A large, slowly-developed effusion is not as threatening or immediately dangerous, and a small effusion is of no greater gravity than fibrinous inflammation.

A prognosis must be influenced largely by the condition of the muscle of the heart at the time of the pericarditis. Chronic alcoholism and the cachexias of cancer, Bright's disease, and tuberculosis, make slight the possibility of prolonging life. Pericarditis in these cachexias is usually due to a terminal infection and is often the immediate cause of death. It is also a much more serious complication of pneumonia than of pulmonary tuberculosis and a very grave complication of sepsis.

As might be expected, it is most dangerous in the earliest and latest years of life.

ANGINA PECTORIS—TRUE AND FALSE.*

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True angina pectoris is, comparatively speaking, an uncommon condition. The mechanism involved in its pathogenesis is imperfectly understood, and the views found recorded on the subject in medical literature are, for the most part, hypothetical. Two facts, however, are generally accepted: First, that structural changes of the cardio-vascular system are present in the immense majority of cases, the chief among them being arterial sclerosis, especially of the coronaries, aortic regurgitation, aortitis (syphilitic?), hypertrophy, aortic aneurism, adherent pericardium and myocardial degeneration—fatty and fibroid. These lesions may be variously combined and the instances in which a certain grade of calcification of the coronary arteries and root of the aorta, associated with more or less myocardial degeneration, has not been found in evidence postmortem, are of rare occurrence.

The second fact which passes current is that irritation or disturbance of the cardiac sensory nerves exists. Unfortunately most of the leading theories concerning the nature of angina attempt to explain the cause and seat of the pain, their authors forgetting that this symptom is but one of a group of characteristic features. While, therefore, a universally acceptable theory of the pathogenesis of this syndrome lies before us in the future, yet if the few isolated facts, which appear to be amply attested either by clinical experience or post-mortem observations, were to be brought into harmonious union, our conception of the symptom-group under discussion would doubtless become less vague and indefinite than it has been hitherto.

ETIOLOGY.

Multiple views are held regarding the question of the immediate cause or causes of the precordial paroxysms and to refer briefly to the more important ones will serve my purpose. In the first place, the theory that angina vera is a neuralgia either functional or of neuritic origin, is untenable, since it neither accounts for the mental anguish and other associated phenomena (as pointed out by Osler) nor for the cases of *angina sine dolore*.

The somewhat analogous clinical phenomena presented by intermittent claudication, in which the principal pathologic condition is also arteriosclerosis, are believed by some writers to throw a welcome side light

on the mode of origination of angina in some cases at least. Neurologists and clinicians concur in the opinion that the pathogenesis of intermittent claudication embraces two leading factors, namely, arteriosclerosis and angiospasm of the affected blood vessels, suddenly induced by the usual exciting factors. Typical cases, which later developed angina pectoris with fatal termination, have been reported by Charcot and others.

In speaking of the relation of angina pectoris to arteriosclerosis, Osler remarks, "the pain in angina pectoris is *sui generis*, unlike in intensity any known variety, and while its association with the coronary artery sclerosis is unquestionable, there is something additional, some other element for which, as yet, we have no explanation."¹

There are not a few eminent observers who seriously question whether the vasomotor spasm affecting the blood vessels in angina pectoris stands in the relation of a causative factor. Says Allbutt,² "Are not secondary vasomotor events translated from cause to effect?" A Morrison³ from an interesting series of sphygmograms infers that constriction of the peripheral vessels either during the course of or after an attack of angina is often the result of the attack, rather than the cause. Hill and Barnard² seem to have shown that intracardiac diastolic pressure is constant during large changes brought about in other pressure areas by vasomotor constriction. To effect a more or less complete relaxation of the vasoconstriction in any case in which it accompanies the seizure, however, serves to ameliorate the attack.

It must be recollected also that the leading exciting causes of angina pectoris—muscular exertion, mental emotion, gastrectasis, exposure to cold—all quickly raise the arterial tension. The occurrence of associated vasomotor spasm is true of most forms belonging to either true or false angina. The theory of intermittent claudication originally advanced by Allen Burns still finds a place in the realm of speculation, since it is impossible to conceive of coronary ischemia in connection with an unimpaired rhythm of the heart as is witnessed in many cases of angina.

The seat of the pain is undoubtedly in the heart in angina pectoris and the paroxysm itself is the result of an extra stress or strain thrown on an organ that fails to respond to a normal environment, or to the usual external stimuli. MacKenzie⁴ holds that since angina pectoris arises in connection with a great variety of gross morbid lesions of the heart, there must be an exciting cause common to all cases, and this he believes to be impairment of the muscular contractility of the heart.

As a *priori* evidence, MacKenzie cites the fact that this is the function "directly concerned in furnishing the motive force to the circulation of the blood and that it is the function that will necessarily become exhausted when an excessive resistance is opposed to the contraction of the heart muscle. The opposed arterial pressure may be abnormally high, but as a rule greatly lowered, since angina vera is a late complication, or to speak more accurately, a terminal condition. In either cases the extra stress or strain imposed by the action of one or other of the existing causes will exceed the contractile power of the heart and as a consequence suddenly induce an attack."

1. THE JOURNAL A. M. A., Sept. 17, 1904, page 775.

2. Lancet, July 18, 1903.

3. Lancet, Nov. 1, 1902.

4. "An Inquiry into the Cause of Angina Pectoris," J. MacKenzie, British Medical Journal, Oct. 7, 1905, p. 845.

* Read in the Section on Practice of Medicine of the American Medical Association, at the Fifty-seventh Annual Session, June, 1906.

