks were precipitated both by emotional and physical stresses and he had been obliged to give up his work because of the increasing severity and occurrence of the attacks.

During the initial examination in the office of his family physician he developed an attack which Dr. Conley at once diagnosed as true angina. In addition to the severe precordial pain, shock and tremendous agony, during the attacks the heart rate fell to a regular rhythm 38 to 40 beats per minute. Heart block was diagnosed but no electrocardiogram was available at the time. Fluoroscopic examination showed a normal sized and shaped heart but there was definite widening of the first portion of the aorta. He at first denied the history of syphilis and the Wassermann reaction was negative.

He had been married three or four years and while his healthy appearing wife had several times

become pregnant, in each instance she lost the foetus in the early weeks of gestation. Her Wassermann reaction was found positive and she showed dermal and mucous membrane evidences of the disease. When these facts were presented to the patient he then confessed that he had contracted lues some three or four years before his marriage and that he had been vigorously treated for a time, but with the disappearance of disfiguring lesions he had ceased treatment.

He was promptly put to bed and notwithstanding the fact that he was spared every unnecessary effort he continued to develop very severe attacks of angina generally coming on during sound sleep. The stress of an ordinary gentle bed examination sufficed to send him into a severe paroxysm and an apparent heart block. Nitrites gave early and good relief, but they failed utterly in so far as prevention of the attacks was concerned. He was put on the iodides and mercury in heavy dosage, but he experienced no relief of a substantial character for two months, after which time he received tremendous relief. The attacks of angina disappeared, the heart block disappeared, and a week or so later he was again able to get up and to return to his work, but the specific treatment was nonetheless continued to the full point of drug toleration. Within three months from the time of

diagnosis he was returned to his work; he was able to do full duty without further angina, dyspnœa, arrhythmia or any unusual cardiac fatigue. Naturally the specific treatment will be continued and after a few months longer under the mercury and the iodides, he will receive arsenic intravenously.

Where scar tissue, gumma or arterial closures have resulted in the aorta, in the coronaries or in the myocardium, cure is not to be expected, though it occasionally occurs. In nearly all instances, even of very long standing, considerable mitigation of symptoms and decrease in frequency of attacks follow properly administered specific treatment, associated of course with correct general treatment of the angina. Von Neusser cites Fornier, Vitorco and Hallopean as curing cases by specific treatment.

To a much more limited degree the same may be said of angina pectoris caused by gout. The writer has previously reported a case of very severe and frequent angina which occurred in an overly fed and emotionally active, physically vigorous broker. The attacks had become so severe he had been obliged to go to bed. The seizures were so frequent and agonizing that he required almost constant morphine administration to make life tolerable. His reactions were so acute that even a nurse

walking across his sick room floor was sufficient to incite an attack. Physical examination, except when he was under the effect of morphine, invariably brought on attacks. He showed typical active tophi of the ears and very characteristic lesions of the fingers and toes. He was not only a tremendous worker in an exceedingly stressful occupation, but he was also a large drinker of champagne and a highly cultivated gourmet. He was put upon atophan and colchicine in large doses. Within seventy-two hours he had experienced great relief and in a week he was out of bed and able to walk without inducing paroxysms. He was eventually able to return to his work and to do full duty. Meantime he was on a strict anti-gout regime. After a few months of complete relief he returned to his customary habits of eating and drinking, with the result that the angina also returned and with much less prompt relief under anti-gout measures and atophan.

As a rule colchicine given in full doses is more efficient in the hands of the writer, but others report better results from atophan, cinchophen and from the alkalies. A strict adherence to anti-gout measures is imperative. While as a rule considerable benefit may follow treatment of this kind in gouty cases, startling benefit is not the usual fortune of the author, especially in long-standing cases

of gout where presumably permanent lesions or scars have appeared, still the use of anti-gout measures in gouty cases is imperative for the maximum result in cases caused by gout.

In cases of angina developing in the course of acute rheumatism a considerable amount of relief is occasionally afforded by the salicylates. It is, however, usually nécessary to administer the salicylates in large doses and while many cases will tolerate adequate doses of the salicylates by stomach, there are many more instances in which only rectal administration is possible in adequate dosage which may vary from 120 to 300 grains per twenty-four hours.

The author had for a time under observation an eminent colleague who suffered severely from anginal attacks, and his most satisfactory relief was afforded for a long time by the use of tremendous doses of salicylate, self administered, and usually taken by stomach. This physician would take as high as 300 grains daily. Meantime he was carrying on an active operative practice. He tolerated such doses with apparent impunity, and for several years was able thus to afford himself relative comfort. He finally died, apparently from a coronary thrombosis but several months after the salicylate had ceased to be effective. He was the subject

of an aortitis, a myocarditis, and of a final coronary thrombosis.

While undoubtedly the salicylates are effectual in acute instances of angina of presumably rheumatic origin, the author has not been impressed with their value in chronic cases of rheumatic heart in which angina had developed, but in such cases the drug should undoubtedly be tried out.

A colleague has reported to the writer an instance in which attacks of apparent angina pectoris followed a case of diphtheria. The diphtheria had not been specifically treated until relatively late in the disease, due to the fact that its specific nature had not been recognized as such. The attacks completely subsided under three weeks rest in bed and the intravenous administration of full doses of diphtheria antitoxin. Four weeks later fluoroscopic, physical and electrocardiographic examinations of the heart showed no apparent lesions present. The writer has thus far never seen an instance of acute angina in the course of a convalescent diphtheria, unless this instance cited is such, but he sees no reason why angina might not so develop, nor why benefit from specific medication might not occur in such instances. It would certainly be the natural thing to do.

So exceedingly important does the writer consider rest in the treatment of all cases of angina

pectoris that he wishes to discuss it here under the heading of Specific Treatment. In effect and in directness of rational it may certainly be properly so considered.

Rest is the most important general measure to be employed, especially in those instances of angina pectoris which develop as a result of any of the acute infections. It is quite as important as in certain of the chronic or subacute infections, notably such as typhoid fever. Rest is the most effective measure in all the acute infections, notably after scarlet fever and influenza. As a general rule physicians do not sufficiently appreciate the grave cardiovascular damage which is likely to occur after the acute infections, and no doubt considerable blame should be attached in some of the cases of angina pectoris which develop after the acute infections where this fact has not been adequately recognized and the parent accordingly advised.

