THE PUPIL SYMPTOMS IN THORACIC ANEURYSM.  
A CLINICAL LECTURE. RADCLIFFE INFIRMARY.

By WILLIAM OSLER, M.D., F.R.S.,  
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The patient before you attracts attention by his healthy appearance, a fresh complexion, iron grey hair, and a general bearing which suggests at once the man who has served "with the colours," whether on land or sea. To outward appearance the most vigorous person in the ward; you will all have noticed one peculiarity of great importance, marked inequality of the pupils. The right, the larger, is moderately dilated; both react to light, and on accommodation. It seems an instance of simple anisocoria. On more careful inspection no change is noticed in the eye-lids or eye-balls. The skin on the two sides of the face has the same tint and moisture, and the ears have the same degree of pinkness. Stripped he shows a strong frame; both sides of the neck are equal; there is no special congestion of the skin; impulse is visible in both carotids; the arms look natural; both hands are large and show slight congestion. Sitting directly in front and looking closely at the chest, nothing is to be noticed, but if he is turned, or if you move so as to look obliquely, there is seen to the right of the sternum, extending from the clavicle to the third rib, a slight heaving impulse. Once your attention is called it is easily recognised, and one may even see that the sterno-clavicular articulation is slightly moved with each beat. There is no prominence, and it is just the sort of impulse that requires careful inspection, good eyes, and a good light. I would ask you to note particularly that, sitting in front of him and looking directly at the chest, the pulsation is scarcely visible. The cardiac impulse is not seen, and there is no pulsation behind on either side of the spine.

On palpation the heart beat is not to be felt; the hand placed to the right of the sternum in the first, second, and third interspaces, feels a heaving impulse, which is appreciated.
slightly at the sterno-clavicular joint, on the clavicle itself, and is strong enough to move the fingers. It is not felt on the manubrium or in the notch above. There is no shock of either sound to be felt. Both carotids throb, the one not more than the other. The brachials and radials appear about equal on both sides. There is no retardation of the right radial pulse. The superficial arteries feel stiffened. The blood pressure, which has been taken by Dr. Gibson, is a little higher in the left arm than the right. On putting the trachea on the stretch with the fingers hooked under the cricoid cartilage, one appreciates immediately a very characteristic tugging, synchronous with the throb of the arteries. Percussion gives an area of impaired resonance from the clavicle to the third rib on the right side, and to a point three inches from the sternal margin. The area of cardiac flatness is not increased.

On auscultation, the sounds are clear at the apex and over the body of the heart; towards the base the aortic second sound is loud; over the area of impulse there is a soft, only just audible systolic murmur, and the second aortic sound is well heard. There is no murmur to be heard in the sternal notch or along the carotid arteries.

These are the main points to be determined by examination. I may add that there is nothing of any moment in the abdomen. The diagnosis is evident—an aneurysm of the arch of the aorta, projecting to the right, possibly involving the beginning of the innominate. The X-ray photograph taken by Dr. Sankey shows very clearly a large aneurysm extending above as high as the clavicle and projecting farther to the right than the examination suggests.

We may now ask a few points in his history. He has been a soldier, and has had fairly good health, but, as is so often the case, Mars and Venus have been in conjunction, and he had syphilis 15 years ago, not a severe attack, and for which he says he was well treated. He has had no symptoms for many years. He has worked hard; is a moderate drinker, and until a few months ago regarded himself as quite well. He then began to have irregular pains in the chest and shoulder, which were thought to be rheumatic. He had occasional flushes and giddiness, but no special shortness of breath. He
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has evidently not had very active symptoms, and the aneurysm, which has come on slowly and gradually, is now one of physical signs rather than of symptoms.

Our interest to-day is in the state of the pupils in relation to aneurysm. It is an old story which you will find very fully discussed in Gairdner's *Clinical Medicine*, and in his article in the first edition of Allbutt's *System of Medicine*, and by Ogle in his classical paper in the *Royal Medical and Chirurgical Transactions*, 1858.

We have been in the habit of explaining a condition such as exists in this patient in very simple terms; the unilateral dilatation is due to irritation of the dilator fibres of the sympathetic nerve by pressure of aneurysm. If the pupil on one side is contracted the dilator influences were supposed to be completely suppressed, and allowed the unopposed action of the sphincter controlled by the third nerve. Of late years we have found that the matter is not quite so simple; there are in reality three groups of cases.

I. Cases due to Involvement of the Sympathetic Nerve.—The distance from the right margin of the arch of the aorta to the cord of the sympathetic is only a few centimetres, so that one can readily understand how an aneurysmal sac growing to the right may involve the nerve. In reality in the post-mortem examination one very rarely sees the sympathetic cord compressed. Clinically, in the great majority of all cases of aneurysm, pupil features are present without other indication of the involvement of the sympathetic system. Only in a few instances, in my experience not more than four or five were there other signs, such as flushing and sweating of one side of the face, ptosis, and retraction of the eye-ball. In this patient, for example, there is no indication either of irritation or of paralysis of the cord of the sympathetic, and what you see is present in a large majority of cases of aneurysm with pupil symptoms, viz., simple dilation. It is possible, however, that the nerve may be irritated, as dilatation of the pupil on one side may be the sole indication of pressure. We see it sometimes in pneumonia, in tuberculous disease of the apex of the lung, or in a chronic pleurisy, in which cases the pupil symptoms usually occur alone. The X-ray picture shows the sac to pass far over the right, quite far enough, one would say,
to reach the sympathetic cord. Unilateral flushing, increased heat and sweating with mydriasis are less common than myosis with profuse sweating, and occasionally slight ptosis. The arm may be involved, and I have seen the skin of the hand wrinkled like that of a washerwoman; but these are very rare cases.

II. Cases due to Changes in the Vascular Condition of the Iris.—It has long been known that with a low blood pressure the pupils are large. On the other hand, small pupils are often seen in association with the high arterial tension of chronic interstitial nephritis, arterio-sclerosis, and old age. It has been much discussed whether changes in the blood vessels of the iris are accompanied with narrowing or dilatation of the pupil, and one well-known theory explains these variations as due to a diminution or increase in the contents of the vessels. Working on this theory, Wall and Ainley Walker¹ have studied a series of cases of thoracic aneurysm, and have come to the conclusion that the most common cause of anisocoria is unequal blood pressure in the ophthalmic arteries. They explain the relationship between the arterial blood pressure and the size of the pupils by the anatomical peculiarities of the vessels of the iris. "As Waller originally showed, they are spiral or zig-zag, so that during contraction or dilatation their lumen is not changed in calibre. It is a well-known physical fact that the raising of the pressure in a fluid tends to cause elongation and straightening of the tube. From this it follows that a rise of blood pressure in the spiral blood vessels of the iris would tend to lengthen them and lead to narrowing of the pupil, and vice versa, a fall in the blood pressure to shortening of the vessels and enlargement of the pupil." Local variations in blood pressure occur in aneurysm. The radial pulse on one side is often smaller, and inequality of the carotid and temporal pulses is by no means uncommon. In 26 cases of thoracic aneurysm in which notes were made concerning the relative size of the radial pulse and of the pupils, these authors found that in 11 the radial pulse was larger on the side on which the pupil was smaller; so it does not necessarily follow that because there is inequality

¹ Lancet, July 12, 1902.
of the radial pulses there must be a similar inequality in the ophthalmic arteries. They studied and compared the relative sizes of the temporal arteries, and found that in all their cases the smaller pupil corresponded to the larger temporal pulse. Experimentally, digital compression of one carotid sufficient to abolish or partially arrest the temporal pulsation was associated with gradual enlargement of the pupil on the same side, followed a little later by enlargement of the pupil on the other side. They attributed the dilatation on the same side to the immediate fall of pressure in the ocular vessels, which results from the compression of the carotid. The gradual enlargement on the other side is due to the general fall of pressure in the circle of Willis, which results from the same cause. The explanation of the anisocoria which these authors give holds good in a large proportion of all cases of aneurysm of the thoracic aorta. In this patient the situation of the sac suggests involvement of the orifice of the innominate or of the vessel itself, though palpation of the carotids and temporals cannot determine any difference between the two sides, and the blood pressure in the brachials is only a few millimetres lower in the right arm.

III. The Babinski Syndrome—the Association of Pupil Symptoms, Aneurysm, and Tabes.—In a third group the pupil features and the aneurysm itself are part and parcel of a syphilitic infection. The distinguished Parisian clinician, Babinski, in 1901, first called attention to certain cases of aneurysm in which the irregularity of the pupils, or the myosis, had nothing to do with compression by the sac, but were the ocular manifestation of a tabetic or a tabo-paretic state. In the cases which he reported,¹ both in women, both syphilitic, with aneurysm of the arch of the aorta, in one the right pupil was smaller than the left, neither reacted to light, but did on accommodation; the knee-jerks were absent. In the other case the left pupil was dilated, the right pupil was normal, and the light reflex was not completely abolished. The knee-jerk on the right side was lost. Since the publication of this paper Babinski’s syndrom has been used to designate a condition in which aneurysm is present, in association with tabetic features—the Argyll-Robertson pupil or unequal pupils

with absent knee-jerks or lightning pains. There have been several additional papers on the question, and the condition is now well recognised.

The man before you has had syphilis. There is a large aneurysm, and the pupils are unequal, and now on testing the knee-jerks we find that they are absent. He has never had any lightning pains, his station is good, and there is nothing else to suggest locomotor ataxia. We know that a large proportion of all cases of aneurysm in young and middle-aged men are due to a syphilitic mesoartitis. Absence of knee-jerks alone may be scarcely sufficient to warrant a diagnosis of tabes, but, taken in conjunction with the history and the presence of an aneurysm, we may say at any rate that this man is in the quaternary stage of the infection, in which among the earliest symptoms of locomotor ataxia is abolition of the reflexes. The pupil symptoms here are more likely to be vascular than tabetic. The light reflex is not abolished, and while one can make out no difference between the radials, carotids, or temporals on the two sides, the position of the aneurysm in the X-ray picture indicates that the innominate artery is involved, and the blood pressure on the right arm is lower than on the left side.

A more characteristic case seen last year was a professional man living in South America, aged 46, who consulted me for unpleasant sensations in the head and chest, and irregular shooting pains in the legs. He had had syphilis 20 years before, and considered himself cured. There was pulsation of the manubrium, trachial tugging, and well-marked signs of aneurysm of the arch, though the X-ray picture showed a condition suggestive rather of diffuse dilatation. Both pupils were contracted, and did not respond to light, the knee-jerks were absent, and he had had well-marked attacks of lightning pains.
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The Lumleian Lectures

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Delivered before the Royal College of Physicians of London

BY

WILLIAM OSLER, M.D., F.R.S.

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LECTURE I.
Delivered on March 10th.

INTRODUCTION.

MR. PRESIDENT AND FELLOWS,—Twenty-five years have passed since I stood here, a much embarrassed junior, as Goulstonian lecturer. I have always had a keen sense of gratitude to the College for according recognition to a colonial worker at the time of life when such an action counts for so much, and I recall the intense pleasure of my colleagues at Montreal that one of their number had been selected for the honour. The subject of those lectures came within the ken of the younger Fellows, whose work is, or should be, largely in the post-mortem room and laboratory. And now kindly time has moved me among the seniors, and I have to thank you, Sir, for the opportunity to deliver the course distinguished among all others in the College, since in these Lumleian lectures the incomparable Harvey laid the sure foundations of modern experimental medicine.

I make no apology for the subject I have chosen—Angina Pectoris. In a very special way it is our disease, having been first fully described at this College by the English Celsus, William Heberden, and in a manner so graphic and complete as to compel the admiration and envy of all subsequent writers.

Like books, diseases have their destiny. Could Heberden return for a month’s busy practice his surprise would be not less at the new cohorts of disease than at the disappearance of familiar enemies. How staggered he would be at the Nomenclature of the College! And he would be keen to write new commentaries upon old diseases with new names. How the word appendicitis would jar his critical ear, but how rejoiced he would be to see light on that dark malady, "inflammation of the bowels." Living through a century of
theory, he died at the outset of the great awakening in clinical medicine, bequeathing a precious legacy of experience greatly appreciated by several generations of students, and leaving in this College a precious memory which it is our delight to cherish.

Looking through the famous Commentaries, one is impressed with the value, with the rarity too, of the old-fashioned, plain, objective description of disease; and one is impressed also with the great gulf which separates the clinical medicine of to-day from that of our great-grandfathers. Page after page of the Commentaries are as arid as those of Cullen or of Boerhaave, and then we light upon an imperishable gem in the brilliant setting of a master workman, whose kinship we recognise with the great of old—with Hippocrates, with Aretæus, and Sydenham. Such a clinical gem is the account which Heberden read at the College, July 21st, 1768, "of a Disorder of the Breast," to which he gave the name "Angina Pectoris," based on the study of 20 cases. When he incorporated the description in his Commentaries (written in 1782) his experience had extended to 100 cases.

Historical details I have dealt with at length elsewhere, but in passing one must just mention the predecessors of Heberden, particularly Rougnon, the old Besançon professor. There is no question as to the nature of the case which he describes; you can read for yourselves, as through the kindness of Professor Roland, a distinguished successor of Rougnon at Besançon, I am enabled to show, for the first time, I believe, in this country the rare "Lettre," published in March, 1768. As Rougnon antedated Heberden a few months, so did Morgagni precede Rougnon, and in his excellent report the symptoms are even more fully described, including for the first time the brachial numbness and the aortic lesion; and we get back to classical days if Seneca's disease, which he calls meditatio mortis, and the "paradoxon" of Erasistratus, are regarded as angina.

For more than a century the chief contributions to the pathology of the disease have been made by members of this society, and to-day our Fellows number many of its best known students, among whom, Sir, you rank primus inter pares. And yet so far as I can ascertain angina pectoris has never been formally considered in one of the College courses. It is, too, a disease for a senior to discuss, since juniors see it but rarely; indeed, I had reached the Fellowship before I saw a case in hospital or private practice. And then I take it that in this course the College wishes an expression of opinion on some affection to which the lecturer has paid special attention. Circumstances have given me a somewhat unusual experience. The lectures published in 1897 were based on a study of the literature and 60 cases; since then I have seen 208 additional cases, and I propose to present very largely my own impressions of the disease.

Let me ask at the outset, What is angina pectoris? Who
will give an answer to satisfy all of us? The subject is full of knotty problems, which lend themselves to speculation. I could wish for a more active scientific imagination that amid webs of fancy I might entangle and darken the maturec counsels of some of my distinguished auditors. But with neither the brains nor the inclination for such a task, in a more modest flight I shall consider it as—A disease, characterised by paroxysmal attacks of pain, pectoral or extra-pectoral, associated with changes in the vascular walls, organic or functional.

Primarily an affection of the arterial system—of the pump and the pipes, of the system in which are literally the issues of life and death—its protean features cannot be understood unless we remember that between the chief parts of this system, the heart and the arteries, there is no essential difference, since the arteries are only a long-drawn-out heart and the heart but a bulbous expansion of an artery. A physical unit, and worked as such, it is controlled at every moment by an outside mechanism, an elaborate system of nerves which penetrate every part, and even lose themselves in its structures.

The problem before us is the anginal paroxysm in all its grades, from the trifling sense of substernal distress to the vascular iotus by which a man is felled as with a club. After a few etiological details I shall discuss briefly the clinical types and certain extra-cardiac features of the disease. In the second lecture I shall consider the pathology, and in the concluding one speak of prognosis and treatment.

GENERAL ETIOLOGY.

Has angina pectoris increased in the community? Has the high-pressure life of modern days made the disease more common? There is an impression among consultants in the United States that there has been an increase of late years, a view not borne out for this country by the figures available. In 1908 there were 929 fatal cases in England and Wales—617 men and 312 women. For the 20 years, 1888 to 1907, the average number of deaths was about 700; in 1905 the number rose above 800; and in 1907 to 942; but the average number of deaths per 1,000,000 living has not materially increased, ranging from 20 to 25, but in 1907 it reached 28. In England the population of the registration districts of England and Wales is about 35,000,000. The statistics for the United States in a registration area, embracing 45,000,000 of people, show a decidedly greater prevalence of the disease. The total deaths in 1908 were just under 3000. But the average number of deaths per 1,000,000 of population have not varied much within the past ten years, but it is more than double that of England and Wales, ranging from 66 to 70 per 1,000,000 of inhabitants.
It is not a disease for which hospital figures are of much service, and yet it is interesting to compare the large institutions on the two sides of the Atlantic. At the Montreal General Hospital in 10,934 admissions for the ten years 1900 to 1909 there were 6 cases diagnosed as angina pectoris. At the Royal Victoria Hospital, Montreal, among 10,510 admissions in ten years in the medical ward there were 9 cases. At St. Bartholomew's Hospital in 1907 there were 2 cases in 2602 medical admissions; in the same year at St. Thomas's Hospital in 2261 medical admissions there was only 1 case. This gives an average of 1 case a year in the wards of the large general hospitals. The figures at the Johns Hopkins Hospital are scarcely available for comparison, since they embrace a very large number of patients admitted to the private wards, and even into the public wards many of the farmer class are admitted from the country at large.