This theory readily explains the cases of angina that occasionally follow an attack of pneumonia or other severe infection in advanced life in subjects of coronary disease with myocardial degeneration. I have recently seen a case in point (as a sequel of pneumonia) with Dr. Jungman, of Philadelphia. The theory of MacKenzie also elucidates a number of cases, which have been shown by observations carefully analyzed, to be independent of any coronary involvement as well as those of *angina sine dolore*.

A leading view ascribes the attack to a cramp-like contraction of the myocardium and although such a state of things might be excited by coronary infarctions, it would, as previously stated, preclude the possibility of regular and orderly rhythm of the systole.

A. Morrison³ holds that the paroxysms are due to "the distinctive throb of the blood cast by the dilated yet powerful ventricle into the aorta and coronary system;" also that the condition may be due to the effects of neuritis, to muscular spasm and to hemic conditions, the blood exercising pressure on local diseased areas. Colbeck⁵ contends that the cause of the paroxysm is a localized distension and stretching of the diseased areas in the ventricular wall, and Traube has advanced much the same opinion.

It is a common observation that stimulation of the heart in which predisposing conditions exist, more especially by the use of digitalis, which induces more energetic contraction and raises the peripheral tension, may provoke a recurrence or at all events, aggravate existing stenocardial pains. On the contrary, it is an equally familiar fact that the exhibition of arterial relaxants, e. g., nitroglycerin, is often followed by decided relief. Such considerations as the foregoing, however, do not explain why angina is admittedly uncommon among the working classes or hospital patients who present cardiac lesions identical with those met among the better-to-do in whom the syndrome is of far greater frequency of occurrence.

TRUE ANGINA.

Angina vera occurs principally in men engaged in sedentary and, for the most part literary pursuits, as well as those who follow an inordinately strenuous business career; in short, persons whose nervous mechanism is minus as the result of overwork, principally mental, with insufficient rest. An impaired nervous vitality, therefore, is to be looked on as a marked predisposing element. Is not the fact that angina is a neurosis of the heart too constantly overlooked?

This condition of the nervous system may be looked on as being neither less nor more than neurasthenia, but may there not be added some peculiar, nutritional or degenerative change in the nervous system, brought about by high nerve pressure, which finds its expression in a peculiar neurosis that serves as the predisposing element in angina pectoris?

No theory of the genesis of this syndrome is complete that does not account for *angina sine dolore*, in which the main feature is an indescribable almost painless, sensation or precordial oppression. The true essence of the condition may yet be discovered in connection with this minor group of cases. The history of an illustrative case may be briefly narrated, more particularly because of its intrinsic interest and anomalous character.

History.—M. D., female, married, aged 59 years; height, 5 ft., 2 in., weight 153 lbs. Two paternal uncles died suddenly of "heart failure," after the seventieth year of life, and one brother died at 54 of organic heart disease. The patient had had numerous recurrent attacks of follicular tonsillitis up to four years ago, and rheumatic pains at intervals, more particularly in the shoulders.

The social history embraces nothing worthy of mention save only that the menopause had been passed five years previously. The principal complaint at my first visit was constipation, which dated from the time of change of life. There was, however, slight dyspnea on exertion.

Physical examination revealed a moderate degree of emphysema and mitral incompetency in the stage of compensation.

Treatment.—The treatment during the next four years consisted of a rearrangement of the diet and an occasional laxative pill and a small daily amount of Celestine vichy.

Course.—Oct. 5, 1905, the patient complained of slight pains in both shoulders and the upper half of the precordial region with radiation to the left arm, which was also the seat of numbness. While the pains were never severe, they behaved like those of true angina, being excited by walking, facing the cold winds and mental excitement. Associated with the precordial pains was a sensation of marked cardiac oppression, and there was a moderate degree of anguish detectable. The seizures became recurrent first at intervals of a few days, and towards the close of life of about six hours. With the advance of the case the thoracic oppression was attended with a feeling of smothering or suffocation, and dyspnea also entered into the syndrome. The principal seat of pain, which was at no time severe, shifted later to the epigastrium for a time, to be followed in the course of a couple of weeks by *angina sine dolore*, the starting point of the seizures being in the epigastrium. A well marked restlessness and an almost uncontrollable desire on the part of the patient to leave her bed was present during the last few days of life. A striking feature was the manner in which the oppression recurred during a period of two or three weeks prior to death; it invariably originated in the epigastrium and right hypochondriac region, then traveled wave-like upward over the thorax, becoming most marked in the lower sternal region, and attended with dyspnea and suffocative sensations as already remarked. Neither in the epigastrium nor thorax (the heart apart) could be found evidences of disease to account for these symptoms. Objectively, the usual signs of angina pectoris were noted, especially the anxious facies and the ashy-gray appearance of the countenance.

Finally death came quickly on Jan. 15, 1906, after a pleasant chat with the nurse, without manifesting any distressing symptoms that were observed. There was neither arrhythmia nor evidence of arteriosclerosis present.

This particular form is found engrafted on a variety of structural cardiac diseases and may, therefore, be looked on as a terminal condition. I had never previously observed a case belonging to this category arise in the course of simple mitral incompetency, but one instance in the course of mitral stenosis. G. Steele⁶ has reported two cases of angina pectoris occurring in patients with mitral stenosis and he believes that symptoms so similar to angina occur in this disease that there is no reason for giving them another name.