Rest as a means of treatment in this complex receives perhaps its most logical justification on a physical basis. Many cases of the syndrome are precipitated almost exclusively by exercise or by physical stress. In such naturally, the most sensible method of treatment is rest. The more absolute the rest at the onset, the more desirable. This should particularly pertain to the special physical stresses which in that individual appear most fre-

quently to precipitate the paroxysm. In acute cases and even in many instances of very long standing, absolute confinement to bed is the logical procedure. This should be continued until the patient is able to sit up without the development of a seizure. In all instances a return to the usual physical stresses of life must be accomplished most gradually and the patient must be warned that very slight symptoms must be reported in this progressive relief from rest, so that the method of treatment may receive its full value. While the patient is in bed, undergoing the rest phase of cure he must avoid any effort which he finds causes the return of even the most mild symptoms. He may not be allowed to turn over in bed without assistance. He should at least in the early phases of this treatment use the urinal and the bed pan. Later on, these may perhaps be replaced by the use of a convenient commode arranged at the height of the bed on to which the patient should be assisted by the nurse or attendant. This privilege, small as it may seem to be to the well man, is a great and much appreciated concession to the spirited patient who usually abhors the bed pan and its attendant humiliations more than any other restriction which is imposed upon him. Satisfactory and comfortable emptying of the lower bowel in the supine posture is at best a difficult matter, and especially so when

the patient is warned against undue straining. The writer has found it wise in most instances, especially in previously healthy individuals, to substitute the commode as soon as it is permissible. Active and passive movements, massage and the like serve to prepare the patient for the time when he is allowed to sit up in bed, or in a chair by the bed side. Slowly he is allowed to increase his activities; the chair is moved a few steps from the bed, across the room and so on, until he is permitted to walk perhaps to another room and to go out of the house to take rides in the quieter parts of the day, and so on up to the time when he is permitted to go as it were on his own responsibilities.

The physician must recall in respect to this method of treatment, the social and economic obligations of the patient. Most patients have obligations which sooner or later become equally important as life itself, and a return to the office or study, or even to mild physical methods of work sooner or later becomes imperative for most persons.

The writer recalls a patient, a relative of a well known physician who had been kept on absolute physical rest in bed for three months, during which time little improvement had taken place, the attacks still occurring at frequent intervals. A very frank talk with the patient revealed the fact that

his business affairs were in a most critical condition, requiring his attendance at his office, if but for a very few moments daily. He felt that but a few moments daily at the office would suffice to avert a most serious financial condition for his family who, should he die then, would be left in helpless penury; while if he were able to adjust his affairs, they might possibly be indefinitely assured the comfort and luxury to which they were accustomed. Realizing the unhappiness of the patient, and the sinister effects that this state of mind imposed on him, he was at once permitted to be carried to his car, driven to the office where he was carried to his desk, and allowed twenty minutes conference with his business staff. No attack took place, and on the following and for many succeeding days, the same effort was permitted, with but occasional slight attacks occurring. He was allowed to increase these excursions until he spent daily three hours at the office, returning quietly to a period of rest in bed before his chief meal. With slow and most cautiously considered increase in his activities, he gradually again assumed full control of his business affairs, recouped his fortune and after some ten years of careful and relatively happy life, he died, as he had wished, at his desk, "decently and as a gentleman should," leaving his family well

provided for and his business in a prosperous condition.

Instances such as the above serve to emphasize the tremendous rôle which emotional rest and nervous quiet play in the rest treatment of this condition. The physician must realize that for some individuals the highest degree of rest is obtained not necessarily in bed, but under greater activity that theoretically should not be permissible. Throughout the rest treatment, this phase of the question must receive dominant consideration. An annoying or disagreeable nurse, too frequent visitors to the sick room, too much solicitude on the part of relatives and attendants, may suffice to entirely defeat the whole purpose of rest, and rest may frequently require more of nervous and emotional factors than merely physical ones. All this, the astute physician must divine for himself, or discover by discreet experimentation. In this, as in few other diseases, emotional and nervous rest are almost equally important with physical relaxation.

Rest of this character frequently requires the discreet use of drugs, a fact which Heberden realized fully, but which the ultra-scientific attendant may unwittingly fail to comprehend. The bromides are truly invaluable in some such instances, chloral hydrate, particularly given at night, luminal, and

as mentioned by Heberden himself, opium given at night may become imperative especially where the rest is otherwise broken, and when night attacks occur as they so frequently do.

Sleep is the acme of the rest treatment of angina pectoris, and it must be accomplished, if need be by the use of drugs, though physical and psychic treatment, if skillfully applied, usually suffice. Content and happiness are agents of the utmost value in this phase of the treatment of angina pectoris, relaxations and diversions are to be assured in so far as possible. The use of the radio in the sick room, music, reading, writing, quieting games, are all to be used as therapeutic measures, each well adapted to the likes and needs of the patient. It is a shame to the medical profession that in some respects non-medical persons cater more adequately to such methods of treatment than we do. The physician interested in the relief or cure of his patient will do well to consider carefully the whole life needs of his patient, especially in instances of angina pectoris. Such means add immeasurably to the production of rest, which is the needful key-note in the successful management of this condition. With proper handling, many patients of this status may live out long, useful and often happy lives, despite their handicap.

CHAPTER XIII

GENERAL TREATMENT

A STUDY of the food requirements of patients suffering from angina pectoris is a very important matter. In practically every instance this depends almost entirely on the individuality of the patient, or on other conditions which lie perhaps far distant from the syndrome itself. There is no diet for the complex of angina pectoris, but the best diet for the person suffering from the angina may be a most critical matter.

As to the character of foods to be prescribed, this must rest elementally on a basis of those which the patient has himself found most easily tolerated, or it must be determined by other basic conditions associated in the complex. Thus in patients with angina pectoris who suffer from diabetes mellitus, the diet must necessarily be based on the diabetes; the same is true in gout, complicated by angina, in nephritis, and so on. Obviously also, some concession and interpretation must be given to the national or personal food habits of the patient. One can not expect to feed a Spaniard on the diet commonly acceptable to a man brought up in New

England, nor vice versa. Sufficient food must be furnished to supply the demands of the particular individual and his dietetic needs, and the form and type must at least in some degree conform to the desires and likes of the patient; otherwise he will not take sufficient nourishment.

In nearly all cases overloading of the stomach will suffice to induce seizures, and obviously this is not permissible. As a rule it is best to give, if necessary, small feedings; and the heavier meal, where regular periods of feeding are observed, should be given at midday. It is very unwise to allow the patient to lie down soon after taking food, if the food taken has been sufficient to distend the gastro-intestinal tube to the usual degree. Attacks are very commonly brought about by this custom, and most intelligent patients soon learn that they can not tolerate large feedings, especially at night or nearing bedtime.

Similarly foods which from their bulk or character cause undue distention are to be avoided. Bulky vegetables such as cabbage, beans, salads, and the like, are to be used only experimentally, and then only if it is found that they do not cause discomfort when taken in the desired or permitted amounts.