These figures bear out a remarkable fact with which we are all familiar—that angina pectoris is an affection of the better classes, and not often seen except in private practice. During 10 years I did not see a case at the Montreal General Hospital, and only one case at the University Hospital, Philadelphia; and I have no notes of a case seen at the large Philadelphia hospital. It is only as the consultant's work increases that he begins to see the disease, and then a man in active practice may see 10, 15, or more cases in the course of a year. This was about my average, and I see from the statements of our President, and of the late George Balfour of Edinburgh, that this is about the figure reached in this country by the consultant with recognised cardiovascular leanings. Once there was the unusual experience of eight cases in a month (May, 1899), three of which died in the same street within a short distance of each other, or, to be more accurate, one died on the steps of the cathedral, the other two in adjacent houses not far away.

Let me give as briefly as possible my personal statistics. I have notes of 268 cases in all—231 men, 37 women. If we recognise, as was my custom, mild neurotic or pseudo, and a grave organic or true form, there were of the former 225, and of the latter 43. I have not counted *les formes frustes* unless a case had subsequent severe attacks. As Heberden remarked, women are rarely affected, only 3 of his 100 cases. Of the severer form of 225 cases there were only 14 women. On the other hand, of the minor type, of 43 cases there were 23 women. It is somewhat surprising to see that in this country in the registered fatal cases of angina for the past 20 years the ratio of women to men was 1 to 1.8—5133 women to 9303 men.

The age incidence is late, the largest number of cases occurring in persons over 50. Of the 612 deaths in England and Wales, only 36 occurred between the ages of 35 and 45; while between 45 and 65 there were 291 deaths. In my list
the age was much the same. There were under 30 years of age, 9; between 30 and 40, 41; between 40 and 50, 59; between 50 and 60, 81; between 60 and 70, 62; between 70 and 80, 13; above 80, 3. In women the age incidence is, on the whole, a little lower than in men.

An interesting point in my series relates to the race incidence. Of 268 cases 37 were in Jews. Nowhere in the world are members of this gifted race seen to greater advantage than in the United States, where the opportunities of a rapidly growing country give scope to their exceptional genius for business. Living an intense life, absorbed in his work, devoted to his pleasures, passionately devoted to his home, the nervous energy of the Jew is taxed to the utmost, and his system subjected to that stress and strain which seems a basic factor of so many cases of angina pectoris. It is only fair to state that this high percentage scarcely represents a true state of affairs, since certain circumstances gave me an exceptional clientele among the Hebrews.

Angina in doctors.—A point that stands out prominently in my experience is the frequency of the disease in our profession. For the same reason doubtless that Sydenham gives for the incidence of gout "more wise men than fools are afflicted," angina may almost be called "morbus medicorum." 33 of my cases were in physicians, a larger number than all the other professions put together. Curtin in his study of 60 fatal cases notes that a fourth were in physicians. This large percentage in my list may in part be attributed to the circumstance of the publication of lectures on the subject in 1897. But the frequency with which doctors die from the disease has become the subject of common remark. From John Hunter onwards a long list of most distinguished men have been its victims. Not to mention the older physicians, among our contemporaries was Nothnagel, himself one of the ablest students of the disease, whose last act in life was to describe his own fatal attack. A tragic interest relates to this incident in the career of the great Vienna clinician. I do not know that the note has ever been transcribed in English; it reads as follows: "Anginal attacks with very severe pains. Pulse in the attack very variable, at one time slow, 56 to 60, quite regular, high tension, and then again rapid, 80 to 90, tolerably even and regular; then again quite unrhythmic, unequal at one time, rapid another, slow with changed tension. The first sensation of this attack dates three or four years back, at first slight, gradually becoming more pronounced. Very severe attacks with great pain have only come on within the last five or six days. Written on July 6th late in the evening, after three very severe attacks." Within a few

1 Transactions of the American Climatological Society, vol. xxiii.
hours after this note the end came. Charcot, the founder of modern neurology, died in an attack in the arms of his friend Straus, who himself succumbed to the same disease not long after. The distinguished neurologist Joffroy died from it in Paris last winter. Our much-beloved friend and fellow, Cullingworth, was its victim, and the list could be much extended. The most brilliant and devoted physician of his generation in the United States, the late William Pepper, died with coronary arteries like pipe-stems. The Provost, indeed the maker, of a great University, the very head and front of every important public movement in a city of a million inhabitants, a universally sought consultant, an enthusiastic teacher, a prolific author, in him was incarnate the restless American spirit, which drove him into a premature grave at the height of his career at the comparatively early age of 55.

I have looked over carefully the notes of the 33 cases to see if any factors could be said to favour. Only 7 were above 60 years of age, one a man of 80 with aortic valve disease. The only comparatively young man in the list, 35, was seen nearly 20 years ago in an attack of the greatest severity. Worry and tobacco seem to have been the cause. He has had no attack now for years. Two cases were in the fourth decade, 13 were in the fifth, and 11 in the sixth.

For the purpose of this analysis we may exclude the cases above the age of 60, after which age no man, much less a doctor, need apologise for an attack of angina pectoris. Neither alcohol nor syphilis was a factor in any case; of the 26 cases under 60, 18 had pronounced arterio-sclerosis and 5 had valvular disease. In a group of 20 men, every one of whom I knew personally, the outstanding feature was the incessant treadmill of practice; and yet if hard work—that "badge of all our tribe"—was alone responsible would there not be a great many more cases? Every one of these men had an added factor—worry; in not a single case under 50 years of age was this feature absent, except in Dr. G., who had aortic insufficiency, and who had had severe attacks of angina years before, probably in connexion with his aortitis. Listen to some of the comments which I jotted down of the circumstances connected with the onset of attack: "A man of great mental and bodily energy, working early and late in a practice, involved in speculations in land"; "domestic infelicities"; "worries in the Faculty of Medicine"; "troubles with the trustees of his institution"; "lawsuits"; "domestic worries"; and so through the list. At least six or seven men of the sixth decade were carrying loads light enough for the fifth but too much for a machine with an ever-lessening reserve.

It is a significant fact that in Ogle's well-known study

2 Transactions of the Royal Medical and Chirurgical Society, vol. lxix.
"Statistics of Mortality in the Medical Profession," among 3865 deaths 444 were undefined diseases of the heart and circulatory system, though only 34 deaths were specified as due to angina pectoris. The same dominance of cardiovascular disease is indicated in the Registrar-General's Report.

**Clinical Types.**

It is interesting to look over a long series of some one affection with a view to classification. Angina pectoris offers notorious difficulties, and I do not suppose there are any two of us who would agree.

As far as symptoms are concerned my cases fall into three groups: (1) Les formes frustes; (2) mild; and (3) severe.

1. The mildest form, "les formes frustes" of the French.—Substernal tension, uneasiness, distress, rising gradually to positive pain, a not infrequent complaint, one, indeed, from which few of us escape, is associated with three conditions. Emotion is the most common and the least serious cause. How often does it happen on getting up to speak, or when beginning to read a paper, that a man experiences a sense of tension just beneath the breast bone, a curious indescribable feeling, not of pain, yet sometimes working to a degree of uneasiness that is only relieved by firm pressure? The slight associated pallor indicates a vaso-motor disturbance which may increase, and a man may have to stop speaking; indeed, I have known instances in which fainting has occurred. In one of my physician-patients, a well-known author, the attacks of true angina began in this way; while lecturing he would experience a feeling of substernal tension and for years had nothing else and had it under no other circumstances. He could play golf and ride, and do an extraordinary amount of work without any uneasiness. Only a single action may bring it on. Dr B. could lecture and hold his clinic without experiencing any difficulty, but if he read a paper before the Medical Society, or if he gave an address in public, the substernal tension was certain to come on. In this form the condition is very transient; while sometimes a danger signal, in many cases it may be disregarded altogether. Not so the second form, in which this substernal distress is associated only with muscular effort—the slightest ascent, the extra round of golf, a sudden hurrying, as to catch a train. Much more frequently the precursor of angina, it is remarkable for how long a person may have slight attacks without aggravation. In a tranquil life the individual is perfectly comfortable. As old Dr K—of Philadelphia used to say to me, "I can stand anything in life but a hill or a stair." A Mr W—could walk a mile

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3 Decennial Supplement, Part II., 1908.
from his home to his office, but for several years he could not walk back on account of the slight up-grade. He never had an attack of angina, though he died from myocarditis and a dilated heart. In these emotional and muscular types of the "formes frustes" the condition is usually transient. But there is a third variety, the *high-pressure form*, in which day after day, for weeks or months, the individual may never be free from a sense of tension beneath the breast-bone. It is not pain; it has no accurate localisation except that it is directly substernal; there is no radiation; it is not increased by emotion or by exertion, but obtruding itself into consciousness as an unpleasant reminder it means just one thing—that the machine is being driven at too high a speed. The general manager of one of the railways of the Southern States used to call it his "hot box"—i.e., his "hot axle." It is met with in men who are burning the candle at both ends—working hard at business or in a profession and at the same time treading the "primrose path." It is not always of sufficient severity to cause the patient to consult a physician. The Sunday rest may cause its disappearance. Not always a high-pressure affair alone, it is aggravated by worries, particularly the possibility of not carrying through some big scheme or the onset of a financial crisis. With the harness off it may disappear completely. One man writes: "The second day out on the steamer from New York I am free, not the slightest sensation of my enemy." In looking up the history of these cases, in three only did severe angina follow. I have not included "les formes frustes" in my list unless there were other features, such as definite paroxysmal attacks. It is only occasionally severe enough to make a patient seek advice. It is significant that of five cases of which I have notes of the blood pressure, in all it was above 180 and in one 250.

2. *The mild form.*—Under the mild form, *angina minor*, come 43 cases of my series. I have grouped under these the neurotic, vaso-motor, and toxic forms, the varieties which we used to speak of as false or pseudo-angina, a term which I agree with Gibson and others is best given up, since, as I hope to show in the next lecture, the basic features of all forms are identical. Still, it is a very useful exoteric term, a comfort to the patient and his friends. The special features of this variety are—the greater frequency in women, the milder character of the attacks, and the hopeful outlook.

3. *Severe angina, angina major.*—This group is represented in my series by 225 cases, of which 211 were in men. The two special features here are the existence in a large proportion of all cases of organic change in the arteries and the liability to sudden death. It is not easy, nor is it wise, to class cases by symptoms alone, but all the same there is
an interest in so doing, and my list may be divided into four groups.

A. Cases in which death occurred in the first or second attack, or in connexion with a series of rapidly recurring attacks—the so-called status anginosus. We do not know how many instances of sudden death in the street or in bed at night are due to angina pectoris, but only three cases in my series died in the first attack. The mode of death is not always the same. In certain cases it is the most rapid we see—without warning, or after a few minutes of unpleasant substernal sensation, or possibly in some act, combining intense emotion with muscular effort, there is a rapid change, a sudden unconsciousness, a stony stare, a slight change in the facial expression, and then with two or three gasps all is over; no pulse is to be felt at the wrist; the respiration stops, but even when the patient is apparently dead a feeble heart impulse may be felt or faint heart sounds heard.

B. The patients have had a series of characteristic attacks ranging from two or three to scores during the course of a few months or a year or more, and in a severe paroxysm or in a series death occurs. The final event has not the same suddenness, nor is there the rapid loss of consciousness; the patient may indeed be moribund and quite conscious, though this is unusual. The mode of dying in these cases is very remarkable. In a number of instances I have made careful notes. Two are worth quoting:

On Thursday, May 25th, 1899, while at work in the ward, Dr. Knox called me to see a patient in an attack of angina. I found a man, aged 41, who had been admitted the previous day complaining of pain in the heart. He had been a heavy worker, a large eater, had not had syphilis. Five years ago while rowing he had an attack of pain and shortness of breath, which lasted for a few minutes. On and off since then similar attacks have occurred, always brought on by exertion, now even by very slight effort. He had aortic insufficiency, and a very soft low tension pulse. About 9.30 A.M. his hands and feet had become cold and a little cyanosed, and he had a slight attack. It continued on and off all the morning. I saw him at five minutes past 12; he was propped up in bed moaning with pain, but was not sweating; the pulse was soft, regular, and feeble—100 to the minute, the left smaller than the right. Everywhere over the chest in front and back were medium-sized râles; the pulmonary resonance reached almost to the costal border on the right side, and the superficial cardiac flatness was completely obliterated; there was a soft apex systolic murmur, and a soft diastolic aortic heard along the sternal border. At 12.15 he had a sudden collapse, became pulseless, the features set, and he gave one or two gasping respirations, which recurred at intervals of about five minutes. No pulse could be felt in carotids or brachials. Remembering the remarkable case reported by Sloan, I performed cardio puncture, thrusting a long thin aspirator needle into the heart through the fourth right interspace. This was followed at once by one or two faint inspirations; the needle showed a cardiac impulse very plainly, beating 52 to the minute. At 12.25 the needle was moving much more slowly and more feebly—44 to the minute. A saline injection was made directly into a vein. At 12.28 the cardiac beats as shown by the needle were still regular—32 to the minute,
without any tremor in the interval, such as might be given by fibrillation. At 12.37 the excursion of the needle was a little stronger. No heart sounds could be heard; no pulsation in the veins of the neck. At 12.44 I injected a tenth of a grain of strychnine directly into the wall of the left ventricle. The needle in the right fourth interspace continued to show definite movements, gradually getting feebler, and stopped at five minutes past one, 50 minutes from the onset of the collapse, and exactly 45 minutes from the last inspiratory gasp.

Nov. 4th, 1901.—I was sent for hurriedly this morning to see a stout, healthy-looking man, aged 57, whom I had known for some years, and had seen at intervals. As I entered the room at 8.15 the patient apparently was in articulo mortis; indeed, I thought he was dead. The eyes were fixed, the pupils dilated, the face of an ashy colour, and he was not breathing. Seeing my startled look, Dr. Atkinson, who was just preparing a hypodermic injection of ether, said: "It is all right; he will come to; he has had four such attacks in the night." There was no pulse in either radial, but listening over the heart one could hear feeble distant heart sounds. In about a minute (it seemed to me longer) the patient began to breathe; inspiration and expiration were somewhat noisy and deep, and accompanied everywhere with loud bronchial râles. He did not recover consciousness. He became more livid in the hands and face, and the pupils became contracted. The pulse could be felt, small at first, but it then became of much better volume and ranged from 56 to 68. In the course of ten minutes the breathing became less laboured; his colour improved and lost the ashy look, but he did not regain consciousness. The heart sounds could be well heard; the pulse was full and soft, 62. At 11.15 the patient died in another attack. He had not regained consciousness. He had had his first attack of angina on Nov. 2nd.

C. A chronic form, represented in my series by 10 cases, all of which were characterised by frequent recurring attacks over a period of more than 10 years. John Hunter, you remember, had had his first seizure in 1773, 20 years before his death, and he had many in the intervals. One special feature of this form is the frequency with which certain special actions are associated with the attacks. A patient may be perfectly comfortable and remain free if he leads a tranquil life with little or no muscular effort: a slight hill, the act of dressing himself, may be sufficient to bring on an attack; or in another patient an indiscretion in diet. This is a form with which the patient may feel comfortable for a great many years. I have had several friends, two of them medical men, who have managed very comfortably for more than 10 years, in spite of the liability to attacks. On the other hand, they may be among the most distressing cases we see. A Mr. D. of Wilmington lived a life of martyrdom for more than 10 years; emotion, cold, exercise, eating, would bring on the attacks, and his existence was a burden—not a week passed without an attack. He was thought to be neurotic and hysterical, though a man of 60. He had remarkable quivering of the fingers in the attacks, and on several occasions became unconscious. He threw himself about and was in intense distress during the paroxysm. I saw him in an attack and felt sure the condition was serious. The left radial pulse became very much smaller than the right. He died suddenly after nearly 11 years of suffering. Both coronary arteries were calcified.
D. A group of cases, not large, I am sorry to say, in which after attacks of great severity, recurring for months, or for as long as two years, complete recovery takes place. These are cases I shall deal with more fully in speaking of prognosis.