Angina sine dolore is not uncommon, as pointed out by Oliver,⁷ in dilatation of the heart, aneurism of the first portion of the arch and adherent pericardium. What exactly is the essential cause of the absence of intense pains in these cases is still an unsettled question. It has been held to be on account of ensuing dilatation as was true of cases reported by Musser,⁸ but

6. Lancet, Nov. 23, 1903.

7. "Angina Pectoris and Allied Conditions," Lancet, Sept. 16, 1905.

8. American Journal of the Medical Sciences, vol. cxiv, 1897, pp. 294-302.

5. Lancet, March 21, 1903.

apart from a slight increase in weakness of the systole toward the close of life, no change in the physical signs were noted in my case reported above. From the fact, however, that the use of moderate sized doses of digitalis (given as a test) aggravated the precordial pains in the earlier stages of my case, the view that increased endocardial pressure stands casually related to this symptom receives a considerable degree of coloring.

This class of cases reminds us forcibly that a distinction is to be made between intense paroxysmal precordial pains and angina pectoris; that the essence of the essential difference between so-called false and true angina is to be found in some as yet unknown agency or possibly in the different degrees of impairment of the muscular contractility in the two conditions, this being slight in the former while it is carried to a dangerous extent in the latter, principally because of the associated structural lesions.

The claim has been made by some writers that cases in which the pain originates below the diaphragm are less threatening to life, but in this opinion I do not concur. Finally, true angina pectoris may be dependent on a diet containing an excess of proteids. The toxins in the circulating blood in chronic intestinal toxemia or autointoxication may primarily cause the toxic form of false angina; this occurs oftentimes in the young at a period of life when the balance of power is with the organism and gross structural changes do not arise, but at a later period the balance of forces is with the circulating toxins and slowly progressive degenerative changes of the cardiovascular system are the result.

Obese persons who exhibit high arterial tension with arteriosclerosis and left ventricle hypertrophy may, as I have observed, develop angina pectoris as the result of a rearrangement of the dietary, so that proteids in liberal amounts are allowed and the carbohydrates greatly restricted, with a view of overcoming the corpulency. In this class of cases, there is a strong probability of effecting a cure by appropriate measures, which consist principally in rest, arterial relaxants, stimulation of the emunctories, and a return to a mixed dietary.

PSEUDOANGINA.

In 1812, Latham described false angina. The term "false" or "pseudoangina" is applied to a large group of cases in which the predominating feature is precordial pain, sometimes with radiation. This term has been objected to by many eminent authorities, but inasmuch as the condition is allied symptomatically to true angina, rendering a differential diagnosis well-nigh impossible in many instances, I favor the use of the term pseudoangina until additional light shall have been thrown on the whole subject.

At the outset, I insist that many of the slightly painful conditions affecting the precordial region in rheumatic, neurotic and hysterical subjects can not be properly regarded as falling under the head of pseudoangina.

Doubtless, the majority of cases originate in vasomotor instability and irritability, and as would be expected, they are met with in the female, more especially the hysterical type, and about the period of the menopause. Heartstrain occasioned by suddenly-induced vasoconstriction will result in pseudoangina (Nothnagel) characterized by a special set of symptoms—palpitation, a feeling of faintness, precordial pain, perspiration with coldness of the extremities and lividity. Again, the instances of angina directly traceable to

autointoxication from errors of diet should be placed in the same category provided that no sub-basis in the nature of an organic lesion of the cardiovascular system is discernible. As previously stated, this variety of angina pectoris may be induced by an excess of proteids in the dietary. The recognition of the special cause and its removal if carried out sufficiently early in the course of the affection will, as a rule, lead to an absolute cure.

It will be seen that true angina and pseudoangina have causative factors and symptoms (as pains and vasomotor disturbance) in common. Spurious angina, however, differs from the true form in three leading particulars—first, gross organic lesions are absent; secondly, it differs symptomatically, and thirdly, it usually yields to appropriate treatment.

The cases of pseudoangina are divisible into three groups, namely, the neurotic, the toxic (two groups suggested by Osler) and reflex pseudoangina. The last-named clinical or etiologic type is rare in the experience of some writers and is usually classified as a subvariety of the neurotic class of cases. In my opinion, these instances are of sufficient importance to demand separate grouping. I do not include here the gastrointestinal phenomena that are found associated with true angina, but those that induce paroxysmal and intense breast-pang in subjects who present no gross cardiovascular lesions.

The fact that certain contributing factors in true angina are present in this variety is of great practical importance and explains why, apparently, typical cases of the spurious form, after the development of some gross cardiac lesion, may merge into true angina.

Cases in which apparent transition from false to true angina occurred, have been reported by Duffield,⁹ Allbutt,¹⁰ Morel-Lavallee¹¹ and others. I have seen two examples; the first occurred in a neurasthenic female who, while under observation for pseudoangina, developed arteriosclerosis, including coronary disease. The intensity of the pains remained uninfluenced, but mental anguish and dyspnea of a pronounced character became superadded features. A fatal attack occurred in the course of five years after the first true attack.

I may be permitted to refer to a second instance which recently fell under my notice.

A woman, aged about 40 years, who was distinctly hysterical, developed well-characterized attacks of pseudoangina at long intervals. A couple of years later indefinite signs of arteriosclerosis about the base of the heart developed, after which the symptom-complex simulated more and more closely true angina pectoris, and finally became typical of the latter condition. When I last saw the patient a slight mitral incompetency was present; this had obviously diminished the severity of the anginal seizures. I have been unable to learn the condition of the patient, who resides at the south, during the last four months.