The same general precautions are to be observed as regards the fluid foods. As a rule, milk taken

as the major element of a diet is not desirable because of the large gastric clot which it forms. Neither should any fluids, even water, be taken rapidly, for distention of the stomach or oesophagus may induce an attack. Either very hot or very cold fluids should not be taken except in such small amounts that the temperature of the fluid when it reaches the stomach has become standardized to about that of body heat. Most patients quickly find for themselves the importance of this consideration.

The alcoholics have been highly recommended by most serious students of the condition, used of course in suitable individuals and in proper forms. Heberden called attention to the fact that "It helped to restore patients who are nearly exhausted, and to dispell the effects of a fit which does not soon go off." Its therapeutic effects as a sedative have been universally observed in properly selected cases, and it is of especial value in elderly persons both because of its quickly realized nutritional value, and its entire lack of stress on the metabolism, and its quick absorption. There is no doubt also as to the desirable euphoristic effects of the alcoholics in suitable persons. It is useful also for its carminative action. Large amounts of alcohol, such as are sufficient to produce a certain grade of anesthesia, are beyond doubt of value in severe attacks of the disease. It is also possible that it may

have some favorable action because of its vasodilator effect on the coronaries when given more or less as a matter of routine.

Some recent literature on the subject has asserted the undesirability of sodium chloride in the diet of sufferers from angina pectoris. Except in cases in which a deficient excretory ability to handle this salt exists, there is no real, rational basis for this prejudice. Salt is of course inadvisable except in very small amounts in cases which have a hypertension, or in which oedema is present. It probably has no bad effects except in conditions of this nature.

Similarly, tea and coffee are interdicted by some therapeutists apparently on grounds of general prejudice, for it has been shown very certainly, that in most cases caffeine, and the large group of like drugs notable among which is euphallin, cause relative dilatation of the coronary arteries, and thus are supposed to exert a favorable effect on the complex. There is however a very real basis for the interdiction of tea and coffee in certain cases, either in which it has been found by experiment that these condiments tend to induce attacks, or in which these substances increase the emotional and nervous excitement of the patient, or cause insomnia and emotional unrest. It does seem absurd, as is so frequently done, to forbid the use of tea and coffee

which are very agreeable to most patients, and at the same time to give caffeine, theophylin, euphallin, and the like, which are but the active principals found in these beverages.

As a general rule, the writer is prepared to say that tobacco should be forbidden in cases of angina pectoris. The author does not believe that tobacco has ever caused or can cause the basic lesions which develop true angina pectoris, and yet his clinical experience has been such that he believes that tobacco in most instances, sensitizes the patient to attacks, and that it increases the severity of the seizures when they do develop. This refers not only to the heavy usage of tobacco, but also to those not infrequent instances in which persons are so sensitized to minute doses of the drug that unfavorable effects are produced by even infinitesimal amounts. There are however exceptions. The author has observed cases in which the use of tobacco apparently mitigated the seizure very materially, much as stramonium acts in bronchial asthma. Obviously in such cases its temperate use should be permitted. This again emphasizes the point which the writer feels can not be too frequently stated, namely, that each individual case of angina is a problem in itself.

The bowels should be kept freely open in subjects of this complex. The reason for such a statement is apparent, for any unnecessary distention

of the gastrointestinal tube tends to precipitate the attack. It is also highly probable that the absorption of products of abnormal intestinal fermentation such as must occur in constipation is undesirable on general principles. Attacks of acute seizure are frequently precipitated by straining at stool; this should be made unnecessary by proper attention to the bowel.

The author has already mentioned the frequency with which attacks of the most acute nature are precipitated by coitus, this is one of the most frequent immediate excitors of death in the anginal status. It is also brought about by sex excitement, and the patient suffering from angina should be cautioned against anything beyond the utmost temperance in this respect. It must however be admitted that normal gratification of the sex instinct at reasonable intervals is probably less dangerous than severe ungratified desire extending for long intervals. The patient should himself be appraised of the real facts in the case, and he will therefore, thrown as it were on his own responsibility, more correctly solve his individual requirements in this regard than if he is permitted to throw this responsibility on his physician.

Most cases of angina pectoris are almost wholly wanting in the evidences of definite cardiac disease. In such instances the complex is almost exclusively

a symptomatic picture only. Notwithstanding this lack of evidence of elemental cardiac defect, nonetheless all these instances are subject to the same likelihood of sudden death, as those cases in which there has been preexisting evidence, physical or symptomatic of cardiac defect. It is these cases in particular, which are called to mind, and which terrorize both physician and patient when the diagnosis of angina pectoris is suggested. The writer recalls in particular an instance of this kind which occurred in his professional youth. A lady, a neighbor and friend, in the late forties, one afternoon called the writer to her bedside. She was apparently in sound health and good present condition, but she gave the history of having suffered for some months from severe precordial distress, developed after exercise, under emotional stress, and frequently from no discernable cause whatsoever. She stated that she was then suffering very severe agony from the condition. Notwithstanding this statement, she had an apparently excellent pulse, she was not cyanosed, nor did her general aspect confirm her statement of great suffering. After looking her over to the best of his ability, the writer told her that she was probably suffering from an attack of pseudo angina and he went to some extent in explaining to her the innocent nature of this condition. The patient responded that this was un-

necessary, for the reason that the elder Dr. Janeway had made the same statement and explanations but a few hours before. Feeling secure in his diagnosis therefore, and somewhat elated at his own cleverness in arriving at the same diagnosis as that of his revered chief, the writer gave her a hypodermic of water with the patient's understanding that she was receiving, as she should have received, morphine. Within a few hours she peacefully, but absolutely died. In talking over the occurrence with Dr. Janeway, this truly great man quaintly stated that when he disregarded the patient's story, and relied solely on his own calm judgment, he usually made a mistake.

The absence then of any physical confirmation does not therefore exclude grave circulatory defect. The physician should not be afraid to give treatment in suspected cases even though he is not absolutely sure in his own mind as to the correctness of diagnosis. In such instances, the experimental use of the nitrites and of the sedatives may often leave the physician's mind in doubt, but he is not so likely to needlessly leave his patient in suffering. The vasodilators are to be employed as a matter of routine in cases in which the complaint of frequent attacks is given, even in instances in which no hypertension is evident. The writer however, does not feel that this is advisable practice in those

frequent instances in which a marked hypotension is present during apparent attacks; direct sedatives, as the bromides, paraldehyd, opium, or morphine should be given. For those cases in which a constant or frequent tendency toward paroxysms is shown, the writer feels that the best effects are obtained by the more or less constant exhibition of sodium nitrite or of erythroltetranitrate. Chloral hydrate acts very well in many of these instances, and the systematic use of the bromides or of luminal and other similar sedatives is wise, at least until the clinician has become from observation entirely convinced that they fail to improve the condition.