EXTRA-PECTORAL FORMS.

Angina pectoris is an affection of the arteries. The studies of Nothnagel in 1867 on the vaso-motor phenomena of the attacks widened enormously our conception of the nature of the disease, and we have come more and more to regard the symptoms of the attack as an expression of a vascular crisis, to use the apt term introduced by James Collier. I shall try in the next lecture to discuss the disease from this point of view. Meanwhile here may most conveniently be considered certain extra-pectoral, or, more properly speaking, extra-cardiac features, which have a direct bearing upon our conception of its pathology. Following this wider conception it is interesting to note in the literature the use of such terms as "angina abdominis," "angina cruris," "angina brachialis." In looking over my list I find a considerable number of instances in which prominent features of the disease were extra-pectoral or there were symptoms suggestive of vascular disturbance in distant parts. It is difficult to make a classification of the symptoms, and certainly one cannot take for granted that they were always due to vascular crises. But I may roughly group the cases into those with (A) peripheral, (B) abdominal, (C) pulmonary, and (D) cerebral features.

A. Peripheral angina.—Heberden first recognised that the patient could die from angina pectoris without any pain in his chest. In his Commentaries he describes the case of a man, aged 60, who began to feel, while walking, an uneasy sensation in the left arm, never while in a carriage. After continuing for ten years it would come upon him two or three times a week at night, and he would have to sit up for an hour or two. In all other respects he was healthy and strong, and he never had a pain in the chest. Then he added: "This disorder, its seat excepted, perfectly resembled the angina pectoris, gradually increasing in the same manner, and being both excited and relieved by all the same causes. He died suddenly without a groan at the age of 75 years." In the case of Lord Clarendon's father, as noted by Blackall in his famous book on Dropsies, the pain was brachial, even in the fatal attack.

Four cases in my series presented in some degree this brachial peculiarity. In L. N. H., aged 58, the pain began in the middle of both forearms; when walking he would be warned at once of the onset by sharp pains appearing simultaneously about the middle of both arms; if
he stopped to look at a window the pain would disappear, if he attempted to proceed it would increase in severity, and he would begin to feel faint; sometimes then the pain would extend to the chest. The first occasion on which I saw him the pain was entirely in the arms; there was no cramp, and he pointed to a position about the centre of each forearm. There was moderate sclerosis of the brachial and radial arteries. In Mr. D., aged 42, the pain always began in the left elbow where it would sometimes stay, though more frequently it extended up the arm to the heart.

Dr. M. C. G., aged 69, insisted that the pain which "pulled him up short" on exertion was in the elbow, or rather at a point 2 inches above it, in the biceps muscle itself. So severe would it sometimes be that he would turn pale and sweat. He subsequently died in an attack.

In three instances the chief symptom was at first in the legs. My distinguished friend, Dr. W. W. J., for many years the leading physician of Washington, D.C., at the onset of the attacks had remarkable pains, with numbness in the left leg; there were no cramps, but the pains were sometimes intense, and he called it his "signal symptom." In a letter containing a very accurate analysis of his attacks he says that the painful sensations in the left leg seemed to initiate the outburst, though sometimes the pains remained limited to the leg. In F. L., aged 58, whom I saw in many attacks, some of maximum severity, they began with pains in the legs. I never saw a patient so drenched with sweat in the paroxysm, he was literally dripping, and could not have been more soaked had a hose been turned upon him. As he expressed it, he did not know which caused him the greater trouble, the painful cramps in the legs or the pain in the chest. He had indeed typical features of intermittent claudication, as the pain, sometimes with, sometimes without, actual cramp in the muscle, would pull him up short in the street.

It is a noteworthy circumstance that all the vaso-motor phenomena, even to fainting, may be associated with extrapectoral pain. Colonel E., aged 66, had agonising attacks of pain without cramp in the left leg, in the first of which he turned pale and fainted. The true nature of the attacks was not recognised until he began to have substernal pain and angina pectoris, in an attack of which he died.

I witnessed a remarkable attack in which the pain was limited to the right pectoral muscle. The man, aged 55, when shown into my consulting-room had an ashen grey colour and looked faint. He said at once: "Doctor, I am in an attack." Though his pulse was 172, and he looked faint and was sweating, he preferred to walk about. I was apprehensive lest he should drop dead on the spot. He pointed to and grasped the right pectoral fold, repeating the words, "Here is all my trouble." He had had attacks of great severity for four years. The pain always began in the
right pectoral muscle, and sometimes stayed there, but if very severe it went down the right arm. In the attack in which I saw him the skin was not hypersensitive, there were no tender spots anywhere, but the muscle itself was very sensitive; he winced at once when it was touched. It would remain sore sometimes for hours after the attack had passed off.

In only one of these cases were the features those of intermittent claudication, angina cruris, as Walton terms it, which has close analogies with angina pectoris, particularly its paroxysmal character, the pain, which is not necessarily associated with cramp, and the cessation when the patient comes to rest. In a syphilitic woman, aged 37, admitted on March 7th, 1900, with severe angina, in two attacks in December the left foot and leg became oedematous and painful. The arteries and veins of the limb were normal.

Pain in the testicle, sometimes with swelling, is mentioned by one or two writers as an occasional feature during the attack. In the case of our late colleague, Dr. C., the pain was sometimes very severe in this region, and the attack would even begin there. It is possible that the pain may be limited to the testis; at any rate, the following is a very suspicious case. A man, aged 56, while speaking at a meeting, was seized with an agonising pain in the left testis, and became pale and faint; there was no swelling, no tenderness, no radiation of the pain, but his condition must have been very serious, since the doctor did not think he would survive. He had attacks at intervals of every two or three months. Four days before I saw him he had a very severe paroxysm, in which he became faint and sweatend, and for the first time had a sense of tension and distress just below the ensiform cartilage. He was extremely feeble and collapsed, and the attack lasted for 12 hours. He had slight arterio-sclerosis, with accentuation of the aortic second sound.

B. Angina abdominis.—That angina attacks may begin in the abdomen has long been recognised. Several writers have called attention to the similarity of the gastric crisis of locomotor ataxia to angina. In my series there have been three groups of cases with abdominal symptoms. An important form is met with in nervous and hysterical patients with the combination of throbbing, tender, and mobile abdominal aorta and recurring crises of gastric pain. The pains may radiate to the chest, or gastric and thoracic crises may alternate, as in an extraordinary case in a lad aged 17—"a bundle of nerves"!—who nearly died in the paroxysms. The extreme tenderness of the aorta in some of these cases led Potain to suggest the existence of aortitis, while the radiation of the pain, &c., has given the term "syndrome solaire" to the group of symptoms. In some 10 or 12 cases in men of middle age the anginal symptoms at first were abdominal, and the true nature was not suspected. In
many cases the attack starts in the upper abdomen. As Mr. C. expressed it, "a wave starts here"—pointing to his stomach—"and passes up with a feeling of fright, and when it gets beneath the breast-bone it cuts me short as though the machinery of life had stopped." In my "Lectures" I have reported several cases of the pseudo-gastralgic type, as Huchard calls it, but in reality Leared in 1867 first called attention to these disguised cases. The difficulties in diagnosis may be very great, particularly in the form which resembles the tabetic crisis. A Mr. P., aged 58, seen on Feb. 11th, 1897, had attacks of cramp-like pains in the upper abdomen passing to the back, in which he became completely prostrated and sweated and vomited, and only morphia gave relief. The pains began just under the ensiform cartilage but never ascended. He had not had syphilis, the knee-jerks were present, the pupils were normal. He had aortic insufficiency and sclerotic arteries. There was no lead-line. He was a very healthy, robust man, only every month or few weeks he would be prostrated in an attack, in one of which, Dec. 20th, 1899, he died. In three other instances in the series gastralgia had been diagnosed, in one the malarial form, but the character of the attacks altered and left no doubt as to the nature of the trouble. And lastly, I have a group of three cases in which attacks of typical angina pectoris were complicated with abdominal pains, like gall-stone colic and jaundice. The combination may be quite accidental, but abstracts of the cases are worth recording.

Captain M——, aged 60, seen Jan. 13th, 1897, had had in 1890 attacks of angina pectoris of the most characteristic type. In 1895 he had pains in the region of the liver with jaundice; these recurrent at intervals, and he was operated upon by Mr. Mayo Robson at Leeds, May, 1895, but no gall-stones were found. He consulted me for pain in the abdomen, crossing from one side of the costal arch to the other, like a band or constriction; at times it became very aggravated. Food made no difference, and there was no tenderness on pressure and no enlargement of the liver. While more or less constant the abdominal pain comes on in spells, and sometimes makes him feel faint and sick at the stomach.

Mr. A. W.——, seen April 4th, 1903, aged 59, in the first attack of angina four years ago had pains in the abdomen of great severity, and he was jaundiced after the attack. It was supposed to be gall-stones. Four months ago he had a very bad night, with attacks of pain in the chest and much discomfort in the epigastrium; on the following day he was jaundiced. The curious thing was that he could not differentiate the pain in this epigastric attack from the furious seizures of definite angina, in many of which I saw him during the six weeks that he was in hospital. He died in an attack of angina some months later.

Dr. J. C. T., aged 64. After a day or two of indigestion and irregular pains had on Oct. 27th a severe attack of pain in the chest, with extension into the arm, evidently of great severity. On the 30th the pain was more abdominal, and he was very sensitive over the region of the gall-bladder; on Nov. 1st and 2nd the pain was abdominal, and tenderness marked over the gall-bladder, and he had become jaundiced. This relieved our minds, as we were afraid from the character of the early attacks that it was angina. By Nov. 9th he was very much better, and the pains had almost disappeared, and at 6 o'clock when I
saw him he seemed nearly well; but at 8 o'clock, while his son and the nurse were in the room, he gave one or two short, quick, sighing inspirations, his head dropped on his chest, and he died instantly.

C. Respiratory features of angina.—The pulmonary symptoms of the attack have been carefully studied by many observers. A man may die from angina without any change in the respiratory function other than abrupt cessation; indeed, the death may be at the medulla, so suddenly does respiration cease, even for nearly an hour before the heart ceases to beat. Ordinary cardiac asthma is a common symptom—the orthopnoea, the light cyanosis, and the feeble pulse of myocardial weakness. Cheyne-Stoke breathing is not infrequent, and in the status anginosus may be present for weeks. But there is another type much more characteristic and quite unlike the ordinary cardiac asthma: it is paroxysmal, occurring abruptly, often at night; it is not associated necessarily with signs of myocardial weakness; it is accompanied with very high tension; it may alternate with attacks of angina or come on in one, and sometimes bears the stamp of an acute oedema of the lungs. There are four special features.

First, the universal distension of the lungs in an acute emphysema as Goodhard described it; the inspiratory excursion is limited; expiration is prolonged; in one patient the chest even looked larger; and the increase in the volume of the lungs may be demonstrated by percussion. The condition is what von Basch has called "lungenschwellung" and "lungenstarrheit," a state which he believed to be due to distension of the capillary network, with swelling of the alveolar walls.

Secondly, the rapid onset of the physical signs, like those of an acute attack of bronchial asthma, wheezing, fine bubbling râles, and prolonged expiration. Within ten minutes the attack may be in full swing.

Thirdly, acute oedema of the lungs may follow, indicated by a great increase in the bubbling râles and the rapid expectoration of large quantities of a thin, frothy, sometimes blood-tinged, liquid. The attacks may be transient, lasting only for a few hours, and give a very special pulmonary stamp to the attack.

Fourthly, increased blood pressure, rising to 250 millimetres and over (Riva-Rocci). In one case (Dr. J. H. K.) the record was 340 millimetres during an attack, in the intervals it was 250. On three occasions he brought up large quantities of a clear fluid, with blood.

In May, 1899, I saw on several occasions with Dr. Atkinson Mr. G., aged 65, who had had his first and very
characteristic attack while walking up a hill on April 27th. It was probably an attack associated with acute infarct of the ventricle, as a pericardial rub was detected the next day. On the first occasion I saw him he was propped up in bed, wheezing audibly, expiration was prolonged, and cardiac flatness was obliterated. The pulmonary resonance on the right side extended to the costal margin. He was coughing and bringing up a considerable quantity of frothy, liquid expectoration. The pulse was full, regular, 100, and of high tension; the heart sounds were muffled and obscure; he had at the time no pain, but this acute condition of emphysema had followed the attack in the morning; within a few hours it had disappeared and Dr. Atkinson thought that there was a definite reduction of the lower limits of the lungs.

The condition may be associated, as just mentioned, with extraordinarily high tension. A man, aged 43, was admitted to Ward C, Nov. 17th, 1900, complaining of shortness of breath and attacks of substernal pain. He weighed 190 pounds, was a very heavy eater, and had not had syphilis; the blood-vessels were sclerotic; he had loud aortic second sound; the blood pressure was very high. I saw him in an attack of pain on Dec. 11th; he was not cyanotic, the pulse was regular; very high tension; the heart sounds were clear, loud, and sharp; he then turned pale and sweated, and the pulse at the wrist became almost imperceptible. He began to wheeze, and in a few minutes one heard on auscultation high-pitched sibilant rãles mixed with fine crepitations. These were very intense and sounded close to the ear, suggesting at first a friction rub; the percussion note over the lungs became hyper-resonant and on the right side reached almost to the sternal margin. A hypodermic of morphia relieved his pain.

The hyper-resonant condition of the lungs persisted for two days; a remarkable change in his facial expression occurred, as after the attack he had for days retraction of the upper eyelids. A Mr. C., aged 62, who had very characteristic attacks of angina pectoris, had several severe but transient pulmonary attacks, in one of which he had hæmoptysis.

One of the most remarkable cases was that of a coloured woman, aged 54 years, who was in the hospital for several weeks, and who died in an attack on Jan. 28th, 1897. She would wake at night from a sound sleep with pain in the region of the heart, and in five minutes she would have urgent dyspnœa, noisy respiration, with coarse rãles everywhere over the chest. A hypodermic of morphia with atropine would quiet her within an hour or two. She was under observation on and off for more than a year, and she had scores of the pulmonary attacks, sometimes with pain but very frequently without. In the severer attacks she would have strong tonic contractions of her fingers and hands. We often discussed the character of this paroxysm, as it was suggested that she might have had asthma previously. In
the suddenness of the onset and the rapidity with which the attacks came on, the limited duration and prolonged expiration suggested bronchial asthma. She did not always have a cough and rarely had expectoration.

One case in the series had indications of consolidation of the lower lobe of one lung in connexion with an attack, and curiously enough, while preparing this lecture, Dr. T. McCrae, knowing my interest in the subject, sent notes of the case of a man, aged 35 years, who died from angina. The attacks yielded promptly to amyl nitrite, but in the last one there were acute pulmonary symptoms for 24 hours, oppression, cough, &c., and scattered areas of consolidation with tubular breathing and râles.

A case with extraordinary respiratory features was that of Captain H., whom I frequently saw with Dr. McCormick. A man of extraordinary vigour and health, aged 71 years, and of great intelligence, he had, as is not uncommon, studied his case most closely. For years he had had to be very careful in doing all the small actions which necessitated stooping, as in tying his shoes or in getting into his bath-tub, as he would experience such intense pressure in the chest which would render him immobile for a moment or two. Then he had a second variety, which corresponded to ordinary severe attacks of angina, terrible paroxysms of great severity. And thirdly, he had respiratory attacks, in which, waking from a sound sleep, he would gasp as if every breath would be his last, and then he would choke and begin to cough; the respiration became wheezing and the piping râles came everywhere over the whole chest just as in a severe attack of asthma. In from 10 to 15 minutes he would bring up large quantities of frothy, sometimes blood-stained, expectoration, as much as a large tea-cupful. He died in an attack of angina.

D. Cerebral features of angina.—Unconsciousness may occur during an attack. Epilepsy, believed by Trousseau to be closely related to angina, occurred in two cases, in neither in direct connexion with the paroxysm. Certain of the reported cases may have been the Stokes-Adams disease, in which the epileptiform attacks are sometimes preceded by pain about the heart. In only three cases transient cerebral symptoms occurred during attacks. T. S., aged 51 years, had had for three years occasional paroxysms. On Sept. 20th, 1901, while in a seizure, he had transient numbness of the left hand and foot and side of the face. The left leg dragged. These symptoms disappeared in the course of the day, but he complained of his eyesight and was found to have lateral hemianopia. The condition resembled closely the transient cerebral attacks so common in arterio-sclerosis. Aphasia is a rare complication. I. B., aged 63 years, seen Jan. 14th, 1900, had had angina on and off for five years. In four attacks he had had transient aphasia without losing
consciousness. In a recent attack he was speechless for an hour, and once eight hours passed before he could say a word. Dr. McKnew said that there was no trace of paralysis and that he could understand everything and write. He had widespread sclerosis of the vessels. He died in an attack.