From personal studies and experience I feel privileged to speak confidently regarding certain cases, which so far as can be determined by known methods of examination at least are non-coronary, but sometimes prove to be fraught with lurking dangers. In many cases of true angina pain alone is experienced (Osler) and this is more especially true of the milder forms. I recall a fatal case of *angina vera*, which I saw with Dr. Shivers, of Haddonfield, New Jersey, in which mental anguish and cardiac oppression were absent and during the par-

9. The Pathology of Angina Pectoris, Proceedings of Mich. State Med. Society, 1901.

10. Philadelphia Med. Jour., June 16-30, 1900.

11. Revue de Médecine, Paris, Nov. 10, 1890.

oxysms the patient exhibited great restlessness and commotion, such as commonly occurs in the false variety.

DIFFERENTIAL DIAGNOSIS.

When angina pectoris is well characterized we can differentiate it in a decisive way from the false variety by the *angor animi* and the strong sense of imminent dissolution. Many additional symptoms may be associated during the paroxysms, but are not necessarily present and only serve to corroborate the diagnosis of true angina. Among these, I would mention more particularly, respiratory disturbances, including asthma, dyspeptic symptoms and vasomotor disturbances, such as pallor of the face (rarely lividity), sweats and coldness of the surface.

On the other hand, pseudoangina of pure form is often associated with the menstrual function and its effect on the vasomotor nervous system. The rôle played by hysteria is also conspicuous as shown by the presence in some cases of globus during the attack. Unlike in true angina we note marked throbbing of the cervical vessels and epigastrium as well as flushing of the countenance during the seizures. There may be a clear history of toxic poisoning (tobacco) or reflex visceral irritation to support the diagnosis of pseudoangina. The paroxysms are not provoked by muscular exertion, mental emotion or worry and feeling cold winds as in real angina, but arise spontaneously as a rule.

It may be argued with considerable force that since real angina pectoris may occur in hysterical and neurasthenic subjects with or without gross cardiac lesions, there must be a contingent of cases in which a diagnosis within the limits of a reasonable probability only is possible.

It is the clinically mild and doubtful cases that come more particularly within the scope of the present discussion, and since it has yet to be shown that these are incapable of developing into typical angina vera, a most exhaustive survey of examples of the sort must be carried out before pronouncing in favor of false angina. It is permissible only as a rule to determine the inherent probabilities of a given case and opinions must be offered with due caution and reserve. It would also be far more helpful to patients suffering from dubious cases if they were treated for true angina until the question of diagnosis were definitely settled.

RELATION OF PSEUDOANGINA TO ANGINA VERA.

The relation of so-called pseudoangina to true angina pectoris is not definitely known at date of writing. The nascent stage of true angina, which may not be well characterized, is sometimes mistaken for the false form.

It is admitted that cases of angina vera ending fatally may fail to show any cardiovascular lesions. In such instances, the severity of the precordial pains during the paroxysms may cause the arterial pressure to fall so low as to be followed by fatal syncope. On the other hand, death from pseudoangina is unknown.

It can be accepted as a fact that the chronic lesions met with in angina pectoris may be excited or initiated by the toxic and other causes of pseudoangina. Admittedly, transition from functional to organic disease of the stomach occurs, e. g., many cases of chronic gastric catarrh result from pure gastric neuroses. Another illustration is furnished by the toxic effects of tobacco, which ordinarily produce pseudoangina, although rarely the paroxysms finally simulate those of the organic form so closely as to be indistinguishable. Whilst the evidence, chiefly clinical, to establish this remote relationship of the two forms of angina, would appear to be

clear and convincing, it must be granted that the results of improved methods of investigation more particularly as related to the arterial pressure, may in future enable us to interpret correctly all of the phenomena that enter into their clinical history and thus permit of an essential distinction between them or even to show that each is produced by a special cause.

Although there are apparently etiologic affinities between the two forms of angina—real and unreal—my view accords with that of Huchard, Osler and others, who claim that we should adopt this provisional classification and separate the functional from the organic form, whenever practicable.

Finally, it is commonly held, as before stated, that the false variety is solely of vasomotor origin, a sort of unsteadiness, which has been designated by S. S. Cohen¹² as vasomotor ataxia. True angina is allied to the functional form so far as vasomotor disturbances are concerned; it, however, embraces a variety of pathologic lesions whose relations to the symptomatic indications of the syndrome are truly obscure and complicated.

DISCUSSION

ON PAPERS BY DRs. ERLANGER, THAYER, POTTER, DAVIS AND ANDERS.*

DR. EDMOND GROS, Paris, France, referred to the three instruments which are variously used in different countries for the measurement of blood pressure, viz.: the Potain or modified von Basch, the Gærtner and the Riva Rocci. In France the Potain Sphygmomanometer is used almost exclusively. He was interested to know how this compared with the Gærtner and Riva Rocci measurements and was struck by the difference of pressure obtained with these instruments. The reason for these variations no doubt lies in the fact that each instrument measures the blood pressure in a different manner and in a different region of the body. Furthermore, there is in each case a factor of error which does not seem to be avoidable. The Potain, when properly applied, seems to give a relatively accurate measurement of blood pressure, but the pressure will be found too high if the recurrent circulation be not carefully obliterated. The Gærtner ring is applied in a region so rich in capillary blood vessels that various vasomotor influences such as pain, emotion, fever, etc., cause rapid changes in blood pressure. In fact Tessier has found that the very fact of tightening or loosening the Gærtner ring on the finger in eruptive fevers, raises the pressure in the radial artery from 10 to 30 m.m., evidently acting in the manner of a slight trauma. Gros exhibited an instrument which, he explained, is an ingenious adaptation of the Gærtner ring to the sphygmomanometer devised by Bouloimé of Vittel. It greatly facilitates comparison of the pressures obtained by these two methods. He has found that the blood pressure measured in the radial artery with the Potain ranges from 120 to 160 m.m.; the Gærtner indicates a pressure of from 10 to 30 m.m. lower, but the breach gradually widens when the radial pressure runs above 200 or 220 so that a pressure of 260 with the Potain corresponds to about 220 with the Gærtner.