Where the patient complains of, or is shown to have, dyspnœa, œdema, cyanosis, or when he gives other evidence that a real gross cardiac defect is present, even in the absence of definite physical findings and always in the presence of positive lesions, he should receive treatment according to the lesion present, irrespective of the presence of angina. This question arises frequently in patients subject to angina, especially as regards the employment of the digitalis group of drugs. In the experience of the writer, digitalis in cases of angina pectoris caused by lesions of the coronary vessels commonly increases the frequency and severity of the complex, except when the angina is associated with definite evidences of muscle deficiency. When this

is the case, as it frequently is, digitalis will ordinarily improve the anginal condition in fairly direct proportion to the improvement of the evidences of muscle defect. The presence even of a heart block is no contraindication to the use of the digitalis group in cases of this kind, neither should any form of arrythmia contraindicate its use. In cases in which the presumable basis of the angina is a defective muscle condition as after typhoid, influenza, scarlet fever, rheumatism, and the like, digitalis is definitely indicated, in addition of course to rest and such special and local measures as may be indicated. Many cases of angina of such origin entirely disappear under the correct administration of the digitalis group of drugs.

After coronary thrombosis, when it is possible to give the indicated rest with the use of powerful sedatives and the like, one does not ordinarily use digitalis, but emergencies such as acute cardiac failure or pulmonary oedema may arise at any time, and the use of digitalis intravenously in very large dosage even, may become imperative. The writer has seen cases in which this measure has apparently saved life, but in the ordinary case, if no critical emergency arise, he is of the opinion that digitalis and other drugs of this class are contraindicated.

Drugs of the caffeine class are much favored in

the general treatment of angina pectoris because of their effect on the coronary vessels. Pharmacologists have shown that these drugs dilate the coronary arteries, and so increase the flow of blood through them, according to the experiments of Oswald Loeb and many other subsequent students. Of these drugs caffeine, theobromine, theophyllin and most recently and according to the pharmacologists most effective of all, euphylin has been generally used. Theocalcine is also valuable. The author has used them all, and thus far without any apparent bad effect. He is however, still rather skeptical as to their beneficial effects either as to prevention or in the treatment of the actual paroxysm and its aftermath. Their sole indication is to augment circulation through the heart muscle, but it is also probable that they may each of them, caffeine most of all, also have a stimulative effect on the heart muscle itself which may be beneficial in some instances. It is also highly probable that any of them, perhaps again most of all caffeine, will stimulate the nervous and emotional functions to such an extent that they may do harm instead of good.

Iodine, generally as the salt of sodium or of potassium, is very extensively employed in the inter-attack phases of angina pectoris. The theory for this indication is supposed to reside in its favorable

effect on arteriosclerotic changes which are concerned in so high a proportion of the cases. The fact that iodides are still used by so many clinicians in spite of the fact that a clear case for their action or indication has as yet not been made out, certainly indicates that they have something of value. The writer entirely coincides with this general opinion, though he is also unable to give any very definite reasons for their favorable action or yet to cite instances in which they have shown definite and certain influence along favorable lines.

No doubt the iodides are also useful in some of the chronic fibroid hearts which develop an angina. Their use is also indicated in the chronic rheumatic heart and the angina which is so commonly associated with, or dependent upon it.

Much stress is laid, in some sources, on the value of the bath treatment in angina pectoris. There can be no doubt but that in a good many instances the general regime and procedure in vogue at the various spas has a beneficial effect on the course of the disease. The careful regulation of the diet, the reduction under medical supervision of obese cases, the carefully observed and regulated exercise, such as are for example given at Nauheim, gives undoubted benefit to many instances of the complex, but that this good result is the effect of the bath treatment in greater measure than the general ben-

efit conferred in other ways seems very doubtful. In the better sanitaria which supply this class of treatment both here and abroad, much and entirely proper attention is given to all these measures. In the observation of the writer, the average cases benefit at least in some degree. The writer is inclined to attach great importance to the value of carefully graduated and observed physical exercise, massage, and allied measures. It is rather unusual that a patient can be induced to adequately carry out such measures in his own home, and when their use is determined upon it is far better to send the patient to one of the various sanitaria in this country or abroad where the treatment can be properly carried out, and where the average patient is likely to properly coöperate.

In following out any line of treatment specific or otherwise, even when the best possible result is attained, to the extent even that the patient is entirely freed from seizures, it must never be forgotten that irrespective of the apparent result, the heart can rarely return to an entirely normal condition, though such may indeed seem to be the case. The patient must therefore be cautioned that the disappearance of the symptoms does not necessarily indicate that the causative factors have also entirely disappeared. If permanent good results are to be obtained, it must be at the expense of a con-

tinuation of a considerable degree of careful supervision and full coöperation on the part of the patient. The physician can be of tremendous service to his patient by advising him as to the future control of his life, and of his habits of work and play.

Climatic methods of treatment are exceedingly beneficial in many instances. As a rule patients do very much better in mild rather than cold or temperate climates. Climates in which boisterous storms and violent changes in temperature recur frequently are particularly trying for subjects of angina pectoris. Where social conditions permit, patients living in the cold or temperate zones should plan to spend the more disagreeable months of their season in some such climate as that of Southern California, Florida, or Southern France, or Italy. A good many cases do exceedingly well in the Hawaiian Islands. Patients with this disease however, should not be sent to places where the comforts of life are inadequate, where they are unhappy, or where they must be confined to the house for long periods of time. As a rule, they do not do well at sea, though they may get on exceedingly well at the seaside.

They do not prosper at high altitudes, though there is ordinarily no reason why they may not go to an altitude under 2000 feet, provided that other conditions are satisfactory for them. Moisture ap-

pears to be an unimportant factor in determining climate for the subjects of angina.

Social possibilities naturally enter very largely into the treatment of angina pectoris. Persons living under heavy expense are frequently much relieved of their attacks when they are able to curtail their obligations, and to live therefore a life of much less tension and responsibility. Freedom from responsibility and from stress is highly conducive to improvement in the physical aspects of the disease. When patients are thoroughly unhappy little can be done to improve their condition. Readjustment of their life should therefore include a relief from as much responsibility as possible, and the primary object in any form of treatment should be to lessen the emotional and physical burden under which the patient must carry on.

Adjustments must be made which permit of regular rest and recreation. Sleep must be adequately provided for and stresses of all sorts must be averted. This applies equally to physical and emotional stress which are in this condition apparently of about equal importance. Very frequently persons once severe sufferers may be reduced to a class in which attacks occur at but wide intervals, and the intervening periods of life may be rich in both productiveness and in pleasure. The important

thing is always to recall that the heart once the subject of angina should never again be expected to rise to normal levels, and a frank acceptance of this fact on the part of the patient is a long step in advance.