A remarkable feature existed in the case of Mr. M., who in the attacks had an intense pain at the back of the head, inside he insisted, and he would hold his head between the hands to avoid pain on movement.

L. S., aged 31 years, was admitted on Feb. 21st, 1898, with paroxysms of severe angina, which had recurred for several years, usually following exertion. Five attacks occurred in the month before admission, one with unconsciousness. He had not had syphilis, nor was there heart disease. He had had on several occasions temporary loss of power in the left hand with numbness. In the paroxysm just before admission, which was of great intensity, the left arm and leg were paralysed for two days. The face was not involved.

I have dwelt upon these extra-cardial features with a special object, as they throw light upon the essential nature of the disease, which I propose to discuss in the next lecture.
LECTURE II.

Delivered on March 15th.

PATHOLOGY.

Had Heberden listened to my first lecture he could have remarked very justly: "Well! they have not got much ahead since my day." In descriptive symptomatology we have not, and among 100 cases of angina pectoris there is no reason why Heberden should not have met all the important anomalies and complications. He had the good sense not to say much about the cause of the disease, and the good fortune to get very close to the truth in what he did say. I do not propose to weary you in a vain repetition of the scores of explanations which have been offered since his day. The older ones are to be found in the monographs of Parry and Jurine, the more recent in the Traité of Huchard, in the writings of our President, and in those of Allbutt, Bramwell, Gibson, Morrison, MacKenzie, and others. At the outset let us frankly face certain obscurities which have not yet been cleared up. Why is it more common in the upper classes? Why do we not see it more often in hospital practice? Worry and work are the lot and portion of the poor, among whom vascular degeneration is more widespread. It is as though only a special strain of tissue reacted anginally, so to speak, a type evolved amid special surroundings or which existed in certain families. Or there may be a perverted internal secretion which favours spasm of the arteries, as Harvey at Cambridge has shown to be the case with pituitary extract and the coronary vessels. And a case of aortic valve disease is reported in which the use of this extract caused anginal attacks. This suggestion is supported by the fact that in myxœdema anginal attacks may be caused by thyroid extract. I saw last year a patient of Dr. Lafleur's of Montreal with this most distressing peculiarity, which was mentioned to me also by Dr. Allan Starr of New York. The disease may occur in three generations, as in the Arnolds, and a father and four children have been affected. In three instances of my series father and son were attacked; in two, brothers; and in one, a brother and sister. It is not the delicate neurotic person who is prone to angina, but the
robust, the vigorous in mind and body, the keen and ambitious man, the indicator of whose engines is always at “full speed ahead.” There is, indeed, a frame and facies at once suggestive of angina—the well “set” man of from 45 to 55 years of age, with military bearing, iron-grey hair, and florid complexion. More than once as such a man entered my consulting-room the suggested diagnosis of angina has flashed through my mind. Still more extraordinary and inexplicable is an imitative feature, if one may so speak of it, by which the repeated witnessing of attacks may induce one in the observer. The case of Senator Sumner attracted widespread interest on account of his distinguished public position. Two weeks after his death Dr. Hitchcock, his physician, died in an attack with coronary artery disease and acute infarct of the myocardium. Tabor Johnson, his other physician, at that time a young man, had two attacks, diagnosed by Brown-Sequard as angina, and he had seen some twenty cases of what may be called the manufactured variety. Straus died not long after his friend Charcot. A young man, aged 28, whose father, a very vigorous planter, had through the spring and summer of 1900 severe attacks and died in one Sept. 28th, consulted me the following January for angina. I had seen the father, and had been a witness to the devotion of the son during the terrible paroxysms. Within a month of the death of the father he began to have severe pain in the chest, with pallor, sweating, the pains down the left arm, which became numb and tingled. The sister said the paroxysms were identical with those of the father, and naturally the family were greatly distressed. The patient was a healthy, robust fellow, very neurotic, and almost frightened to death. A reassuring prognosis was all the treatment he required. He has had no further attacks. A woman, aged 38, after her father’s death from angina, had severe pains about the heart, and attacks which she insisted were of the same character, but she, too, got quite well. A still more remarkable illustration of the imitative, emotional influence was seen in the outbreak of angina-like attacks among the sailors of the French corvette L’Embuscade reported by Gelineau.

There are two primary features of the disease, pain and sudden death—pain, paroxysmal, intense, peculiar, usually pectoral, and with the well-known lines of radiation—death in a higher percentage than any known disorder, and usually sudden. Often, indeed, it is, as the poet says, “Life struck sharp on death.” The problems for solution are: What is the cause of the pain? Why the sudden death? The secondary features of the attack, the vaso-motor phenomena, the radiation of the pain, the cardiac, respiratory, and gastric symptoms are of subsidiary interest.
Naturally, in the presence of a disease with such startling characters, men have sought an explanation in the bodies of its victims. And angina pectoris has a very definite morbid anatomy, few affections more so, since in practically all cases vascular disease exists. With Morgagni, Jenner, Fothergill, and Parry, a majority of authors have correlated the fatal symptoms with the arterial disease; others have reached the less satisfactory, if more philosophical, position of Rougon, who, taking all the circumstances into consideration, concluded, "Monsieur Charles est mort parce qu'il est mort." Not a hospital disease, one naturally does not see many necropsies. I have notes of 17 post-mortem examinations, all in men, 8 of them in men under 40 and 4 of them with a history of syphilis, and dying at the ages of 34, 38, 37, 39. They fall in three groups—aortitis, coronary artery disease, and a negative case.

A. Aortitis.—From the publication of Morgagni’s famous case writers have recognised the importance of aortic changes at its root. The special importance of this has been dwelt upon by my brother regius of Cambridge, whose many publications upon the subject, dating from his remarkable study of syphilitic arteritis in 1868, have edified his colleague and students. For our purposes here there is but one aortitis—the syphilitic. Occasionally a fairly acute process occurs at the root of the aorta in the specific fevers, but this is very uncommon, except in connexion with endocarditis. Chronic atheromatous changes in the aorta of the aged are very rarely associated with angina unless the coronary arteries are involved. Syphilitic aortitis is a most distinctive lesion. I pass round the beautiful plate of Corrigan’s paper, in which he brings out for the first time I think, and with great clearness, the connexion of the disease with this lesion. The frontispiece of Balfour’s book on “The Senile Heart” gives an equally good representation. Upon its anatomical features I need not dwell further than to refer to its predilection for the supra-sigmoidal region, the sectional limitation, and the great frequency of its association with aneurysm.

Of the post mortem examinations of my series only one offered a good illustration of the supra-sigmoidal type; a negro, aged 38, who had had syphilis about a year before. The attacks of angina began in December, 1904; they lasted for from 15 minutes to half an hour, with very characteristic distribution of the pain; in severer paroxysms he had fallen unconscious. The attacks recurred even when he was in bed and quiet. There was diffuse cardiac impulse, the area of

2 St. George’s Hospital Reports, 1868.
flatness was increased, but there were no murmurs; the blood pressure was 188 mm. Hg. On the evening of admission he had a very sharp attack, and another at 1.30 A.M., in which the pain was chiefly epigastric; he sweated profusely and became very weak, and at 2.30 was found unconscious, and died at 4 A.M. The heart weighed 490 grammes; the free edges of the valves were a little thickened; the only important lesion was an extensive fresh-looking aortitis, involving the root of the vessel and narrowing the orifice of the left coronary. The right coronary orifice was normal; the coronary arteries themselves were not affected.

Another syphilitic patient, W. A. M., aged 38, admitted Feb. 20th, 1895, had very severe paroxysms of angina, with aortic insufficiency. The aortic segments were thickened and curled; the coronary arteries were small but healthy; there was the characteristic sclerotic aortitis not confined to the root. The smaller arteries of the body, particularly the splanchnic, were tortuous and thickened.

In a third syphilitic case, J. W., negro, aged 34, admitted May 25th, 1897, the paroxysms were most characteristic, and had recurred since March; in several attacks he had become unconscious, and following them he had transient weakness of the left arm. During the fortnight he was in hospital he had several severe attacks; the left arm was distinctly weaker than the right, particularly the grasp of the hand; the heart appeared to be normal. On June 8th he complained of a great deal of coldness of the hands and feet; at 6.10 in the evening he threw up his hands suddenly and died within a few minutes. Widespread aortitis of the sclerotic type, with here and there plaques of atheroma, were the only lesions. The coronary arteries were not involved; they looked small, the walls thin, but there was no occlusion.

B. Coronary arteries.—We are all united in the acceptance of the Jennerian view of the close connexion of lesions of the coronary arteries with the disease. As shown in the extensive analysis by Huchard, a very large proportion of all the cases show changes in these vessels. Of the 17 necropsies of my list, 13 illustrated all the varieties of the lesions.

(a) Narrowing of the orifices is a very common occurrence, particularly in the syphilitic aortitis, but not often met with without some involvement of the branches. In the case of a man who died suddenly in my wards after recurring attacks the sclerosis of the ascending part of the arch was marked and the orifices of the coronary arteries were extensively contracted; as the post-mortem report states, “they admitted only a bristle.” The arteries beyond were nearly normal, showing only slight sclerotic change.

(b) Blocking of a branch with a fresh thrombus is very
common in cases of sudden death in angina. In my post-mortem experience this has been more frequent in the medico-legal cases of sudden death without symptoms of angina. One of the main stems or a small branch may be plugged with the formation of fresh infarct. In patients who live some time the infarct may soften and pericarditis may be excited. A specimen in McGill College, from a man who died suddenly the day after an attack, shows the left coronary artery blocked by the thrombus and perforation of the softened anterior wall of the ventricle.

(c) Obliterative endarteritis, if we may judge from the reports of fatal cases collected by Huchard and others, is the lesion of the disease; it was present in nine cases of my series. The most remarkable peculiarity is the variation in the extent of involvement. The angina may be associated with obliteration of a comparatively small branch, or with a most widespread involvement of all the vessels. In the younger subjects the process is a gradual endarteritis with narrowing, and even complete occlusion of the vessel. In older subjects, the arteries may be converted, as in John Hunter and in William Pepper, into "open bony tubes." In one instance of my series the vessels were calcified to their smallest branches. Four cases showed disease of the coronary arteries alone; five in connexion with aortitis. In looking over these notes one is astonished at the comparatively small extent of coronary tubing which is sufficient to carry on the myocardial circulation. Mr. G., aged 39 years, an extraordinarily vigorous muscular man, after a day full of effort and strain, had read an important paper at a college society and died the following night in an attack. Not more than a third of his coronary vessels were in use. It has long been known that advanced coronary artery disease may be present without much disturbance of the function of the heart. There is not a clinician among us who could not furnish from his notes a dozen cases of this kind. A man may get on very comfortably with only the main branch of one coronary, practically a fourth of the whole system. A heart once in my possession showed almost complete obliteration of the left coronary, only a pin-point channel could be traced for a short distance. Of the right branch, the main division passing between the auricle and the ventricle was completely obliterated, so that the only one of full size passed in the posterior interventricular groove. The heart came from a large, very muscular imbecile, aged 36, an inmate of the Institution for the Feeble-Minded, at Elwin. I knew him well; a good-natured, helpful fellow, constantly employed in carrying about, and attending to, the more helpless children. He died suddenly one day in a fit. The coronaries are not endarteries in the sense of Cohnheim, and disease of their branches is not necessarily associated with angina.

(d) And in a few fatal cases no lesions whatever are found;
we must accept the fact that angina pectoris may kill without signs of obvious disease in heart or blood-vessels. Such an instance has been reported by Dr. Bullard and myself. The case was regarded by all who saw it as one of so-called functional angina. The patient, aged 26, was very strong and robust, devoted to athletics, and a heavy smoker. He had served in the United States Army, but was discharged in the spring of 1896 for attacks of angina. The chief feature was pain in the heart, and "awful cramps," as he described them, in his arms. The attacks were so severe that at times he became unconscious, and after one he was thought to be dead, and was about to be removed to the dead-house! The attacks were brought on by cold and exertion. The pain was evidently very severe, and in the major paroxysms respiration would cease, and his pulse would become so feeble that he seemed to be dead. Only chloroform and morphia were of any avail in the attacks. He had an extraordinary number of attacks in 1896-7; Dr. Bullard had notes of 105. In 1898 he was better and had not nearly so many attacks, and was able to be at work. On Nov. 27th at 11.30 he had an attack of great severity; at 12.55 the doctor gave him chloroform; the attack was very prolonged, and the muscles of the chest became fixed, and remained so; he had a series of paroxysms and died at 6.40 in the morning. Except a few pleural adhesions, there was nothing special to be noted. The heart weighed 14 ounces; the muscle and the valves were normal. Just above the ring the aorta measured not quite 6 centimetres, a small vessel for a man of 5 feet 10 inches, weighing just over 13 stones. There was no disease except a flake here and there of atheroma. There was no thickening about the pericardium, and the sections showed no changes in the cardiac nerves.

**Pump and Pipes.**

The circulation as a whole may be compared to a vast irrigation system, with innumerable sub-districts of varied extent, under the control of local officers, but all under one central bureau, with which they are connected by telephone and an automatic signalling apparatus. The engine, pumping night and day, keeps a steady, uniform supply in the mains. The efficiency of the system depends upon the care with which the managers of the sub-stations regulate the flow to different plantations as occasion demands; the slightest disturbance in the most distant district is at once indicated by telephone to the central office, or in some instances automatically to the pump itself. Into certain vast irrigation areas with large sluice-ways all the water of the system can be diverted; and through carelessness of the men in control.

or through misinterpretation of a message from the head office, it sometimes happens that these sluice-ways are left wide open and the whole system is wrecked. Or strikes arise in local, outlying districts, the distributing mains are closed, and the pumping reservoir is flooded and permanently disabled. Or things go wrong in the central bureau—supplies are not forthcoming to keep up the plant, or there is litigation with neighbours, and the works are shut down, sometimes abruptly and without warning. What happens in a great irrigation plant happens also in the vascular system of the animal body, the mechanism of which, pump (heart), mains (aorta), sluices (arteries), and lakes (capillaries), is very much the same. Take two illustrations of its working. In Hill's experiment—hold a tame rabbit up with the forelimbs spread, and the gates of its splanchnic sluices will open so wide that the head office, pumping station, and whole irrigation system are wrecked in a few minutes. Try the same with the wild rabbit, whose splanchnic dam is under the control of trained officials—nothing happens. The pumping-engine itself is as sensitive as a galvanometer and has a marvellous mechanism for relieving and preventing any strain or tension on its machinery. Irritate with a probe, as in Stewart's experiment, the inner surface of the left ventricle, just enough to suggest or imitate tension, and automatically messages are sent, opening wide the most distant sluice-gates to prevent any strain on the pump itself. Or damage the main valve of the pump so that there is a leak with increased central strain, as in aortic insufficiency, and all the outlying territories open their sluice-ways to relieve the pressure. The circulation is maintained, equalised, and regulated by one working element—the muscle in the walls of its system, a peculiar, indeed a unique, type in the pump, ordinary unstriped fibre in the distributing channels. Both constituents, heart and arteries, are elaborately "wired" with nerves, which end about possibly in the muscle fibres, and there are peculiar end organs widely distributed (Paccinian bodies). Just as in the irrigation fields, pump and channels are connected by wires with the local and central offices of control, so the arteries and the heart are connected with centres, local and general, which act directly upon their muscular elements, by which the whole system is worked and regulated—an automatic set of fibres which keeps the head office constantly informed as to pressure conditions in the engine, a set which slows, and a set which hastens its action. Moreover, a complicated, subsidiary system coördinates the different parts of the heart ministered to by a tissue of special type, unlike the fibres of the heart itself or of the arteries. Not only does the muscular element maintain the circulation, but it keeps the vascular walls in a state of tension, a tonus or tautness which has an all-important influence in relieving the strain on the non-muscular elements. As Harry Campbell remarks
in his recent Study of the Circulation,¹ "The greater the
tonus of the muscular elements the more exclusively does the
vascular strain caused by the blood pressure fall on them."