The Riva Rocci, which represents the blood pressure in the whole of the arm, is relatively accurate, but here again the error factor comes into play, the tightening of the cuff first obstructing the venous circulation before obliterating the brachial artery. Therefore, the pressures obtained are higher than either with the Potain or the Gærtner.

Here, then, are three instruments in use in different countries giving markedly different measurements in each case, the figure not ever varying in any direct ratio. Until a standard of blood pressure is determined the figures found by different authors lose much of their value and can hardly bear comparison.

DR. WOODS HUTCHINSON, Redlands, Cal., thought that what stood out in the symposium as of prime importance, is, first

12. Transactions of the Association of American Physicians, 1902.

* The papers by Drs. Erlanger, Thayer and Potter appeared in THE JOURNAL, October 27.

of all, that too exclusive attention had been paid to the condition of the valves in heart disease. We have been too much inclined to regard them from purely a mechanical point of view, and to say that damage to the valvular mechanism was the most important thing. To-day most men go to the opposite extreme. Valvular lesions are relatively harmless so long as the endocardium is intact. The fact should be recognized that the heart is a pulsating mechanism with a sphincter valve action; not only do the valve flaps play a purely mechanical bucket-pump action in checking the regurgitation of the blood, but contraction of the ring of muscles surrounding the valvular orifice occurs and should be considered as well. There are a large number of murmurs, so-called valvular murmurs, which are due to disturbances of the musculature of the heart and not to the valve at all. He referred to one of his first cases, which went to postmortem, in which all the symptoms had pointed to a mitral insufficiency, and no lesions whatever of the valves were found, but the mitral orifice was extremely large, making the valve flaps inadequate to close it. The contraction of this sphincter auriculo-ventricular ring he considered a very important factor in carrying on the circulation. A heart with a perfect musculature can compensate very well even in cases of severe valvular lesions.

Secondly, too much attention should not be paid to the heart itself in cardiac troubles; serious damage of the heart may be compensated perfectly so long as the walls of the blood vessels and the general nutrition of the patient are in perfect trim. No better illustration can be given than to note how often patients with valvular mischief have outlived the men who condemned them to death. Valvular damage must be regarded, much as the gastroenterologists formerly did hyperchlorhydria, but so long as it does not cause the patient any discomfort it is of little consequence. He thinks somewhat the same position might be assumed as taken by an old German colleague on the alcohol question; he said that "alcohol injures no one except those with whom it happens to disagree." Even heart lesions may do little harm unless there is failure in compensation and lack of general recuperative power. With regard to the cause of heart mischief, by searching inquiry he was able to trace 70 per cent. of his cases to some acute infection, not so often to rheumatism as to the minor infections and exanthemata, tonsillitis, influenza, measles, typhoid, even summer diarrheas. If cardiac conditions were treated as sequelæ of infections and toxic conditions, better results would be obtained than by direct treatment of the heart itself. Indeed, the latter is often positively injurious instead of beneficial in the long run.

Dr. FRANK JONES, Memphis, Tenn., discussing the paper read by Dr. Davis on "Pericarditis," said he had been struck in studying that disease by the analogy which could be drawn between diseases of the pericardium and diseases of the pleura; the same conditions may produce diseases of the pericardium that produce diseases of the pleura, such as rheumatism, pneumonia, tuberculosis, etc. He has been struck with the coincident pleural and pericardial affections, whether hemorrhagic, sero-fibrinous or purulent. Dr. Jones said that he has frequently observed cases in which the symptoms were held in abeyance, but the physical signs were marked. He has seen patients in the clinic with no dyspnea, no cough, no acceleration of the pulse and no temperature; but in whom physical examination revealed a pericardial sac two-thirds filled with liquid. In one case 16 ounces of pus were removed and this patient walked one-half mile. Dr. Jones believes that in the majority of instances of effusions of fluid in the pericardium the more insidious they are the more often will they be found to be tuberculous in nature. There are more cases of tuberculosis of the pericardium than is generally supposed.

In discussing Dr. Ander's paper on "True and False Angina Pectoris," he said that so far as he is concerned he has little patience with the nomenclature of false angina; there must be some pathologic condition back of it somewhere. In the production of pathologic conditions we hear of cardiac insufficiency, of hepatic insufficiency, of renal insufficiency, etc., but we do not hear enough of gastrointestinal insufficiency, frequently the *alma mater* of all insufficiencies. He said he was struck not long since in reading a paper on so-called pseudoangina pec-

toris, by the statement that if these cases were examined critically and the patients went on and lived a more vigorous life, the false would be run out and the true run in.