CHAPTER XIV

TREATMENT OF THE ATTACK

THE treatment of the acute attack is perhaps the most important of all. It is of particular importance that the attack, no matter how mild it may be, be checked as promptly as possible. There can be no doubt but that the less frequent the attacks, the less severe they are likely to be. Habits of bar or resistance toward attacks are unquestionably developed by the prompt abortion of minor attacks. The more frequent the attacks, the more serious does the matter become.

Not only is this important from the standpoint of the attempted cure of the condition, but also it is important because no one can say what may be the outcome of any one attack no matter how insignificant it may seem to be at the outset. The sufferings of the patient are also to be considered, and in all probability, each attack further lessens the resistance of the heart and still further breaks down its muscle reserve.

When the patient is seized with an attack he should be allowed to assume any position which his experience has in the past taught him is most com-

fortable for him. Usually the patient stands perfectly still if taken with an attack while standing. If seated or lying down at the onset he will correspondingly remain fixed in the same position, for each movement seems greatly to augment his agony. There is no advantage to be gained by the supine or prone posture, and each patient has for himself usually discovered what is the most comfortable; it differs materially in individual cases. One of the favorite positions is slightly bent forward with the hands, shoulders or elbows resting on the back of a chair, a posture somewhat similar to that assumed in bronchial asthma. Occasionally the patient finds greatest comfort in lying face downward across a pillow or bed bolster, with some semisolid body as a pillow, or book firmly pressed against the precordium. In almost all instances the muscles are held firmly fixed, and rigid. Occasionally great relief can be afforded by the immediate application of a hot water bag to the precordium, or a turkish towel wrung out of hot water may be held firmly against the precordium. Occasionally relief is also given by the application of sinapisms, such as oil of mustard, menthol ointments, and the like. A combination of moisture and heat is as a rule most helpful.

The patient must not be questioned or required to speak, for in severe attacks he is almost speech-

less with agony, and the queries of solicitous relatives are likely to exact from the patient anything but cordial feelings, and he usually wisely affords no reply. Except in long standing and severe instances, prompt relief is commonly afforded by the administration of nitroglycerin, preferably given in the form of the Spirits of Glonoïn, one to three drops given in a little water. The tablets of nitroglycerin may be used, swallowed with a little water. For this purpose the patient will do well to carry in his pocket a small vial of hypodermic tablets of 1/100 to 1/50 grain dissolved in water and ready to take promptly. The dose may be repeated if necessary in ten or fifteen minutes until the characteristic nitroglycerin effect is produced, dilated capillaries, flushed face, and throbbing superficial vessels. It is recommended by many that a hypodermic tablet of nitroglycerin be placed beneath the tongue because of a supposed quicker absorption when so administered, but there is no advantage in this method, as has been recently shown experimentally to be the case. The chief disadvantage of this method of administration is that many tablets of nitroglycerin soon become inert and of no value whatever; for this reason the writer ordinarily specifies hypodermic tablets which are less likely to be sold when inert. The writer prefers however to advise the Spirits of Glonoïn carried in a little

pocket vial ready for instant use. The dose should be renewed daily.

Hypodermic or intravenous administration of the nitroglycerin is slightly quicker in action, and if the patient be of proper stability and intelligence, it may be well for him to carry with him constantly hypodermic units which are supplied by several standard drug dealers, and which may be promptly given if necessary. Erythrol tetranitrate tablets may be similarly used by mouth if preferred; they appear to the writer to be a little less prompt in result, but their effect is definitely more prolonged.

Ampoules or pearls of amyl nitrite act beautifully in many cases. The pearl or ampoule is crushed in a handkerchief and the fumes are inhaled. A good many patients prefer to take the drug in this manner, partly because of its greater convenience, but some patients fail to get an equal effect if the nitrite is so taken: the matter must be determined in each case by individual reaction and preference which should decide the point for any particular case.

When taking the nitrites the patient should if convenient, sit down or lean against some firm body for the vertigo which develops from the nitrite occasionally is so severe that the patient may fall and injure himself. Patients new to the effects of the drug should be fully prepared for the char-

acteristic nitrite effect by full explanation before the drug is first given; otherwise the fright engendered may very seriously affect the drug effect, and the attack also. The chief point to be observed in the proper use of the nitrites is that they shall be given very promptly, and while they may have a beneficial effect late in the attack, in fact at any time, the drug is much more effective when given very early, both in the promptness and the degree of relief. This fact must be impressed upon the patient. There is very definite evidence to support the contention that with the relief of the distress which should promptly follow the nitrite effect, great diminution in the danger of the attack also takes place. This drug has nothing of the effect of a hypnotic, but it affords relief entirely through vasodilation, though several pharmacologists assert that instead of a drop in pressure, a rise takes place in so far as the general circulation is concerned. Of course it is the effect of the drug on the coronary arteries and on the aorta which counts chiefly in this complex, and the fact remains, all theories to the contrary, that the drug almost invariably gives startling relief to the attack in suitable cases.

General hypotension, or hypotension at the time of the paroxysm, is no contraindication to the use of the nitrites, nor is the effectiveness of the drug

apparently determined by general pressure conditions. If the attack persists, the drug may be given several times at fifteen minute intervals, but if three or four doses fail to give relief, it is usually useless to persist in its employment. Commonly, in coronary embolism or thrombosis, the nitrites have little or no effect. This affords a diagnostic measure of considerable value at times. The writer has never in all his experience seen any untoward or dangerous effects apparently produced by the nitrites given in attacks of angina pectoris. Occasionally the patient is given considerable discomfort if over dosage is given, or if the drug is prolonged in its effects as occasionally takes place. As before stated, it is very important that the patient be as fully acquainted as possible with the full effects of the drug, so that he may stop it before unpleasant effects arise, but particularly because additional fright at the time of the attack is an undesirable factor, and one very likely to add greatly to the danger of the attack.

When possible the best dosage of the nitrites should be determined by the physician for each case. The writer is insistent that the drug be given as early as possible in the attack, and that in each instance definite attempt be made to abort the attack if possible. He believes that the danger of the attack is thereby greatly diminished. If the

nitrite fails in some form to effect its purpose, prompt recourse to other measures should be had.

R. H. Babcock (Jour. A. M. A., Jan. 19, 1924) has prescribed benzyl benzoate for twenty or more persons suffering angina pectoris. Six at least have been so distinctly improved as to justify the daily use of the remedy for months. In some cases very slight, or no alleviation of pain has taken place. In no instance have harmful effects been observed. Give it in milk or cream. Thirty drops four times daily are suggested as dosage of efficient amount. The author has experienced no constant or definite benefit from the use of benzyl benzoate, and the drug now seems to be but little used for this purpose.