**IN VOLUNTARY MUSCLE PAIN.**

Involuntary muscle pain has its peculiarities, and whether
in artery, bowel, ureter, gall-duct, or uterus, comes in
crises, storms, and outbursts. I have recently taken advan-
tage of an unpleasant experience in my own person to
observe the phenomena of these paroxysms in a ureter
struggling with a calculus. Periods of complete freedom,
extending from two to three, to eight or ten hours, attenuated
with three types of disturbance of sensation—a dull, steady,
localised pain, the situation of which could be covered with
a penny. It could be imitated exactly by firm pressure with
the handle of a knife, or, indeed, with a finger upon a bone,
particularly upon that tender spot on the sternum just a
little above the ensiform cartilage. Lasting for hours and
unmoved it was fairly bearable. Now and then, when free
from pain, there were remarkable flashes, an explosive sort
of sensation, not actually unpleasant, and accompanied by a
glow-like wave along the course of the ureter and out through
the flank, as it were through the muscles. And then
abruptly, or working out of the steady pain, came the
paroxysm, like a twisting, tearing hurricane, with its well-
known radiation, followed by the vaso-vagal features, the
pallor, cold extremities, feeble pulse, sweating, nausea,
vomiting, and in two attacks, a final, not altogether un-
pleasant period, when unconsciousness and the pain seemed
wrestling for a victory reached only with the help of God's
own medicine—morphia.

Any portion of the arterial system taken as a unit may
present the phenomenon of involuntary muscle pain, and
herein, I think, lies the key to the explanation of the anginal
attack. The intermittency, the suddenness of onset, the
steady, dull, enduring pain, and then the paroxysm, with its
associated vaso-motor features, sometimes unconsciousness,
and the radiations are paralleled in other involuntary muscle
crises. Paralleled, but not equalled, and not often associ-
ated with the dangerous collapse symptoms, and rarely
causing sudden death. And yet a man may die in renal or
biliary colic, borne down in a vaso-motor storm, as happened
in the only case of the kind I have seen.

**CARDIO-VASCULAR PAIN.**

What do we know about cardio-vascular involuntary muscle
pain, and under what circumstances do we meet it? Like

other viscera, the heart itself is insensitive to ordinary stimuli. You remember how this so amazed Harvey when handling the apex of the heart of the young Viscount Montgomery. Even his Most excellent Majesty, who studied the case with him, "acknowledged that the heart was without the sense of touch; for the youth never knew when we touched his heart except by a sight or the sensation he had through the external integument."

In most affections of the heart pain is conspicuous by its absence, particularly in the more serious maladies, so that it has almost become an axiom that "not much is the matter when a patient complains of his heart." Pericarditis may pass through all its phases without pain. Occasionally it is present in a marked degree, and it may be a special feature in the chronic mediastino-pericarditis.

In acute endocarditis pain is rarely present, and ulceration of valves or of the wall may proceed to a most extreme degree without any sensory disturbances. Of valvular lesions mitral disease is often associated with slight pain, particularly in children with greatly enlarged heart. And sometimes in women the pain is of great severity and persistence, but it rarely has the characters of true angina. There are a number of cases on my list with mitral lesion, stenotic or regurgitant, but, curiously enough, the only instance of attacks which I could call genuine angina pectoris in the stage of cardiac insufficiency occurred in a young girl of 11. And in this point I see that my experience coincides exactly with that of Nothnagel. On the other hand, lesions of the aortic ring are often painful, and attacks of true angina are common, particularly when the root of the aorta is involved.

Arterial pain is met with under many different circumstances, and may present all the features of angina. In the first place, external pressure directly upon the wall is associated with agonising pain. Those of us who as students took our turn in digital compression of the femoral artery for popliteal aneurysm have a lively recollection of the misery suffered by the poor patient.

Secondly, pressure from within; the pain caused by an embolus may be of the most terrific character. A man admitted to the Radcliffe Infirmary under Dr. Brooks—an old examination case of aortic insufficiency, with a loud, musical, diastolic murmur—had a sudden pain in his right leg, just below the popliteal space, and for days was in such agony that he had to have repeated hypoderms of morphia. As the swelling and pain subsided signs of an aneurysm became evident, and it was noted that the loud, musical murmur had disappeared. A calcified fragment whipped off from the aortic valve had torn the wall of the artery. Not only sharp emboli but the soft ones of ulcerative endocarditis cause intense pain. As I was going up the steps of the house of a patient, the diagnosis of whose trouble had
wavered between typhoid fever and ulcerative endocarditis, I heard loud screams and found a young fellow in great agony, and he pointed to a spot below Poupart's ligament which he would not allow us to touch. He had embolism of the femoral artery, with subsequent gangrene of the leg. The intense colic of mesenteric embolism, such as we see in aneurysm, and occasionally in endocarditis, is of the same character, and it is diagnostic point between thrombosis and embolism of the cerebral arteries.

Thirdly, spontaneous tear of the arterial coats is associated with atrocious pain, with symptoms, indeed, in the case of the aorta of angina pectoris, and many instances have been mistaken for it. In this remarkable drawing which I pass round, of a split, fissured, and healed rupture of the internal coats of the aorta just above the valve, the patient was thought to have angina pectoris, and in the second attack, from which he died a year or more subsequently, a fresh split of the internal coats was found, which had ruptured into the pericardium.

Fourthly, as a result of extreme dilatation, distension, and stretching. Following the application of an Esmarch bandage, the arteries of the limbs dilatate and throb, and there may be pain of a very intense character. In chilblains and in erythromelalgia the pain is probably arterial, and may be greatly aggravated with each systolic distension. In the excessive dilatation of the vessels following frost-bite the more rapid the dilatation the more intense the pain. As boys we had to give practical recognition to this point; if after a snowball fight anyone was foolish enough to put his cold hands into warm water he would be sure to suffer agonies of pain. An every-day cause of arterial pain is met with in aneurysm. In 132 cases of thoracic aneurysm the histories of which were carefully revised for this symptom pain was present in 104, and in 62 the trouble began with it. A feature of special interest to which attention has been called by many writers is the occurrence of attacks of angina pectoris as the first symptom. This happened in four cases in my series, and in every one of them the anginal attacks disappeared with the increase in the size of the aneurysm. There are other mechanical causes of pain in aneurysm, but I think we all accept the fact that pain is a very constant feature in the early formation and growth of the sac. Stretching of the aorta without disease of its coats, as seen in the dynamic dilatation of aortic insufficiency and certain neurotic states, is not necessarily painful.

Fifthly, spasm of the arteries may cause severe pain. Slow, gradual contraction of the peripheral vessels due to cold is associated with a sense of numbness but not of actual pain. Scores of everyday vascular actions illustrate the same thing, and the radials may be contracted to obliteration of the pulse without any abnormal local sensations. On
the other hand, there are types of arterial spasm accompanied with acute pain. Dubois, you may remember, referred the pain of hemicrania to angio-spasm. And our distinguished emeritus registrar, in his classical monograph on the disease, notes a number of instances and discusses this theory very fully. One does occasionally meet with an extraordinary degree of contraction of the temporal arteries during a paroxysm, but I have never seen an arterial distribution of the pain, nor are the vessels themselves sensitive.

The painful extremities in the various pathological states described as Raynaud's disease afford the best illustrations of disturbance of sensation as a direct result of angio-spasm. In a great many of the cases there is either obliterator endarteritis or the thrombo-angitis of Buerger. But numerous observations show that spasm alone may account for all the symptoms. The paroxysmal character of the attacks, the intensity of the pain, the direct association with angio-spasm, suggest its vascular origin. It is an interesting point, too, that angina pectoris has been met as a complication of Raynaud's disease. In the case reported by Cleeman, a man, aged 62 years, had from his fiftieth year severe attacks of Raynaud's disease, chiefly in the hands, and usually in the winter season. Following several pronounced attacks of local asphyxia, and local syncope in the hands, he had one day a very severe paroxysm of angina pectoris; the pain lasted for two hours and was of such intensity that he was greatly prostrated. The association of migraine with angina pectoris, particularly the vaso-motor type, has long been recognised, and is discussed by Dr. E. Lively; two of my patients had been great sufferers with typical migraine.

**Arterial Spasm.**

Let us now consider in what conditions we actually see spasm of the arteries; and by spasm I mean a persistent contraction leading to ischaemia, with disturbance of function of the parts supplied. Raynaud's disease is, of course, the type of an angio-spastic affection. One does not actually see the arteries contract, but one may feel the gradual reduction in volume of the pulse, even to obliteration. One may feel a full, easily palpable radial contracted to a narrow cord, followed by a gradual blanching of the skin of the hands. The spasm may affect the smallest twigs, such as those distributed to the extreme tips of the fingers, or it may be the tip of one finger only. The spasm is not always painful, but it may be associated with intense pain, and I have noted in one or two instances that there is greater pain with the local syncope, and the reactionary intense hyperæmia, than with the cyanosis. Of late in so many
instances of so-called Raynaud's disease arteritis has been
discovered that is well to insist upon the fact that
the most advanced necrosis may occur as a consequence
of spasm in vessels apparently healthy. Russian Jews
are subject to a very remarkable malady studied by
Buerger of the Mount Sinai Hospital, New York. While
similar in some features to Raynaud's disease, it differs
anatomically in having widespread obliterative endar-
teritis, with thrombosis of the veins; indeed, the disease
may begin in the veins, so that Buerger calls it "thrombo-
angiitis." The same condition has been shown by Parkes
Weber in several Russian Jews at the Clinical Section of
the Royal Society of Medicine. Buerger writes me that in
two cases of typical Raynaud's disease in which he performed
amputation of the leg the arteries were found normal;
evidence of exceptional value, as this observer has made a
special study of the condition of the vessels in some 30 or 40
cases of thrombo-angiitis of the leg.

There is one place in which we can actually see spasm
of the arteries associated with loss of function. In numbers
of instances of amaurosis spasm of the papillary arteries has
been noted by Priestley Smith and others. I have seen but
one case myself—a man with small contracted kidneys and
the usual associated vascular changes, became blind while
walking from the outpatient department to the ward, and
was unable to see for some hours. The retinal arteries on
both sides were strongly contracted and I had the advantage
of the confirmation of the observation by my colleague,
Buller, the well-known ophthalmic surgeon. In Raynaud's
disease a similar contraction of these vessels has been seen,
originally by Raynaud himself, since then by a number of
observers, and quite recently in an interesting case reported
by Friedman. In none of these cases has pain been men-
tioned as a symptom. I know of no other conditions in which
we actually see angio-spasm with disturbance of the function.

And now let us leave the solid ground of observation for a
few minutes. As I mentioned in my first lecture, the term
"vascular crises" was introduced by James Collier in dis-
cussing the features of erythromelalgia, and it is a most
useful term which admirably expresses the state of affairs in
the recurring paroxysms of Raynaud's disease. It has been
used with great effect by recent authors, particularly by Pal
of Vienna, in whose monograph, "Gefisskrisen" (Leipsic,
1905), the whole question is exhaustively considered, more
particularly in reference to its association with high
tension and arteriosclerosis. The profession is at present
riding on the top of a cardio-vascular wave, and it is
impossible to approach questions without considering blood
pressure and sclerosis. In Pal's hand the vascular crisis is a
key to unlock many of the mysteries of disease in head,

5 Friedman: American Journal of Medical Sciences, February, 1910.
chest, and abdomen. Paroxysmal high tension we know with its remarkable phenomena—cardiac dyspnoea, cardiac pain, headache, uraemic symptoms, nausea, vomiting, and convulsions. No one who has seen much of blood pressure work can doubt that in patients with arterio-sclerosis these paroxysms play a very important part; but when we come to conditions of local high tension associated with contraction of the arteries, I confess that we are a little bit in the spray of the wave, and yet it may be used as a working hypothesis to explain a whole group of obscure conditions. As briefly stated, Pal's contention is: "Where the tension is produced by contraction in a definite vascular area, local consequences follow and dominate the picture. These are manifest chiefly by a peculiar painful sensation and local disturbance of function. General phenomena to a greater or less extent are manifest at the height of the tension." One is a bit staggered at the very free use which many writers make of the vascular crises, but it is a seductive theory and only the name is new. We have, I think, evidence that sclerotic arteries are specially prone to spasm. In many of Buerger's cases of thromboangitis the symptoms were in part due to spastic contraction; in intermittent claudication vascular spasm plays a part, and one may actually see the foot get pale, as the patient begins to complain of pain and stops walking. We have really very little positive evidence of angio-spasm of the internal vessels. In a few remarkable cases of Raynaud's disease transient cerebral symptoms have occurred—aphasia, monoplegia, epilepsy, either at the time of or alternating with peripheral attacks. So transient has been the disturbance of function that it could be scarcely any other condition than angio-spasm. This was the view I took of the two remarkable cases which I reported some years ago. Now we have in arterio-sclerosis identical transient cerebral attacks for which it is scarcely possible to offer any other explanation. The condition, familiar to me for more than 25 years, was brought to my notice by the illness of a warm personal friend, who before his forty-fifth year was the subject of the most advanced sclerosis, with high tension. He had literally scores of attacks of transient paralysis, of monoplegia, aphasia, occasionally hemiplegia for 24 hours; and once as he got off the steamer after a trip to England he became paraplegic and remained so for nearly two days. The attacks are not always associated with very high tension. The cases are by no means uncommon, and a peculiarity is the extraordinary frequency of the attacks and their transient character. The question has recently been reopened in an interesting discussion on intermittent closure of the cerebral arteries by Lauder Brunton, William Russell, and Hobhouse in the British Medical Journal towards the end of last year. The correspondence shows how numerous are the cases. It will be fresh in your memory how ably Dr. A. E. Russell supported the vaso-motor or arterial-spasm
view of the origin of epilepsy in the Goulstonian lectures of last year.6

What evidence is there of the existence of angio-spasm in angina pectoris? In many cases the attack begins directly as a peripheral vaso-constrictor storm, with cold hands and cold feet, pallor of the face, and sweating. Nor is this simply in the so-called functional type, but in the severest forms an emotional disturbance may initiate a widespread contraction of the arteries. During the paroxysm it is by no means uncommon to find the radial pulse on one side very much smaller than on the other. I have notes of six cases in which this observation was made, and there may be associated pain, numbness, and tingling. In a few instances spasm of local arteries has occurred with the features of Raynaud’s disease. I have already mentioned Cleeman’s case in which the diseases coexisted. And Worton7 has reported an instance in a woman, aged 54 years, in whom after attacks of angina the tip of the left middle finger always became dead white and numb. That is as far as the facts carry us, and they indicate a widespread tendency in the disease to angio-spasm. I discussed in the last lecture with a purpose the extra-pectoral phenomena of the disease, as these receive their most suitable explanation in the spasm of the arteries. In one case the attacks of pain in the leg occurred with intermittent claudication. The sensations in the leg in Dr. W. W. J.’s case, which initiated the attack, were identical with those I have seen in the arm, with definite contraction of its blood-vessels. The three cases with transient aphasia or paralysis during the paroxysms are suggestive of intermittent closure of the vessels, particularly when one considers the similar attacks in Raynaud’s disease and the frequency of such a transient paralysis in arterio-sclerosis. It is quite possible that the pain in the testis, such as I have reported, and of which there are a number of cases in the literature, may be associated with arterial spasm, and a suggestive fact is that the organ may become swollen after the attack.

A consideration of the very important group of cases in which the pain is limited to the abdomen throws light on the question. Except in distribution the symptoms may be identical. Pectoral and abdominal attacks may alternate, or one may spread into the other. Death may occur with only the abdominal pain. Special consideration of this angina abdominis may be found in the “Archiv für Ver-
dauungs-Krankheiten,” Band IX. and X., by Buch. These attacks have a striking resemblance to three well-known forms of abdominal crises—the lead, the tabetic, and that met with in purpura and angio-neurotic cedema.