Dr. W. S. THAYER, Baltimore, agreed with Dr. Jones' views as to the frequency of tuberculous pericarditis; he regards insidious sero-fibrinous pericarditis as being, in the majority of cases, tuberculous in nature; this is supported by the results of necropsies. He is inclined to regard the estimate that 12 per cent. of pericarditides are tuberculous as too low. With regard to angina, he agreed that in cases with anginoid symptoms it is sometimes difficult to determine whether or not grave organic cardiac lesions are present. The most important diagnostic sign is the effect of exercise; if the patient is suffering from true angina, the result of severe organic changes, exercise almost invariably brings it on. With regard to the seat of the pain, he has seen several instances in which the pain had been referred primarily to the back. In one case, now under observation, in which the pain follows any severe physical effort or mental excitement, it occurred first between the shoulders, later, accompanied by pain in the precordial region and numbness down the left arm. In discussing Dr. Potter's paper, Dr. Thayer referred to the interesting observations of Pässler and Heinecke, who have succeeded for the first time in producing cardiac hypertrophy in animals by removing first one kidney and later, in successive operations, portions of the remaining organ. After a certain amount of renal substance had been removed, polyuria followed without any increase in the blood pressure. On removing more kidney substance there followed a rise in the blood pressure which was permanent and associated with characteristic cardiac hypertrophy. They noted also that, in these animals, arterial spasm with further rise of blood pressure quickly followed stimuli which in normal animals would produce little effect. These observations would suggest that the heart hypertrophy is due to increased work dependent on the impediment to the circulation resulting from the tendency to arterial spasm due, in its turn, probably, to retained toxic substances.

Dr. JAMES TYSON, Philadelphia, referred to two interesting cases of pleurisy with effusion and pericarditis with effusion. In the first, in a child of 8 years, the removal of a considerable amount of fluid was successfully accomplished by tapping in the seventh interspace midaxillary line and it was supposed to have come from the pleural cavity, but at autopsy the pleural cavity was found empty and it was found that the fluid came from the pericardial sac. The second case was one of supposed pericardial effusion. The fluid was successfully removed with a trocar introduced in the sixth interspace in the midline, left side; and the vibratory motion conveyed to the distal end of the trocar made it reasonable to believe the instrument was in the pericardial sac. The fluid was also discharged in spurts, corresponding to the cardiac rhythm. Later there appeared signs of pleural effusion. At autopsy a typical tuberculous pericarditis was noted and in addition a tuberculous pleurisy with effusion. The autopsy did not afford evidence as to whether the fluid primarily removed came from the pericardial sac, but there was no reason apparent why it did not. These cases prove the possibility of the two conditions of pericardial and pleuritic effusion being confounded.

Dr. Tyson added that it has long been recognized that large hypertrophies of the various cavities of the heart, especially of the left ventricle, occur in chronic Bright's disease. He is inclined to think that the primary cause of this condition is a toxic condition of the blood, exciting resistance to its movement through the vessels, which in turn excites the primary cardiac hypertrophy. Arteriosclerosis caused by the same agency soon coöperates with this. This arteriosclerosis undoubtedly contributes to the hypertrophy of the left ventricle. Dr. Thayer's remarks as to the effect of experimental removal of portions of the kidney revives the theory of the resistance to the amount of blood passing through the blood vessels of the kidney caused by destruction of the organ and its replacement by fibrous tissue. In the matter of angina and pseudoangina Dr. Tyson said that he is disinclined to acknowledge pseudoangina as an entity; these cases ultimately prove to be either true angina or hysteria. Finally, he directed attention to the analogy between the pain in cramp of the muscles of the leg with the pain in angina

pectoris. Probably the muscular spasm in both is the result of anemia of the muscles and the cramp due to compression of nerves by the cramped muscles.

DR. T. A. CLAYTOR, Washington, considered only the blood pressure in heart conditions. In the first place he said that he has observed in cases of cardiac insufficiency, whether they be from valvular insufficiency or not, that when patients are first seen suffering from dyspnea, edema, and indications of loss of compensation, the blood pressure would be found to be high, even as high as 180 to 190. But after they had been kept in bed and given digitalis, the blood pressure fell. This is very remarkable, but he said he has seen it so often that he believes it to be almost the universal rule. When the blood pressure is high in loss of compensation, after the use of digitalis it falls and becomes almost normal.

He then spoke of a class of cases showing high blood pressure associated with cardiac hypertrophy with probably renal insufficiency. These patients, usually between 40 and 70 years old, give symptoms of slight shortness of breath on exercise and precordial distress. Physical examination reveals a hypertrophied heart, an accentuated second sound, and probably a slight mitral insufficiency murmur. Whether this is due to a real valvular lesion or due to the relative insufficiency it is hard to say. The blood pressure was found to be high. The urine showed the presence of albumin and casts at times. Such cases were probably Bright's disease. The blood pressure varied from 180 to 225 or more. The first idea that comes to all would be that the pressure is too high and should be reduced and the question arises what is the best means to reduce this high blood pressure. The ordinary nitrites are practically useless; nitroglycerin is of little value. Iodid of potassium has more effect. It relieves the headache which is often associated with high pressure and this is the most satisfactory means, aided by rest in bed, purgatives and a milk diet. Since high blood pressure is, in this class of cases, probably a conservative effort on the part of Nature to overcome a pathologic defect, Claytor doubts the advisability of making such strenuous efforts to reduce it in every instance.

DR. C. M. COOPER, San Francisco, said that a young man recently entered the German Hospital in San Francisco in the service of Dr. Jellinek with symptoms of acute gonorrheal septicemia. He developed an acute Adams-Stokes syndrome. The disease ran an acute course and at postmortem an acute lesion was found in the vicinity of His' bundle, thus strongly emphasizing the relationship between a lesion of this bundle and the clinical symptom-complex known as Stokes-Adams disease. Recent work has shown the importance of taking simultaneous tracings of the venous and arterial pulse and of the heart beat in the study of all forms of heart disease. Hitherto, we have had to depend on the cumbersome cylinder drum or the finicky MacKenzie clinical polygraph. Jacquet has lately devised an instrument for this purpose, readily portable, well constructed, and clinically efficient. The practitioner will find it of much service. It must not be forgotten that the x-ray is of distinct service in the study of heart affections. Cooper has lately demonstrated at the San Francisco County Society plates showing distinct evidence of adherent pericardium; and of dilatation of the descending aorta behind the heart. The correlation of these plates with the symptoms and physical signs is of distinct aid in the diagnosis. He emphasized the infection theories advanced by Dr. Woods Hutchinson—probably often unrecognized or little-heeded sore throats, nasal catarrhs, alveolar pyorrheas, and urethral inflammations which by their summation of effect impair the cardiovascular system.