Morphine by all odds is incomparably the most certain drug for the relief of the attack. It is not quite so prompt in effect as the nitrites, nor does the author feel that coincident with the relief goes a rapidly decreasing danger of death, as seems to be the case with the nitrite relief, but when the agony is very great, and when it does not yield to the nitrites rather promptly, morphine should be used. In a chronic long-standing condition such as angina is, one would expect that the morphine habit would be frequently established. Undoubtedly in a certain type of individual, this would be the case, and the author has seen chronic morphinism rather fre-

quently in cases of pseudo angina, but in all his experience he has never known a true angina pectoris subject to develop morphine habit from his therapeutic use of the drug for this purpose.

Ordinarily one-fourth grain given by mouth, but far more preferably by hypodermic injection, will suffice to abort an attack, but if a coronary thrombosis has taken place, far larger doses are required before relief is effected, and in all probability the drug will have to be repeated several times before anything like comfort is afforded the patient. Generally speaking, it is not wise to put morphine in the hands of any patient for self administration, but in angina pectoris an exception to this general rule appears to exist, and the writer in selected instances furnishes his patients with morphine units for hypodermic administration to be used whenever severe attacks unaffected by rest and by the nitrites appear. The agony of these attacks is so great that in the opinion of the writer such a measure is only the humanitarian thing to adopt, especially with patients who are frequently obliged to be far distant from prompt medical aid. It is well in such instances to instruct some intelligent member of the family with the manner of administration by hypodermic, using the sterile hypodermic units now furnished by standard drug houses.

Heberden advises the use of opium given at

night to prevent attacks which develop during sleep. Dovers powder has been largely employed for this purpose, and when other soporific drugs fail to give effect, it may be so used. There are also cases which will require the use of opium in some form almost as a matter of routine for the prevention of attacks. Opium may be thus used for long periods of time without seriously compromising the general health or producing the drug habit. In a disease of this character it frequently becomes imperative that relief be furnished, and until better operative results are obtained, opium must be resorted to in more than infrequent circumstances. It is not only a therapeutic error, but fanatical cruelty which forbids the use of opium when necessary in these cases.

Alcohol given in the form of brandy, whisky, rum, gin or applejack frequently gives great relief from the attacks, especially from those which appear to originate with gastrointestinal distress. It may be combined with peppermint, as crème de menthe, or with other aromatics, or with camphorated tincture of opium; all have their definite uses. Codeine is usually ineffective, but pantopon is almost as reliable as morphine, especially when given hypodermically.

Occasionally a warm tub bath will give great relief in prolonged but not agonizingly acute attacks,

and warm applications to the extremities often give marked relief in the same sort of attacks. The hæmostatic method of Saul Danzer has been tried in a limited number of cases with apparent success; at least patients request its reëmployment and assert that it gives relief from the acute suffering. Danzer's method, which is often of great benefit in other instances of collapse, consists of an arrangement of four blood pressure cuffs, applied about the four extremities, and supplied with pressure from a central distributing point, in which the air pressure is raised by a single check valve pump precisely similar to that used in the ordinary blood pressure apparatus. It stops the arterial flow in all the extremities simultaneously, and though of greatest benefit in conditions of general collapse, it seems to afford considerable relief in attacks of angina which do not subside under the more usual methods of treatment.

After the acute attack has subsided, the patient should be quieted down and urged to fall asleep if possible. A hypnotic for this purpose may be given, preferably some such drug as chloral hydrate, allonal, adalin, or the like. Unless sleep follows, secondary attacks are very likely to appear, and thrombosis of the coronary is probably so favored.

Coronary thrombosis is so frequently a complica-

tion of angina pectoris that it may be looked upon as a phase of the complex. It occurred in 85 out of the 350 cases studied by the writer. In our opinion, it is really part of the same general process. Its treatment must be therefore, very briefly considered here. In a very high percentage of cases, coronary thrombosis is the closing phase of the entire picture.

Nitrites are practically useless in coronary thrombosis and large doses of morphine are imperative. They must be given until relief of some degree is furnished and without morphine or some substitute, death is even more than usually likely to follow. The patient must be kept under morphine if necessary until the pain has been reduced to a bearable degree. This may entail the use of morphine in large doses for days, weeks, or even months. As a rule nothing else gives even moderate relief, and the physician must give surcease in some way or early exhaustion and death will follow or the process extending, death results from cardiac rupture. Hence, the importance of quiet and therefore of relief of pain. In a few instances, the writer has had to resort further to the use of chloroform in addition to the morphine when it seemed imperative to give relief of the pain. He has apparently killed no patients by the chloroform, and has given relief of suffering where

morphine seemed powerless in several cases. Quiet is necessary to check extension of thrombosis, permit healing of the infarction and lessen the probability of cardiac rupture.

A long period of as nearly as possible absolute rest is necessary, and this must be continued until opportunity for scar formation and fibrous repair has taken place. During all this time the patient must be kept quiet and necessarily meantime his pain must be brought under control. The strong probability of coronary thrombosis in long standing cases of anginal pain is always to be considered, and for this reason the necessity of rest in treatment must be fully realized.

Coronary embolism is the manner of termination in most cases of angina pectoris, and it is through this complication of the syndrome that the sinister reputation of angina pectoris has been chiefly achieved. It must be suspected in any instance where the anginal pain is long prolonged, or when it fails to be relieved by the vasodilator group of drugs. The one method of treatment is the relief of pain by the use of the morphine or its derivatives, and time. It must be realized by the physician that the time element is extremely important because scar formation in the infarcted area of heart muscle must take place, and either a canalization of the thrombous brought about or a revascu-

larization effected through subsidiary blood vessels. Both of these processes require much time, and the patient can not be considered as even relatively out of danger until these two conditions have been brought about.

CHAPTER XV

SURGICAL TREATMENT

IN THE management of cases of angina pectoris, the physician is quickly brought to a realization that cure is effected in only a very small number of cases and solely in those instances in which the specific cause of the essential heart lesion is detected and removed. Relief is possible in a much larger group of cases. As a rule it is quickly apparent that the earlier the complete control of a case is assumed by the physician, the better the outlook of the patient in this respect. Even in these favorable instances, the danger of sudden death is not removed, nor can the condition ever exist except as a severe handicap in every respect.

The suffering in angina pectoris probably equals or exceeds that present in any other disease condition, and it is therefore small wonder that the surgeon has been appealed to for relief from this almost intolerable agony. Obviously any attempts at surgical relief must be based upon an understanding of the basic physiology involved in the production of the syndrome. It is clearly impossible to attack the complex surgically by any

operative procedures on the heart or aorta, and therefore all that can be attempted is the relief of the sensation of pain by eliminating the pathways through which the sensation of pain is transmitted or realized.