6 The Lancet, April 3rd (p. 963), 10th (p. 1031), and 17th (p. 1093), 1909.
7 The Lancet, April 16th, 1898, p. 1053.
The whole question of visceral pain has of late years undergone revision. Haller, Mackenzie, Lennander, and others conclude that practically all organs innervated by the sympathetic and vagus are insensitive to ordinary stimuli. After having gone through a painful visceral experience, one appreciates the force of Dr. Johnson's method of refuting Bishop Berkeley's theory of the non-existence of matter. "I refute it thus," he said, kicking a stone; but of course they refer to ordinary extrinsic stimuli. It was Riegel, I believe, who first suggested that the pains of lead colic were due to spasm of the branches of the intestinal arteries, with ischaemia and oedema of the wall. Pal explains the abdominal pains in tabes as a vascular high-tension crisis, with spasm of the smaller intestinal arteries, and stretching of the nerve plexuses of the proximal arterial wall. Very high peripheral blood pressure exists during the paroxysms, as in a case recently reported by L. F. Barker, in which, in a woman, the pressure rose to 210 mm. Hg. It is a reasonable inference that the crises of these two states are really vascular. We have positive evidence of it in the extraordinary abdominal crises of purpura and angio-neurotic oedema. There are now nearly a score of cases which have been operated upon for "the acute abdomen." Oedema of the wall of the stomach or bowel, or oedema with haemorrhage, has been the usual condition found; though, as in the remarkable case reported in last week's LANCET by Collinson, intussusception may be present, and it would be very apt to follow a localised oedema. I do not know that these cases specially favour a view of angio-spasm, but they, at any rate, point to a vascular origin. We may accept the view that the bowel wall is insensitive to ordinary stimuli, but the visible peristaltic colic of any chronic obstruction, with its character of smooth muscle pain, demonstrates in blunt Johnsonian fashion the existence of nerves capable of transmitting painful sensations from intrinsic stimuli, whether they exist in the arteries alone, as some believe, or are distributed among the tissue elements of the wall itself.

THE CONCLUSION OF THE MATTER.

After all this talk, what in a few words is a reasonable explanation of the pain in angina? Angina results from an alteration in the working of the muscle fibres in any part of the cardio-vascular system, whereby painful afferent stimuli are excited. Cold, emotion, toxic agents interfering with the orderly action of the peripheral mechanism, increase the tension in the pump walls or in the larger central mains, causing strain, and a type of abnormal con-

traction enough to excite in the involuntary muscles painful afferent stimuli. Mackenzie suggests that there is rapid exhaustion of the function of contractability, which is after all only the fatigue on which Allan Burns laid stress; but I feel that in disturbance of this Gaskellian function is to be sought the origin of the pain, whether in heart or arteries. This is practically the explanation given by the late T. K. Chambers and by Lauder Brunton and has received, Sir, your sanction. In stretching, in disturbance of the wall tension at any point, and in a pain-producing resistance to this by the muscle elements, lie the essence of the phenomena. In a man with arterio-sclerosis and high pressure, and all the more likely if he has a local lesion, a syphilitic aortitis for example, disturbance, at any point, of the tension of the wall permits the stretching of its tissues. Spasm or narrowing of a coronary artery, or even of one branch, may so modify the action of a section of the heart that it works with disturbed tension, and there are stretching and strain sufficient to arouse painful sensations. Or the heart may be in the same state as the leg muscles of a man with intermittent claudication, working smoothly when quiet, but instantly an effort is made, or a wave of emotion touches the peripheral vessels, anything which heightens the pressure and disturbs the normal contraction, brings on a crisis of pain. I do not know of any better explanation of anginal pain, and it is nice to think that in its main features it came from one of the earliest and ablest of British student of diseases of the heart—Allan Burns.

What is the explanation of the sudden death? There are three modes of dying in angina pectoris. The one which specially interests us here is the form which, as Walshe says, "is sudden, instantaneous, coeval, with a single pang." It is the quickest death we see, and is that which may have been in John Henry Newman's mind when he penned the lines describing the death of his mother—

"One moment here, the next she trod
The viewless mansons of her God."

No form of death so placid, so peaceful, and so much to be envied, as it probably is without a pang. The functions of life appear to stop abruptly, with a gasp or two all is over. It is extraordinary how little a man may be disturbed in this death. An old doctor whom I knew well stopped at his house to write a prescription. With pen in hand he died at the desk, where I found him, as if in sleep, with his head peacefully on his arm and pen in hand. Another friend the subject of angina, whom I had only left a few minutes previously, talking quietly to Dr. Thayer, fell over on his bed, both pulse and breathing seemed to stop simultaneously. It must be a vagal death, a sudden inhibition of the inspiratory centre in the medulla. No other explanation seems possible for such a condition as that.
which I described in the last lecture, in which the respiration stopped abruptly and in which a feeble heart's action continued for 45 minutes. It is exactly paralleled in chloroform death, when the inspiration stops abruptly, while the heart may continue to beat. In a third case in which I saw the death the suddenness with which the change took place was extraordinary. He was a man, aged 48, who had had very severe attacks. During my visit he was very comfortable, and he talked pleasantly and hopefully about getting back to his work. I was about to leave the room when he gave a sudden cry, clasped his hands over his heart, the eyes became fixed, and he fell over dead after giving two inspiratory gasps. No pulse could be felt at the wrists, but feeble heart sounds could be heard for three minutes.

A second mode of death is also seen in which, following a series of severe attacks, the heart grows gradually feeble, and the patient dies in progressive asthenia, often with Cheyne-Stokes respiration.

And thirdly, a certain number of patients die in the cardiac complications, and it is interesting to note how after great misery, caused by repeated attacks, when cardiac insufficiency is established, even with the dyspnœa, the patient is much happier, and dies slowly, if not so suddenly and placidly.
LECTURE III.

Delivered on March 17th.

PROGNOSIS.

How well the introduction to the "Prognostics" fits angina pectoris! A very excellent thing is it indeed, Hippocrates says, for the physician to cultivate prognosis, and nothing so much inspires confidence as the power of foreseeing and foretelling in the presence of the sick the present, the past, and the future, and he will indeed manage the cure best who has foreseen what is to happen. And almost as if he had this very disease in view he adds: "For it is impossible to make all the sick well; this indeed would have been better than to be able to foretell what is going to happen; but since men die, some even before calling the physician, from the violence of the disease, and some die immediately after calling him, having lived perhaps only one day, or a little longer, and before the physician could bring his art to counteract the disease, it therefore becomes necessary to know the nature of such affections."

The essence of prognosis lies in recognising, as Hippocrates says, "the nature of affection." And yet the thought must arise how futile to discuss the future in a disease, aptly described in so large a number of cases in Seneca's words as "meditatio mortis." I am sorry my figures do not allow me to agree with my brother regius of Cambridge, that of "all perilous maladies it [angina pectoris] is perhaps the most curable."

A consultant has great difficulty in making his experience of any disease effective, particularly in this matter of prognosis. A patient seen once or twice arouses intense interest, and then vanishes from his clinical ken, and the oft-repeated impressions of other cases leave a blurred image like that of a composite photograph. My practice was to get the notes of each angina case in good order at once, typewritten, and filed away, and then at intervals my secretary could write to the medical man in charge and add a note on the patient's condition.

It takes courage to make a prognosis. Fulness of knowledge does not always bring confidence; the more one
knows the more timidity may grow. The faculty which enables a man to look all round a question, to take a philosophical view of it, may be tempered with doubt, and an inability to reach a conclusion. A cocksure diagnosis and a positive prognosis may express the assurance of ignorance. In reviewing a long series, the high mortality and the great frequency of sudden death give a sombre tint to the picture, and yet I shall hope to show you plenty of bright patches. Seeing the more severe cases, the experience of the consultant is apt to be misleading, nor is always the lesson of his mistakes so thoroughly learned as by the general practitioner who lives in the same town with them. To know of the future is naturally the ardent desire of the patient and his friends, and whether we like it or not an opinion must be expressed. Sometimes out of pure kindness the tongue belies the head and always the great aphorism rings its warning—Experience is fallacious and judgment difficult. Now that we know more of the diagnosis of disease we talk less about prognosis, and to be of any value the latter must follow the former and grow out of it naturally. Often the one thing needful, the diagnosis, may embrace both prognosis and treatment; more than once this has happened in connexion with the disease under consideration. The first step is to get a clear idea of the nature of the affection—Is it an expression of organic disease of heart or arteries or is it only a painful disturbance of vascular function?

**PROGNOSIS IN RELATION TO ETIOLOGY.**

From the standpoint of prognosis the disease may be studied in various ways. Naturally the most important consideration relates to etiology. In any case three questions are suggested: Is it syphilitic mesoaortitis? Are the arteries involved in a general or local sclerosis? Is it a so-called functional condition unassociated with organic lesion of the arteries?

Syphilis is of the first importance, and we have learned to recognise the frequency with which this disease attacks the root of the aorta, causing angina pectoris, aortic insufficiency, or aneurysm. In 17 cases of my series there was a history of syphilis, and it is interesting to note that a majority of the patients were under 45 years of age. In many of the cases it was not possible to connect the attacks directly with the disease. I have already spoken of the lesion—a mesoaortitis—sometimes limited to the supra-sigmoidal area. A characteristic paroxysm may be the first symptom; a patient may, indeed, die in the initial attack. Nothing may be detected on physical examination; there may, perhaps, be slight superficial tenderness on pressure over the region of the aorta. Insufficiency of the valves may come on under observation. A striking result of the extended use of Wassermann's
reaction has been to show the importance of syphilis in aortic valve disease. The disease is not always confined to the root of the aorta; in three of my patients it was of the diffuse sclerotic type without special limitation. The prognosis is often very favourable.

The first case of angina pectoris I saw in Baltimore was a lieutenant in the navy, aged 30 years, who had had syphilis six years before. For a year he had attacks and had had to be off duty for many months. There was no enlargement of the heart, but there were systolic murmurs at both mitral and aortic areas. With the iodide of potassium he improved sufficiently to return to duty. After having lost sight of him for many years I then heard of him again, that he had remained perfectly well. A man, aged 58 years, seen on Oct. 28th, 1904, had severe attacks with moderate arteriosclerosis. I would not have recognised the specific nature of his trouble had I not seen him in 1898 with a most interesting syphilitic nephritis and periosteal nodes. The blood pressure was high, the aortic second sound ringing; but it was a favourable circumstance that the albuminuria had disappeared. He stood an anti-syphilitic treatment very well, progressively improved, the attacks gradually lessened, and he has remained well.

It is not always easy to determine whether or not syphilis is a factor, and yet it is well to give the patient the benefit of the doubt. A man, aged 56 years, who had had for three months very characteristic anginal attacks, had lived just the sort of life likely to bring them on—a gross eater, hard worker, heavy smoker, and yet the arteries were not specially sclerotic; the heart was not enlarged and the sounds were clear. He had had syphilis 30 years before. He improved with the use of iodide of potassium, and had no attacks for more than four years. I saw him very frequently, and it was not always possible to keep him in the "straight and narrow way." He died suddenly one night, probably in an attack, before a medical man could reach him.

In persons under 40 years of age it is always well to bear in mind the possibility of syphilis. A woman, aged 37 years, whose case had been a great anxiety, had paroxysms of angina with hypertension and acute pulmonary symptoms. There was no doubt as to the severity of the disease. The question of syphilis had not been discussed until she complained of an ulceration of the throat, which Dr. Warfield at once diagnosed as syphilitic. The transformation in a month was nothing less than marvellous. I had not thought of a possibility of her recovery.

The outlook is not always so satisfactory. I followed for a couple of years, with Dr. Julius Friedenwald, the various phases of aortic root syphilis in a man, aged 36 years, who had paroxysms of great severity, often associated with collapse, and once on the street with unconsciousness. Aortic insufficiency arose under observation. When I saw
him on Feb. 2nd, 1904, he was having attacks every day, and could not walk more than 100 yards without pain and oppression in the chest, "as though there was a stone under the breast-bone growing larger and larger." Marked improvement followed anti-syphilitic treatment, and he was able to go back to his business for a year or more, and was comparatively free. Then, in spite of vigorous treatment, the attacks returned, and finally in one of them he died.

The sclerotic aortitis may be the only lesion. John W., aged 24 years, a farmer, died in an angina attack, of which he had three or four during the fortnight. He was admitted to the hospital on May 25th, and died on June 8th, so that his illness did not last more than three and a half weeks. He had had syphilis nine years before. He was a healthy, muscular fellow, with a good pulse, sclerotic arteries, and an apex-beat a little out from the normal position. The attacks were severe, and in one he died suddenly. Beyond dilatation of the chambers and some chronic passive congestion there was nothing special; the valves were normal, the orifices not narrowed; characteristic sclerotic aortitis involved the entire vessel. Many of the smaller arteries were thickened.

**INDIVIDUAL HISTORY, CHARACTER OF ATTACKS, AND VASCULAR CONDITION.**

"The cardinal fact in the prognosis of real angina is its uncertainty," with which statement of Walshe we all agree, and further he confessed that he knew no method by which we can reasonably tell whether a sufferer will be cut off the next minute or survive many years. The individual history, the character of the attacks, and the vascular condition are the important elements in the prognosis—taking it for what it is worth.

Much depends on the patient himself—on the life he has led—the life he is willing to lead. The ordinary high-pressure business or professional man may find relief, or even cure, in the simple process of slowing the engines, reducing the speed from the 25 knots an hour of a Lusitania to the 10 knots of a "black Bilbao tramp." The difficulty is to induce a man of this type to lessen "the race, an' rack, an' strain." As William Pepper used to say: "Give me the life of a hare rather than the existence of a tortoise." Not even the terrible outbursts of pain may suffice to check men of this stamp, and yet, like Kipling's ship, The Haliotis, many a sensible fellow, whose engines at 50 or 55 years of age had gone to pieces on the "long trail, the out trail," has been refitted and enabled to reach port in safety. We doctors are notorious sinners in this respect, but it is so hard to lessen work when in full swing, so much harder than to give up altogether, and how few of us at 50 or 55 are able to do this!
A severe attack of angina may save a man's life. A Congressman had burnt the candle at both ends—work and whisky, wine and women, had made a wreck of him at 49; and a spree culminated in a paroxysm of angina in which he nearly died. Five years subsequently he was in excellent health, in spite of a high blood pressure and moderate sclerosis of the arteries. He dated the change of life from the attack which had frightened him into sobriety. A group of most encouraging cases show that long intervals of good health, and even complete freedom, may follow the adoption of reasonable habits. Let me refer to one or two. Last summer I met in London a distinguished American lawyer, whom I had seen ten years ago in a series of severe attacks, one of which nearly proved fatal. Following in the track of the intercostal pain he had an outbreak of herpes, the only instance I have seen of this complication. After six months' rest he resumed work at an easy pace. I saw in the papers a few weeks ago the death of General P. whom I saw in 1901, then aged 65 years. For two months he had attacks of angina, and once had a succession of paroxysms which kept him in bed for three days.

Not much stress can be laid on the character of the attack so far as the ultimate outlook is concerned. The immediate prospect is always doubtful; a mild paroxysm may be succeeded by a severe one, or death may occur with the appalling suddenness which is one of the features of the disease. The vaso-vagal features—pallor, sweating, faintness, and nausea—are serious in proportion to their prolongation and to the resistance they display to remedies. A man may come out of a state which seems absolutely desperate. One does not often see recovery and subsequent good health after such a paroxysm as in the following case, which was studied with great interest by Dr. McCrae and myself.

J. H. W., aged 60 years, admitted to a private ward on March 24th, 1904, was a vigorous, active man, the victim of many acute infections, but not syphilis. He had taken an average quantity of whisky and had worked hard. In June, 1902, when salmon-fishing, he had his first attack, then a second one in January, 1904; a third on Feb. 22nd, which was very severe, as he lost consciousness; a fourth on March 10th, also very severe; and a fifth a few days before he came into the hospital. He was a robust, healthy-looking man with a good colour, a feeble heart impulse, no superficial heart dulness, distant sounds; pulse of good volume, slightly irregular; marked sclerosis of the arteries. On April 4th at 1 P.M. he had an attack in which he looked as if he were dying; the hands and feet were cold and clammy, and at intervals he sighed deeply and threw up the chin; he was conscious; the pulse was from 42 to 48, regular but small; the heart sounds were feeble and distant. By 1.30 the ashen grey colour was replaced by slight lividity; the pulse rose to 52; the blood pressure was 120 mm. of mercury.
At 2 o'clock the pulse was 60, better, quieter, and there was less cyanosis. At 2.30 the pulse was 68; he vomited several times, and at intervals had very deep sighing respirations. He was in a very critical condition all the afternoon, but gradually improved. Two days later, at 1.30 a.m., he had a mild attack of pain in the region of the heart with intermittency of the impulse. He gradually improved and left the hospital on June 23rd. Through Dr. McCrae I have heard from him at intervals, and he has kept very well. This man had six severe cardiac attacks, in one of which he nearly died, and yet he has now remained well and active for the past six years.