DR. G. W. NORRIS, Philadelphia, said that during the past winter he has seen three cases of Stokes-Adams syndrome, one of which he had an opportunity of carefully studying. He has made tracings coincidentally of the jugular and the radial pulses, as well as the cardiac apex beat. These tracings show very distinctly an extra number of auricular contractions. The auricular contractions were accelerated by deep inspiration, the ventricular rhythm was unaffected by this or by change of posture. He believes that by using the Jacquet polygraph we would be enabled to learn a good deal about cardiac diseases which has not previously been known, and that the tracings made by this instrument would prove not only of

scientific interest but also of practical utility, especially in cases of arrhythmia; perhaps, also, in instances of pericardial adhesion; and possibly in differentiating between true and false angina pectoris. As a clinical procedure this method of studying heart affections is rather new, and probably some time will elapse before all the data thus obtained will be properly interpreted, but he thinks that a new field has been opened which holds forth considerable promise.

DR. BERTRAM W. SIPPY, Chicago, mentioned a sign brought out by Dr. Davis, viz., systolic retraction and adherent pericardium. This traction is caused by the outer pericardium being attached posteriorly; the remaining portion of the pericardium is also attached to the thorax; therefore, when the heart contracts, it pulls in on the surrounding structures, pulling in the intercostal spaces and so producing systolic retraction. This, he said, may, lead to an error in the diagnosis of adherent pericardium. When systolic retraction occurs at the apex, that, in connection with other physical signs, is sufficient for diagnosis. But systolic retraction occurring at the base of the heart occurs not infrequently without adherent pericardium. Systolic retraction he considers as an evidence of adherent pericardium only when it occurs at the apex and not at the base, and in connection with other physical signs. The great increase of the work of the heart caused by this condition practically always is associated with incompetency; it must pull on the surrounding tissues, thus handicapping it. Dyspnea, cyanosis, etc. will be present.

DR. DE LANCEY ROCHESTER, Buffalo, said he wished to speak of symptoms which he has observed in connection with pericarditis and to which Dr. Davis had not referred. First, there is pain in the interscapular region posteriorly between the shoulders, a symptom that he has found in many instances.

In reference to pseudoangina and true angina pectoris he agreed with Dr. Tyson that there is difficulty in distinguishing between them. Dr. Rochester then reported the case of a woman, between 45 and 50 years old, who was visiting in Buffalo and who was seized with pain in the epigastrium. He was called and found that she had been indulging in a promiscuous supper, that there was a considerable amount of pain and that this pain was the only symptom; there was no fear of death, no dyspnea, or other symptom. The pain was relieved after the stomach had been emptied. The next day she did not feel very comfortable; on the next day she again was seized with pain, not only in the epigastric region, but in the chest and left arm, and this pain had to be relieved with morphin. Dr. Stockton was called in consultation and they could not find any evidences of disease of the arteries. There was no nephritis and no evidence of arterial disease. The heart was of normal size and the sounds were good. The next night she was again seized with pain and died before he got there. The autopsy revealed a ruptured heart with pronounced arteriosclerosis, a thrombosis of the left coronary and a marked thinning of the muscle at the apex of the heart where the rupture had occurred. The rest of the heart muscle was in good condition. There was an arteriosclerosis of the coronary arteries.

DR. STEPHEN S. BLACK, Pasadena, Cal., said that Dr. Sippy's remarks regarding the diagnosis of adherent pericardium by systolic retraction are very important, but that this is not the only thing which produces it. The apex beat is caused by a lengthening or straightening out of the aortic arch; also by rotation of the heart and, at the same time, by contraction of the ventricles. This all throws the heart against the chest wall. Any interference with the lengthening of the aortic arch, or contraction of ventricles, or rotation of the heart will cause a depression or retraction of the apex beat. He said he has seen very pronounced cases in which the apex was depressed during systole and there were no pericardial adhesions whatever.

DR. NATHANIEL B. POTTER, New York City, quoted statistics from the City Hospital Autopsy Records, 1906, to show that nutrition has to do with cause of cardiac hypertrophy.

DR. J. M. ANDERS, Philadelphia, remarked that Drs. Jones, Tyson and others objected to the term "pseudoangina," and he did not intend to defend this term. In the portion of his paper, which for lack of time could not be read, he had stated that, in view of the symptomatic relationship existing between true

angina and the false variety, rendering a differential diagnosis well nigh impossible in many instances, he would encourage the use of the term "pseudoangina" until additional light should be thrown on the whole subject. These cases of so-called pseudoangina might be regarded as hysterical in nature, as suggested by Dr. Tyson, but even severe precordial pain due to that cause must be separated from true angina for the sake of appropriate treatment. The case related by Dr. Rochester, which occurred in a neurasthenic person, later developed true angina and was similar to two cases reported in his own paper. His view, as previously stated, was that they hold fast to the provisional classification into pseudoangina and angina vera and separate the functional from the organic form whenever practicable.

OBSTRUCTION AT THE TERMINAL PORTION OF THE COMMON BILE DUCT

DUE TO CANCER OF THE AMPULLA. AN ANOMALY IN
THE CYSTIC DUCT. SIMILAR OBSTRUCTION DUE
TO PANCREATIC CANCER.