E. Spiegel (Wiener Klin. Wochenschrift, 40, 853-855, June 30, 1927) through experiments on cats and dogs states that he has shown that pain originating in the heart or aorta takes the following path; passing from the heart by sympathetic fibers, it reaches the stellate ganglion and having passed this, it enters the rami communicantes and so reaches the spinal cord along the posterior roots. The posterior spinal roots involved are those between the 8th cervical and the 4th dorsal vertebræ. In these spinal segments lies the trophic centre of pain perception. Experiments made by Spiegel and Hashimoto show that the stellate ganglion contains sensitive fibers even after the division of the brachial plexus on the same side and the four first intercostal nerves. This would indicate that the central process of the spinal ganglion cells governs the sensibility of the heart and aorta, and that it reaches the spinal cord directly and not through the intermediary of other sensitive elements.

Since surgery can not in any way remove the cause of the pain, or affect the primary and serious lesion which causes its evolution, surgical effects

of a beneficial kind may be obtained only by an effort to interrupt the centripetal path of the sensation of pain before it reaches the central nervous system.

If these animal experiments conducted by Spiegel can be applied to man, and in the main, Spiegel's findings are similar to those of other experimental physiologists, then the centripetal conduction of pain sensations from the heart or aorta in angina pectoris may be interrupted at the stellate ganglion, at the rami communicantes or at the posterior roots from the 8th cervical to the 4th dorsal segments. Whether the first or the second operation may, if performed on both sides, produce any deleterious effects on the heart and aorta appears to be still in question. A sufficient number of operations upon man involving both sides have however already been performed, to justify the step in those instances in which no relief can be afforded by purely medical measures.

Spiegel feels that the division of the posterior roots is the only operation resting on a secure physiological basis and one which can not effect any serious harm to essential structures, while effectually blocking the sensation of pain. Many or most of those who have operated upon these cases feel that the resections of the stellate or even of the

other sympathetic ganglia can be accomplished even on both sides without any serious harm.

Elliot C. Cutler (Annals of Clin. Med. 5:1004-) has summarized the methods in general vogue in a very satisfactory way. He assumes that the innervation of the heart is accomplished entirely through the vagus and the sympathetic nerves. The only afferent fibers of the vagus are included in the depressor nerve which has an extremely inconstant location anatomically, and probably is not a pathway for pain conduction. Both motor and sensory fibers must be considered as associated in the sympathetic trunks.

The sensory sympathetic fibers either enter the stellate ganglion directly or run by way of the middle cardiac nerves to the inferior and middle cervical ganglia. It seems reasonably certain that the superior cervical sympathetic ganglion is purely motor in function. The sensory fibers which reach the middle and inferior cervical ganglia run downward and terminate in the stellate ganglion from which appear the fibers connecting the sympathetic trunks with the spinal nervous system. There seem to be no white rami above this level.

Operations which include the removal of these ganglia rely on the division of both motor and sensory nerves which connect them with the heart. The benefit is achieved from the severance of either

one or the other, afferent or efferent fibers, which, has not been determined. If the connections of the superior cervical ganglion are left intact and if the pain is eliminated by the removal of two lower cervical ganglia and the first dorsal ganglion, we may conclude that the relief is attained by interference on the sensory side of the reflex arc.

Cutler discusses the operations under the heading of four groups: Group I. "Cases in which only motor elements are divided. This group is restricted to procedures upon the superior cervical ganglion and includes only the operations which involve removal of this ganglion or interference with its branches to the cardiac plexus, called the superior cardiac nerves. All procedures in this group have to do with division of the reflex arc on its motor side and concern nerves whose function is primarily in relation to coronary vaso-motor control. Such a technic does not, of course, remove all motor connections."

Group II. "Cases in which only the sensory element is divided. A method of attack restricted to this procedure should in its fullest expression include only those cases in which the fibers connecting the middle and inferior cervical ganglia and the spinal cord, either in the main trunk or via the gray rami to the spinal nerves, and the first dorsal ganglion and its spinal cord connections are inter-

rupted. This would leave intact the motor connections of the sympathetic chain with the heart, since the ganglia and their cardiac branches would remain intact."

Group III. "This group includes the complete Jonnesco operation which consists of removal of the entire cervical chain and the first thoracic ganglion, at once interrupting both motor and sensory elements. With our knowledge of what occurs when the superior cervical ganglion is removed, a greater percentage of good results in this group may be fairly translated as due to interruption of its sensory pathways."

Group IV. "This includes procedures involving the entire cervical sympathetic chain without interference with the stellate ganglion. This group is included because of the great importance of emphasizing the known physiological pathways and in order to indicate again the fact that all afferent impulses passing to any of the cervical sympathetic ganglia can reach the central nervous system only by passing through the stellate ganglion. Successes in this group can have two explanations, neither of which can be convincing, as there remain both sensory and motor connections between the heart and the central nervous system by way of the first dorsal ganglion. Success must mean there

is reduction in the amount of stimulation to a point at which the symptom pain is not evoked."

Cutler is evidently of the opinion that at present the complete operation of Jonnesco on both sides offers the most in the way of lasting relief from the pain; no effect on the danger to life is anticipated. The operative treatment of angina pectoris is as yet not a complete success, perhaps because the connections between the heart and the central nervous system have as yet not been entirely discovered. The expression of pain in angina pectoris may not be entirely cardiac in origin.

Brown and Coffey (Trans. Assn. Am. Phys., Vol. XXXIX, p. 451) first operated by severing the main trunk of the left sympathetic nerve below the superior cervical ganglion and the superior cardiac nerve. Since that time the superior ganglion has been removed. They state "It seems clear to us that our operation removes the main cause of angina." This is based on the supposition (1) that angina is due primarily to spasm of the aorta, but it may be also of the coronaries. (2) that the superior cardiac nerve has a constrictor action, and cutting it prevents the spasm. . . . "It would seem that our operation removes the cause of the anginal attack and the more elaborate operation of Jonnesco removes the cause and also removes the other motor and sensory pathways." Few internists or patholo-

gists, familiar with the clinical and pathological findings in the complex can subscribe to the theory on which Brown and Coffey base their operation, whatever their surgical results may be.

The writer has had too little experience with any of the operative procedures as yet to justify him in the expression of a personal opinion and the literature on the subject is still very discordant as to results and their permanency.

It seems obvious that final and conclusive results are not to be expected in very long-standing cases, and yet it is these alone that may be easily persuaded to submit to the operation. Nor does the cautious internist feel like insisting on the operation in early cases because of the knowledge that relief may be usually afforded in some degree at least and occasionally real cure may be effected by medical means alone. Lilienthal, well known because of his fine surgical judgment and great ability in this particular class of work states (M. J. & Record, June 16, 1926) "If due to coronary disease the chances of improvement by operation are not so good, as if due to aortic disease. Relief by nitrites would be apparently a test of some value in deciding upon sympathectomy."