In the attack itself there are two bad prognostic signs—disturbance of respiration and slow, irregular heart action. The acute orthopnoea with signs of emphysema and oedema of the lung is serious, and not met with except in grave cases. Death may occur or the attacks may replace those of angina. Slow, grasping breathing, particularly if the patient has become unconscious, is of bad omen. Cheyne-Stokes respiration, a frequent accompaniment, is a bad sign when it comes on in a man who has not had it previously, in connexion with cardio-vascular or renal disease. The state of the heart is very deceptive. A man may die shortly after an apparently satisfactory examination. Listen to this report, jotted down at the bedside of a man in an attack, and who died five hours subsequently. "Pulse 92, at first regular and of good volume, but subsequently one or two beats dropped. There is nothing to attract one's attention in the pulse itself. (The blood pressure taken in the same afternoon was 160 mm. of Hg.) The arteries are not sclerotic; the apex beat is not visible; and between a high stomach tympany and an inflated edge of the lung it is not easy to make out the heart limits; the sounds are clear at apex and base, a beat drops occasionally; there is no gallop rhythm, nothing to indicate any serious disturbance; there is no heightened sensibility of the skin over the heart." This man had had anginal attacks on and off for a year. The heart was working with not more than 30 per cent. of a coronary circulation; one main stem was entirely obliterated, the other partially.

The small, feeble, rapid pulse is not of such ill omen as one of fuller volume, with marked arrhythmia; and a slow pulse with unconsciousness is always of grave import.

The circumstances that bring on an attack are almost of as much moment in the prognosis as its character. We all recognise the three chief factors—emotion, distension of the stomach, and muscular effort. Of the three emotion is of the least significance. Many instances of slight anginal attacks are brought on by anger, worry, or sudden shock; and while in individual cases they may be serious, yet the cause is rather easier to avoid, and always has seemed to me to be less dangerous, though John Hunter neither thought so
nor found it so. In many elderly persons with stiff arteries the commonest cause is stomach disturbance; a full meal, indigestion, flatulence, particularly at nights, is certain to bring on an attack. And there are individuals who are sensitive to this cause only. As one patient expressed it: "Had I not to eat I never would suffer."

The angina of effort, the paroxysm that comes on invariably after a man walks a few hundred yards or after using his arms, is a coronary artery affair, and is, as a rule, more serious than that which comes on spontaneously or as a result of emotion. This feature is the most common indication of what you, Sir, call "primary cardiac angina." The parallel between the angina of effort and intermittent claudication has often been drawn, though the cardinal difficulty has been pointed out that in the one case a man stops walking, in the other the heart continues to work. A curious feature in some cases of this type is that a man may be able to walk freely in his place of business, or in his house, and not be able to do so on the street. While constituting some of the most distressing cases, since even the act of dressing may bring on pain (angor de toilette), yet life may be prolonged, as in the case I mentioned of the man who had been a victim for some 11 years, and whose coronary arteries were calcified to their finest ramifications.

And the third important factor in prognosis is the patient's cardio-vascular condition. Persistent high tension with marked sclerosis of the arteries occurs in a very large group of cases, but it is important not to over-estimate their seriousness. The outlook may depend much less upon the existence of these factors than on the sort of man in control of them. An engineer of the "McAndrew" type will so handle his machine that the ship makes the voyage round the Horn to London with safety; whereas a man of a different type will wreck the engines and leave his ship a derelict. A large majority of all cases of angina pectoris have no obvious signs of disease of the heart itself. Of valvular lesions the aortic alone is important—insufficiency in the young man usually means syphilis; stenosis in the old means calcified aorta, narrowed coronary orifices, and rigid coronary arteries. Disease of the aorta itself is not often recognised. The prognosis of cases of aneurysm beginning with angina is, of course, serious, though as the sac grows the attacks may disappear.

**FORMS OF ANGINA PECTORIS.**

It was a distinguished President of this College, John Latham, who in 1812, I believe, first recognised that "certain symptoms did not always denote angina pectoris," and spoke of angina notha; and it is a useful division which recognises a minor and major type of the disease—or what I think is still better, a form without, and a form with, organic
lesion of the cardio-vascular apparatus. A diagnosis of these two forms from each other is an essential preliminary to successful prognosis. It may have surprised some of you that the number of cases of mild or functional angina in my list was so small, but I carefully excluded all trifling forms of heart pain in nervous women, and have only considered the cases which presented the features of an anginal paroxysm. Of the 43 cases there were 23 in women, not so large a percentage as given by many authors. It is a useful division to group these cases of this type into the neurotic and toxic; the former occur much more commonly in women and the latter in men. In looking over the histories of 14 cases of the severer organic angina pectoris in women I see that in only two or three cases was the diagnosis in doubt. The average age was much above the other group—56 years. In not a single instance was there absence of sclerosis, high pressure, or heart disease, and the character of the attack in almost every instance stamped the nature of the case. I have more frequently mistaken organic angina for the functional form than vice versa. Two of my worst mistakes were in medical men, and for their sakes I did not regret it, since both derived great comfort from the thought that they were not the subjects of angina vera. One man would have deceived Heberden himself, as he had hysterical attacks, at any rate, nervous outbreaks with spasm of the larynx, a sort of child-crowing, and a nervous dysphagia. Let me read the note I dictated at the end of his report:—"Against recurring attacks of moderate severity in a man of 53 must be balanced a healthy heart, a very nervous temperament, and the occurrence of laryngeal and oesophageal spasm. The patient is very apprehensive and feels sure he is going to die. I think the balance is in favour of a neurotic, functional condition." I was wrong; three years subsequently he died in an attack. The other patient had not a trace of obvious organic disease, but he had driven his engines very hard. He had smoked excessively, and at the time of the attack was very emotional. He improved so much that we all congratulated ourselves upon the correctness of our treatment and diagnosis. He dropped dead while speaking at the telephone.

Many subdivisions have been made of the neurotic form, but for prognosis they roughly fall into two, the distinction between which is the predominance of the vaso-constrictor or vaso-dilator phenomena. Many cases resemble what Gowers describes as vaso-vagal storms. Beginning with coldness of the hands and feet, numbness and tingling, with small pulse, pains are complained of in the heart itself, often more to the left, and in the pectoral rather than in the sternal region; the radiation may be marked, up the neck and down the arm, and much more than in the organic form superficial sensitiveness of the skin is noted. The attacks may pass off in the course of an hour or two, but in the
severe ones there may be nausea and vomiting; the respiration is quickened, and there is an indescribable sense of fear and dread. With it all the patient may move about, and there is rarely present the characteristic immobility of the severe forms.

When the vaso-dilator features predominate the patient complains of fulness in the head, of a terrible distress in the back of the neck, or a general sense of superficial tension. As one woman expressed it: "I feel just like an inflated balloon on the point of bursting." The heart's action may be forcible, and there is widespread throbbing of the arteries. The face and hands may be congested and hot, the face sometimes slightly livid. Following these symptoms, or coming on at the same time, the patient begins to have heart pain, which may become very severe; the lines of radiation may be characteristic, and with these symptoms there may be remarkable prostration. One patient whom I saw very often in this type of attack never had cold hands or cold feet, and on several occasions so distended was the superficial vascular system that bleeding was suggested, but she would never consent. There is rarely much difficulty in determining the character of these attacks, with which are often associated neurasthenia and hysteria. Occasionally one meets with paroxysms of great intensity, in which as the pain reaches a maximum the patient is thrown into a sort of tetany. In a man aged 32 years, whose troubles began with palpitation, the pain was apparently very severe, and he had stiffness and cramp of the fingers, a sort of tetany in which they could not be moved. Sometimes he would get into a state of general rigidity, all the time complaining of terrible agony in his heart. Many patients have phobia.

The majority of these cases do well, and it sometimes happens, particularly in a man, that a reassuring diagnosis is all the treatment required. In attacks of extraordinary severity a fatal event may happen, as in the patient of Dr. Bullard, to which I referred in the last lecture. While gradual recovery in the course of a few months or a year is the rule, some cases are very obstinate. A man of 28, who had not worked for five years owing to constant recurrences of pain in the heart and along the inner side of the left arm, was exceedingly nervous, and after months of treatment we could do nothing with him. As he wrote pathetically: "I heard you say to the students that there was nothing wrong with my heart, but if you had it for a few nights and felt the pain in it, you would think quite differently."

The worst sort of neurotic angina may follow influenza, and the rapid recurrence of the paroxysms may render a man's life unbearable. An old student and valued friend in practice near Philadelphia, who was a little nervous and had occasionally had migraine, a month after influenza began to have the most extraordinary attacks. The pallor
and coldness of his extremities exceeded anything I have ever witnessed; nothing could warm them; even after his legs had been for 15 minutes in water as hot as he could bear, when taken out he complained that they felt cold. In the attacks the mental distress was agonising, and he had a sense of terrible constriction across the upper part of the chest. He threw himself about the bed, and the condition was most painful to witness. He gradually recovered.

My experience of an angina which may be termed toxic is very limited. I have not seen more than a dozen instances in which to tea, coffee, or tobacco could the attacks be attributed. We may look forward to an increasing number of cases of heart pain, and of the mild type of angina in women, with the rapid increase of cigarette smoking. I saw last winter in Italy an American woman whose daily allowance of cigarettes was never under 25. She had an unusual feature—cardiac pain only after rising from a recumbent posture, and this would worry her for half an hour, so that it made dressing in the morning difficult.

It is interesting to note that very heavy smokers may die a vagus inhibition death, just such as we see in angina pectoris. Three robust, healthy persons of my acquaintance, not known to have had heart disease, but all incessant smokers of very strong cigars, died suddenly in this way, without warning—one aged 53 while walking; one a man of 36 fell off a chair at his club; the other a man of 38 died on the beach after bathing.

**TREATMENT.**

A retrospect of one's experience in treatment will be coloured by the general character of the special disease under consideration. I look back with unmixed satisfaction at my experience with typhoid fever, every aspect of which may indeed be dwelt upon with pride by every member of the profession. On the other hand, pneumonia arouses feelings, also unmixed, but of a totally opposite kind. There have been certain gains: we know our enemy better, but there is no pleasure to be had in looking back upon the record. And the other night, in going over case after case of my typewritten reports, I could not help taking stock in this way of angina pectoris. And on the whole, in spite of the frequency of the broad arrow, the feeling was one of satisfaction. Terrible as it is in certain aspects, angina has many circumstances in its favour: it kills late, it kills quickly, we are able to do much to lessen the sufferings of the victims, and we cure a certain number of cases. There are but few deaths under 40 years of age; no known disease kills so peacefully, so painlessly, and there has been real and solid progress in the advance of our knowledge of how to treat it.
As with prognosis, so with treatment: there are three great groups to be considered—syphilitic, neurotic, and arterio-sclerotic.

When a man gets a specific aortitis it means he has not had efficient treatment. There is nothing in the lesion of the arterial wall which mercury and iodide of potassium cannot control. The spirochaetæ excite a diffuse granulomatous mesaortitis, with destruction of muscle and elastic fibres, and the chief difficulty arises from its insidious progress, so that irreparable damage may be done before any warning is given. The details of treatment offer nothing upon which I need dwell in this audience. The value of mercury, no matter what the stage of the syphilis, is emphasised when one actually sees the spirochaetæ in large numbers in the aortic wall, as has been demonstrated by J. H. Wright, since so far as we know the metallic drug alone acts as a specific. Iodide of potassium clears up the exudate and, as is well known, will cause a node to melt away even faster than will mercury. One thing, too, it does with almost invariable success—relieves pain. Here is the secret of its great influence in aneurysm in which in the early stage it is as good as morphia in giving relief, clearing the exudate in the media and adventitia, and in this way relieving the pressure on the nerve elements.

In the neurotic cases, with a recognition of a basic disturbance in the vaso-motor apparatus, the treatment is most satisfactory, and only a few cases prove refractory. A modified Weir-Mitchell cure with hydrotherapy meets the important indication. Long experience has taught the value of the wet pack in restoring stability in vascular ataxia. Counter irritation over the heart is sometimes helpful. Of drugs the patients have usually had enough by the time they reach the consultant, and one part of the battle is to wean them from all sorts of mixtures. One patient laid out before me 22 prescriptions, and was much aggrieved not to have a twenty-third. Sometimes a reassuring diagnosis is the only treatment needed. I have always been sorry that an article on angina pectoris was in the "British Encyclopædia," since it has helped to make the very name deadly in the ears of the public; and there is an advantage in speaking to nervous patients of a false variety, which may mimic every phase of the true disease. When high tension is present, which is not infrequently the case in neurasthenia, the nitrites are helpful, and they have a very special value in the toxic forms, particularly that due to tobacco. In the cases with extreme vaso-motor ataxia I have long used the extract of ergot (ergotin) with advantage two or three grains three times a day.

I have nothing to add to the general knowledge which we all have of the treatment of the severer type of the disease, but I may give you my experience very briefly: first of the treatment of the paroxysm itself and then of the general
conditions out of which it arises. It was not to be expected that our generation could have in any one disease a second therapeutic boon of equal importance to that given us by our distinguished Fellow, Lauder Brunton. Of the value of amyl nitrite in loosening arterial spasm and relieving the tension and strain upon which the pain depends we are all agreed. We see its remarkable benefit, more particularly in the cases which begin with a widespread vaso-constrictor influence. Many practitioners express disappointment that it does not always relieve the pain promptly in the severe paroxysms; but it is not itself an analgesic, but only loosens the muscle grip; and it may well be that the painful effect of the disturbance of tension may persist after the spasm itself has disappeared. At any rate, experience teaches us that we often have to resort to morphia to relieve the atrocious character of the pain. Moderate doses, as a rule, suffice; but it is to be remembered that, as Burney Yeo pointed out, there are cases extraordinarily resistant; and I have reported an instance of status anginosus in which, between 10 o'clock on Saturday night and 1 P.M. on Sunday, five grains of morphia were given hypodermically and by the mouth with relief of the pain, but without giving sleep. In these terrible cases, in which attack follows attack, the nitrites are of as little use as water. In despair one may have to resort to chloroform. I well remember to have done so at first with fear and trembling, as the patient's heart was feeble, but it seemed imperative to give the poor fellow relief. I have used it many times since, and never with ill effects; in no case did sudden death occur during the administration or immediately after it. Theoretically the practice is a risky one, but neither in my hands nor in those of the late George Balfour of Edinburgh were there ill consequences.

Oxygen inhalations are useful, particularly when there is a dusky cyanosis and in the asthma-like dyspnea. In the weak, failing, irregular heart one is tempted to give digitalis, and it may be tried, preferably by the hypodermic method. There is one type of case in which the drug should be used—when marked cardiac weakness follows an acute single attack in a comparatively healthy man. Post-mortem experience shows that the lesion in such a case is very likely to be blocking one of the coronary vessels. While anatomically the coronaries are not endarteries, functionally they are, and an infarct is, we know, very apt to follow; and if after the attack a pericardial rub is heard we may be certain this has happened. By maintaining the pressure within the myocardial vessels the extent of the anemic necrosis may be lessened.

For the general condition the value of iodide of potassium in arterial lesions, and specially in the syphilitic variety, is universally recognised, and it may be given at intervals for months. Last winter I had an interesting talk with the
distinguished Roman clinician Marchiafava, whose experience in angina pectoris is very large, and he gave me reports on cases treated with theobromia, from 20 to 30 grains in the day. A number of them were greatly benefited. I have only had the opportunity of trying it twice; in one case the use had to be interrupted on account of a complication; the other patient has been remarkably helped, particularly in the capacity of taking more exercise.

In the middle-aged man who has had an attack of angina it is usually a question of high tension with beginning or actual sclerosis of the arteries. So soon as a man has crossed that point in life when the pace is the fastest, whether we put it at 25 with Plato, at 40 with Montaigne, or to be more kind, let us put it with the sexagenarian himself, at the grand climacteric (63), the tubing begins to show signs of wear and tear, and the blood pressure gradually rises. Now there is nothing more difficult than permanently to reduce persistent high blood pressure. Drugs have really very little influence. The nitrates are helpful in temporarily lowering it; but take a man with a persistent pressure of 230 to 240 mm. of Hg, and you may get the record to 210 or 220, but to get it back to 150 or 160 and keep it there is not often within our power. Much more important results may follow change in a man's habits of life. I usually give two prescriptions—"Go slowly," "Eat less"—on which I find a great many patients put about the same value as did Naaman on the prescription of Elisha. A man who has kept a full head of steam in the boilers must learn to lower the pressure and be content with the quiet 10 knots an hour speed. It is very difficult to stoke the engines in due proportion to the work expected. No wonder there is high pressure and the machine goes to pieces when the furnaces are stoked for the Lusitania and the engines are asked to do the work of an ordinary ocean tramp. Like longevity, angina pectoris is largely a question of the arteries. It is an old story, this association of a long life with a small intake, a story well told by Cornaro in the sixteenth century, and in our own day by our own Cornaro, Sir Herman Weber.