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Cases of cancer at the terminal portion of the bile duct within the duodenal wall are sufficiently rare and the signs are sufficiently characteristic to warrant the following report.

Jaundice and pain are the most frequent signs of disease of the bile passages, jaundice belonging especially to common duct disease. Musser found it present in 17 of 18 cases of cancer of the ducts and pain in 12 of the 18. In gall-bladder disease jaundice is present in but 20 per cent. of the cases.

In 16 cases of cancer of the ampulla on record, jaundice has occurred in all, while pain is mentioned as present in but three, and then of an aching or general abdominal character.

This may be partly explained by the fact that when the ducts are the seat of cancer I found a history of gall-stones in but 18 per cent. of 25 cases, while various authors give from 69 per cent. to 95 per cent. of gall-bladder cancers as showing gall stones.

Occasionally the jaundice is due to a lesion, in itself of no consequence, but so placed as to obstruct some important function of the body as, for instance, cancer of the tip of the bile duct or parasites or some kinking at this point. There have been described by a number of authors such primary cancers which originated in the mucous membrane of the ampulla of Vater, the clinical picture of which they claim to be distinctive.

The following case is of interest as one of this class of painless jaundice:

CASE 1.—A male, 66 years of age, a farmer, came in October, 1901, complaining of severe jaundice which had developed rapidly, within four or five days, without pain. This painless jaundice, loss of flesh and frequent bowel movements were the first evidences to him of any illness, but, on further questioning, the following details were obtained:

Two years before, he first noticed a tendency to looseness of the bowels. Four or five months later, the stools became pale-colored, "putty-like," foul-smelling, small in amount and unsatisfactory. By the end of the first year they occurred once or possibly twice during the night, and three or four times during the day. Nine months later he noticed a very slight soreness just below the eighth costal border, which has continued till the present time. Three weeks ago, he first noticed jaundice, severe itching and dryness of the skin. He had lost 40 pounds in weight, his usual weight being 200 pounds. He felt energetic, active, appetite good, and complained of nothing but the jaundice.

His family and past personal history were negative. He had never had a venereal disease, nor had he used alcoholics. He had never been troubled in any way with indigestion; nor had he ever had bilious colic, a previous jaundice, nor any serious pain or discomfort referable to the abdomen.

Physical examination showed temperature normal, pulse 68. The thoracic and abdominal organs showed nothing abnormal except the liver, the upper border of which was in the sixth, eighth and tenth spaces, but the lower edge could be felt rounded, two fingers' breadth below the ribs. The gall bladder was outlinable to percussion and palpable as a pear-shaped, slightly tender body, pointing toward the umbilicus.

The urine was 1,024 acid, dark coffee colored, and contained a trace of albumin, no sugar, a large amount of bile pigments, many large and small hyaline, cellular and pigmented casts, a few renal cells and leucocytes.

A blood count at 10 a. m. showed 6,600 white cells, 5,600,000 reds, and a hemoglobin index of 74 per cent. The normal digestion leucocytosis was present, the count being 10,000 after a meal.

This was the last of November. The patient was not seen again for six weeks, during which time he had a severe tonsillitis with abscess formation. When seen in January, 1902, his urine was scanty and contained a large amount of albumin, bile pigment and casts; no sugar. There was marked edema of the legs and abdominal wall, and a large amount of ascites.

The jaundice persisted until death. There was no pain except that, dull in character, due to the gall bladder distension. Death occurred January 30, thirteen weeks after the first appearance of jaundice; two years and three months after first noticing the frequency and loss of color of the stools.

A summary of his history is:

Two years and 3 months ago frequent putty-like, pale colored stools.

Six months ago soreness at right costal border.

Five months ago sudden painless jaundice, rapidly deepening in 4 to 5 days.

Loss of 40 pounds in weight in one year.

Death 2 years and 3 months after onset.

The diagnosis had been cancer of the head of the pancreas. At the autopsy made by Dr. Parker, formerly assistant in pathology at Western Reserve University, a cancer of the tip of the common bile duct was found within the duodenal wall. The gall bladder was greatly dilated, measuring 12 × 6 cm., and had fallen down till its long diameter was parallel to that of the liver. This explains its not being felt late in the disease. The common, pancreatic and cystic ducts were greatly dilated. The hepatic ducts were dilated up into the liver and were surrounded by a fibrosis.

An anomaly in the ducts was present in that the cystic duct passed alone, parallel to the hepatic duct, entering it just before the latter's entrance into the intestinal wall; also, the hepatic duct united with the duct of Wirsung in the head of the pancreas. The common duct, therefore, extended only from the pancreatic head to the duodenal wall, 1 cm. The bile papilla was very small. Dr. Parker says: "On opening the papilla a mass of dense hard white tissue is found. This tissue occludes all the entering ducts. It is fairly well localized, forming a patch about the size of a quarter." The growth was an adenofibroma. There was no metastases. The pancreas was of increased firmness.

The bit of duct between the junction of the common bile duct and the pancreatic duct and the bile papilla in the duodenum is called the ampulla of Vater. It is contained almost entirely within the duodenal wall as it passes obliquely through it. It is of slightly greater diameter (4 to 5 mm.) than the bile duct above and the orifice below. Its length, as measured by Opie, varies from 0 to 11 mm. In 11 cases in 100 the pancreatic duct opened on the duodenal surface by a separate orifice. This small duct common to the pancreatic juice and bile is the cavity in which gall-stones occasionally lodge and is the second most frequent seat of beginning