Undoubtedly the greatest deterrent to the operation in the minds of most internists is the feeling that while relief from pain may be afforded, no

release or reduction in the danger of the disease is to be expected. Furthermore, as Mackenzie was quick to say when the operations were brought to his consideration, the elimination of pain, takes from the patient at once his chief warning and safeguard against the complex, for if pain be removed without reduction in the danger of the condition, a fact which appears to be universally granted, the patient is very unlikely to cautiously follow out the precautions which the physician knows to be necessary if the life of persons subject to this complex is to be prolonged. The few operative cases which have come under the observation of the author have greatly impressed this fact upon him.

There is unquestionably a field for surgery in some cases of angina pectoris. As any of the operations now stand, the writer feels that they may nonetheless be recommended, but only in those instances in which the suffering is very great and in which the hope of recovery is practically abandoned. The operative treatment is recommended only when all medical means have failed and with the full understanding on the part of the patient or of some of his authoritative relatives that the operation in no way minimizes the danger to life. It is to be accepted as a humanitarian measure only.

If operation be decided upon, the writer feels

that the type of the procedure adopted should be left entirely to the discretion of the operating surgeon. There is little doubt on the part of most physicians that the complete bilateral Jonnesco procedure offers the greatest hope for real relief, but it is accompanied by considerable immediate danger to life and it should be undertaken probably not on one occasion, but with an interval between the two operations.

It goes without saying that these operations should be undertaken only by an expert surgeon, thoroughly acquainted with the procedure and with the anatomy of the structures with which he is to deal.

To be considered in precisely the same light as the operation is the injection method devised and successfully carried out by Swetlow. On paper this procedure may not seem to be either difficult or dangerous. In the publications of Swetlow the method is made so clear and definite that the writer fears that many may underestimate the difficulty, and the highly developed and refined technic necessary if the operation is to be safely or successfully carried out. The difficulties of the procedure are certainly no less than those of the great radical operation, and though Swetlow has experienced no grave results, the writer feels definitely that the

open operation in the hands of an equally expert operator is a no more difficult undertaking. The indications and the contraindications for this procedure are precisely the same as exist as regards the operations.

CHAPTER XVI

PROGNOSIS

PROGNOSIS in angina pectoris is exceedingly uncertain and there are no rules or signs which permit the clinician to prognose with anything like accuracy.

Bouradel quotes Forbes as reporting that of 64 cases of angina, 49 or 76.5% died suddenly. Sudden death in the first attack may not be very common, but Wenckebach is far from correct in his estimation that such a termination is rare. Lambert notes that in 1924 in New York City, 85% of the 410 cases of angina which died from angina were reported through the Medical Examiner's office which indicates that the deaths were sudden, unexpected, and that the patients were not under the care of physicians at the time of their death. Heberden, in his original communication prophetically states, "For if no accident intervene but the disease go on to its height, the patients all suddenly fall down and perish almost immediately."

A good many cases die soon after the onset of recognizable symptoms. Some cases die in the initial attack, usually of a coronary thrombosis or

embolism. Occasional instances make a complete recovery. These are those in which the basic lesion is a remedial one as in syphilis or gout. The average duration of life taken as cases run is low, probably not over three to five years after the initial signs and symptoms appear, but obviously each instance is a law to itself in this as in most other respects.

We are well within the truth when we assert, however, that prognosis is to a very considerable degree influenced by the character of treatment received, and largely on the coöperation which the patient gives in following it out. Primarily, however, the prognosis is determined in greater part by the character of the cardiac lesion which is productive of the syndrome.

INDEX

Age in etiology, 9
Albutt, 8, 52, 59, 83
Alcohol, 122, 144
Altitude, 133
Aneurysm of heart, 39
Angina sine dolore, 24
Anxiety, 24
Arrhythmia, 26, 36, 84
Arteries, disease of the coronary, 49

Baths, 131, 144
Benzyl benzoate, 142
Brown and Coffey, 155
Brunton, Lauder, 4

Caffeine, 130
Chloroform, 146
Climate, 133
Coffee, 81, 87, 123
Coitus, 31, 125
Coronary arteries, disease of, 49
Coronary thrombosis, 27, 34, 95, 129, 145
Cutler, E. C., 152
Cyanosis, 30, 74

Death, 34, 126
Dextrocardia, 19
Diabetes mellitus, 43, 55
Diet, 14, 89, 99, 120
Digitalis, 75, 128
Diphtheria, 113
Dyspnœa, 30, 74, 85

Effort syndrome, 78
Electrocardiogram, 27, 84, 92, 94
Emotion, 8, 12, 31, 45, 65, 75
Etiology, age in, 9
  occupation in, 6
  sex in, 7
Exertion, 30, 74, 84, 100

Familial tendency, 98

Gall-bladder disease, 20, 45
Gout, 60, 110

Heart, aneurysm of, 39
  rupture of, 38
Heberden, 1, 15, 48, 118, 143, 160
Heredity, 11
Herpes zoster, 22
Hirschfelder, 53, 76, 78
Hyperesthesia, 18, 23
Hypertension, 9, 29, 44
Hyperthyroidism, 41, 78
Hypotension, 9, 140
Hysteria, 45, 68, 72

Infections, 41, 101
Iodine, 99, 130

Jenner, 3, 49
Joslin, 43

Lambert, A., 7, 21

Menopause, 79
Morphine, 142
Murmurs, cardiac, 28
Myocardial degeneration, 73, 79

Nausea, 18
Nephritis, 44
Neuralgia, 54
Nitrites, 79, 92, 127, 138

Obesity, 42
Occupation, in etiology, 6
  selection of, 103
Œdema, 74
Operations, 153

Pain, 15, 35, 83, 91
  reflection of, 18, 91
Pardee, 95
Paroxysm, 15, 23, 83
Pericarditis, 54, 75
Pneumonia, 40
Pseudo-angina, 3, 67, 91
Pulse, 26, 29, 36, 77

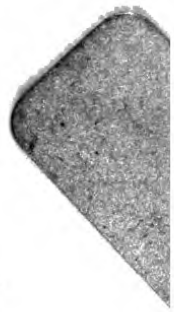
Reeder, 3
Rest, 75, 114, 134
Rheumatism, 112
Rupture of heart, 38

Sedatives, 118, 128
Sex in etiology, 7
Sexual excitement, 13, 31, 106, 125
Sleep, 119
Sodium chloride, 123
Spiegel, E., 150
Sports, 100, 102
Strychnine, 89
Swetlow, 158
Syndrome, effort, 78
Syphilis, 55, 107

Tea, 81, 88, 123
Thrombosis, coronary, 27, 34, 95, 129, 145
Tobacco, 81, 83, 124

Vacations, 105
Vomiting, 18

Wertheimer, R., 22
Willius, F. A., 96

Publishers of BOOKS and of
HARPER'S MAGAZINE

Established 1817