If, Mr. President, I have dealt with this important subject in a somewhat sketchy manner my apology must be that before such an audience I could not discuss trite and everyday features of so familiar a disease, so I thought it would be more interesting to give you my personal experience. It adds salt to life when men react differently to the same impression. It is always with a shade of regret to find a colleague of the same way of thinking with myself on every question, so that I hope you have not all agreed with all of my conclusions. At any rate, Sir, mindful of the wise counsel of Lucretius, I have tried not to base wide opinions on small signs, and so involve myself in the snare of self-deceit.

1 Bk. iv., 816, 817.
CERTAIN VASOMOTOR, SENSORY, AND MUSCULAR
PHENOMENA ASSOCIATED WITH
CERVICAL RIB.

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The symptoms of cervical rib have attracted much attention of
late years, and are discussed in the exhaustive papers of Keen,\textsuperscript{1}
Thorburn,\textsuperscript{2} and Lewis Jones.\textsuperscript{3} The nervous and muscular features
are those which most often attract attention. Last year I saw two
remarkable cases, one of which threw light on a very inexplicable
condition of the arm, which I had described years ago.

Mrs. L., aged thirty-one years, a strong, healthy woman, was
referred to me by Dr. Andrew, of Thame. There was nothing
special in her family or personal history. For ten or twelve years she
had noticed a pulsation above the clavicle on both sides, most
marked on the left; the physicians who first saw her suggested the
possibility of an aneurysm. What has troubled her of late has been
that after using the left arm for a short time there is a sensation of
numbness, sometimes of "pins and needles," and if she continues
to work, the skin gets red, and the arm feels swollen and hot; then
in a little while she is quite unable to use the arm, and even has
dropped things from her hand. The condition has increased very
much of late, and it is for this that I was consulted. When quiet
and at rest the arm feels natural, and she can do the ordinary work
with her fingers. It is only when she attempts to use the arm that
numbness and tingling begin; then if she persists, redness and swelling follow, and finally she has to give up work. She has become
ervous about it, and two months ago she appears to have had an
attack of unusual severity, in which she fainted.

She was a very healthy looking woman with high color, no cyanosis;
the radial pulses were equal; above the clavicles there was pulsation,
somewhat more forcible on the left side on which it extended from
the outer end of the clavicle upward and inward toward the thyroid.
It was very noticeable, and one was not surprised that it had been
regarded as aneurysmal. There was no pulsation to be seen in the
sternal notch. After exertion, and in the erect posture, the left

\textsuperscript{1} AMER. JOUR. MED. SCI., 1907, cxxxiii, 173.
\textsuperscript{2} Med.-Chirurg. Society's Trans., 1905.
\textsuperscript{3} Quarterly Jour. Med., vol. 1.
supraclavicular space looked fuller than the right, and a marked pulsation occupied the whole of the lower triangle. On palpation, no definite tumor could be felt, nor could one grasp a vessel between the fingers, and yet the pulsation was marked and distinctly arterial. The swelling was a little tender; on deep pressure one felt a resistance suggestive of a cervical rib. On auscultation, there was a systolic murmur over the vessel on the left side, none on the right. The left arm looked smaller than the right. There was no wasting of the muscles of the hand. Sensation was everywhere perfect. After moving the arm up and down, and working the muscles, she complained that the skin felt prickly and numb, and a flush extended over it, but no swelling followed. This, she says, only comes on if she persists in using the arm. The heart’s impulse was a little forcible, but the sounds were loud and clear. Dr. Sankey took an x-ray picture, which showed well-marked cervical ribs on both sides, curiously enough, the larger one on the right.

The special point of interest about this case to me was the explanation it offered of two very remarkable cases, one of which I showed at the Philadelphia Neurological Society. I give here a brief abstract: A man, aged forty-eight years, always very healthy and strong, a carpenter by occupation, complained of inability to use the right arm, which had been gradually coming on for some time. When at rest and quiet, it felt perfectly natural, and all the ordinary actions of life could be done without discomfort. There was no pain, no numbness or tingling, and the hand and arm looked natural; but when he worked, or used the right arm for more than a few minutes, he began to feel an unpleasant sensation and numbness and great tenderness; the color of the skin changed, and the whole arm became congested and swollen. This would occur in a very few minutes, and the veins would stand out with great prominence. There was a general dusky lividity of the skin. If the exercises were continued, the arm became visibly swollen. At rest, the circumference of the thickest part of the forearm was eleven inches, after exertion twelve and one-half inches. At rest, the radial pulse on the two sides seemed normal and equal; after exertion the right radial became very small, only just perceptible. When the arm was held up above the head, the congestion and swelling rapidly disappeared. Nothing whatever could be felt in the axilla, or in the course of the bloodvessels. I saw this patient at intervals of six months longer; he could do no heavy work, but all ordinary minor actions could be done without any swelling of the arm. The case was one that excited a good deal of interest, but no very satisfactory explanation could be offered. Unfortunately we did not at that time appreciate the importance of cervical rib, which I do not doubt was the cause of the remarkable disturbance in this case.

The other case was a woman, aged thirty-eight years, who came to my out-patient clinic at the Johns Hopkins Hospital, complaining of redness, pain, and stiffness of the right arm on exertion. At rest, the arm looked natural, but when used for ten or fifteen minutes there was a remarkable change—the skin became flushed, the fingers slightly cyanotic, and she complained of a feeling of stiffness with numbness and tingling, and if she continued to work the hand, the forearm became swollen and so stiff that she had to stop. There was no disturbance of sensation, no atrophy of the small muscles; the pulses were equal; there was nothing to be made out in the chest, or in the course of the arteries. I suspected at first pressure of glands high in the axilla, but nothing could be determined by the most careful examination. I did not think of cervical rib. The condition had persisted at intervals for several years and was the cause of great disability, as she could not work continuously for any length of time. If she did not use the arm there was no inconvenience.

In both these cases the symptoms, though more aggravated, were identical with those complained of by Mrs. L., and I have no doubt that, could we have taken x-ray pictures, cervical ribs would have been found. In Keen’s paper several cases are reported in which the hand and forearm became livid and swollen. But it seems probable that there is a special group in which the symptoms come on only after exertion, and they resemble closely the condition known as intermittent claudication. In many cases the subclavian artery has been compressed in the angle between the rib and the scalenus anticus. When at rest, and with very slight muscular effort, enough blood reaches the limb, but the demand for more blood which follows exertion is not met, and there is stiffness and numbness with vascular changes. So marked may these latter be, that there are cases reported suggesting Raynaud’s disease, and Keen states that in at least seven instances local gangrene has followed.

An important suspicion was raised in this case as to the existence of aneurysm. As Keen remarks: “On the whole, the evidences of true aneurysm in most of the cases in which it has been reported are, to my mind, by no means always convincing. Usually the diagnosis has been based on the strong pulsation, sometimes with bruit and thrill. In a few cases, as in my own, the artery has been found, at operation, moderately enlarged, or in one case (Murphy’s) flattened. In several cases operation has, therefore, disproved the presence of the supposed aneurysm. The postmortem in Adam’s case disclosed a cylindrical aneurysm. Bearing upon the history of pressure or tension of the artery as it crosses the cervical rib, it is significant in Fischer’s (Braun’s) case that when the arm hung down (pressure or tension) a bruit was present, and when it was held up (relief of pressure) the bruit disappeared.”
But unquestionably, in a few cases, dilatation of the vessel, a cylindrical aneurysm, or even a sacculated tumor, has been present. In the case here reported, though the pulsation was diffuse and suggestive, there was no evidence of aneurysmal dilatation. To the Infirmary for Nervous Diseases, Philadelphia, Rose D., aged nineteen years, an inmate of the Pennsylvania Blind Asylum, was brought for a supposed aneurysm of the subclavian. There were the local paralysis and atrophy which one now recognizes readily enough as

![Diagram of the subclavian vessel passing over a cervical rib](attachment:diagram.png)

Scheme to show the changed course and the consequent angulation of the subclavian artery when it passes over a cervical rib. The dotted line (3) represents the normal gentle curve of the artery. 1. Cervical rib. 2. Subclavian artery passing over the cervical rib. Note its high position, its angulation, and the likelihood of pressure by the scalenus anticus 6. 4. First dorsal rib. 5. Axillary artery. 7. Left carotid. 8. Innominate. 9. Aortic arch. (Keen.)

characteristic of cervical rib. Over this, no doubt, the subclavian artery was hooked, as in Keen's diagram, which I here reproduce, as it shows this angulation and elevation. It explains the position of the swelling in these cases, and the high pulsation. In the case of the blind girl just referred to the swelling in the supraclavicular region was marked, and while the mass itself did not pulsate, the vessel could be readily traced, and there was a loud murmur heard along its course.
In Address
ON
THE HOSPITAL UNIT IN UNIVERSITY WORK

Delivered before the Northumberland and Durham Medical Society

BY

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GENTLEMEN,—I offer no apology for bringing before you an academic subject. Though the society has no direct university affiliation, as individual members you are interested in the development of the profession, and you take a pride in the rapid growth of the provincial universities, among which that of Durham, the northern sister of Oxford and Cambridge, takes precedence. What is university work? What is a hospital unit? What connexion have they with each other? And what interest have they for us, and for the community at large?

The Functions of a University.

All are agreed that a university has a dual function—to learn and to advance learning. I use the word "learn" in the old sense—met with in the Bible and still used colloquially—as it expresses the mental attitude of the student towards his alma mater, "totius litteratorii studii altera prima." In mind, manners, and morals the young man seeks life’s equipment when he says to his alma mater in the words of the Psalmist, "O learn me true understanding and knowledge." To learn the use of his mind, to learn good manners, and to learn to drive Plato’s horses, form the marrow of an education within the reach of every citizen, but to which universities minister in a very special way; and it should be comprehensive, fitting a man in Milton’s words "to perform all the offices, private and public, of peace or of war." The other great function of a university is to advance learning, to increase man’s knowledge of man and of nature. Looking over the lecture list of any modern university one is impressed with the bewildering complexity of subjects taught, from Homer to Victor Hugo, from Tamil to internal secretions; but they may be roughly grouped into those dealing with man and those dealing with the cosmos about him. At any time these 800 years this division has been
recognised, and though we have travelled a long way from
the seven liberal arts which once comprised the whole range
of study, it is not so much the nature of the subjects or
their division that characterises modern education as a
new spirit, a new attitude of mind towards them. No
real progress was made until we returned to the Greek
method—the pursuit of knowledge for its own sake. Out of
the laboratories, as the result of work done by men absorbed
in study and usually without the slightest bearing upon
practical problems, came the three great revolutions of the
nineteenth century—the annihilation of time, the substitu-
tion of the machine for the hand, and the conquest of
disease. Physics, chemistry, and biology have given us
control of the forces of nature. Faraday has harnessed
Niagara, the power of which is now transmitted hundreds of
miles away; the Curies have found the magic viril of Bulwer
Lytton's "Coming Race"; and Pasteur has revealed one of
the greatest secrets of life. It is characteristic of
modern conditions that, hovering on the borders of the
charmed circle of pure science, are those keen to turn
every discovery to practical use. What good is knowledge
unless it can be utilised in the service of man, asks
a utilitarian age? The university of to-day, while
ministering to the advancement of learning, is ready
to teach how to make the learning profitable, so that
everything in practical science, from household economy to
aviation, finds its place. Schools specially adapted to
special needs stand out as dominant features in the new
programme, and Oxford and Cambridge, as well as Newcastle,
Leeds, and Bristol, have felt the strong impulsion to develop
the science which deals with human well-being. Of the old
faculties which made up the studium generale medicine has
been the one most profoundly affected by growth of modern
science. What a revolution in our generation! Anatomy,
physiology, and pathology, with their subdivisions of
histology, embryology, physiological chemistry, and pharma-
cology, are now in laboratories controlled by specialists,
whose ideals and work differ in no respect from those
of their colleagues in the departments of physics,
chemistry, and biology; and in many places large separate
institutes are devoted to these subjects. The urgent need
to-day is to extend this type of university work into our
medical schools, so that all branches of the curriculum are
included—medicine as well as pathology, surgery as well as
anatomy, midwifery and gynaecology as well as chemistry.
But here comes a difficulty—the practical schools which deal
with these important subjects and their subdivisions are not
under the control of the university, or at best have a very
feeble affiliation. In this country the hospitals are either
independent corporations, as here, or the medical school has
evolved from the hospital, as in London, or there exists a
mutual arrangement between the university and the hospital,
as in Edinburgh. The difficulty is not in-superable—the same public-spirited citizens support both institutions; the hospital staff includes the teachers in the medical school. It only needs a rearrangement of responsibility, financial and educational.

THE HOSPITAL UNIT.

What is a hospital unit, and how can it be brought into line with university work? First a word on what a hospital stands for in the community. Primarily for the cure of the sick and the relief of suffering; secondly, for the study of the problems of disease; and thirdly, for the training of men and of women to serve the public as doctors and nurses. A majority of hospitals deal only with the first of these objects, and incidentally with the third. All agree that a study of the problems of disease and the training of men and women in the technique of the art come within the sphere of the university. England has suffered sadly from an absence of great medical faculties, such as exist on the Continent; and nowhere is this more evident than in the dissociation of the hospital from the university. One consequence has been that the hospitals have been built by men who had no idea whatever of their scientific needs, and too often staffed by men who knew little and cared less for anything beyond their primary function. The present plan of hospital administration is a legacy from a period when university ideals had not reached the practical side of our medical schools. I need not do more than to refer to the arrangement of the staff which exists everywhere—three or four physicians, two or three surgeons, one or two gynaecologists, other specialists, and a group of juniors who serve as out-patient assistants, waiting for promotion to the wards. The pathological department, often only a dead-house so far as the hospital is concerned, is in no way coordinate with the others. Laboratories of bacteriology, clinical chemistry, microscopy, and of clinical physiology may or may not exist. This English system, which has spread to the United States and to the Dominions, has worked well in some ways, and is responsible for the general excellence of the hospitals, large and small. Go where you may, from the cottage hospital to the big city infirmary, the internal economy, so far as cleanliness, general care of the patients, and nursing, is admirable. Speaking from a comparatively wide experience of hospitals, I say unhesitatingly that the average level of comfort and care is nowhere so high as in this country, and in no small measure is this a tribute to the character and training of the women connected with them. But there are very glaring defects, foremost among which is the absence of proper laboratory accommodation in pathology, bacteriology, clinical chemistry, and microscopy. Time and again in large well-arranged hospitals I have asked for the patho-
logical laboratory, and have been shown a dead-house; for the clinical laboratory, and have been told that it did not exist; and have been chagrined to find that even in so simple a matter as the determination of the nature of a tumour, or the bacteriological examination of a fluid, the institution had to depend upon the excellent clinical research laboratories of London. But an equally grave defect is in the internal organisation. The general hospitals, even those connected with medical schools, are as a rule overstaffed, four physicians or three, where there should be but one or two, and the same with the surgeons. On the other hand, in many the resident staff is miserably inadequate, and their time so taken up with routine that any scientific study of cases is impossible. A keenly interested physician has a capable house physician in good training—off he goes at the end of six months! and the same weary process begins of putting a fresh man into harness. In the very best hospitals, with medical school affiliation, the arrangements are on old and very unsatisfactory lines. In long, uphill years the ambitious young man goes through the position of resident physician, medical registrar, assistant physician, and at 40 (if he is lucky!) gets wards. Then a visit two or three times in the week with a house physician, a certain amount of teaching, and possibly some laboratory work, but he has a living to get and practice becomes the first consideration. He has precious little pay, if any; there are no paid assistants; there is no continuity in the organisation; in fact, there is no organisation on modern lines. This can be changed if we can convince the authorities that the subjects of clinical work come directly within the sphere of the university, and that certain hospitals must be adapted to meet the demands of the scientific study of disease and the scientific training of students. The problem is how to place a dozen or more teachers in every medical school in the same relation with the university as the professors of physiology and of physics—how to give to each one of them a department organised on university lines, in which the three functions of a hospital may be utilised and coordinated. The hospital unit meets the condition—a department under the complete control of the university, or under the joint control of hospital and university. Take a medical faculty with, say, 300 students, for which the necessary hospital accommodation would be about 500–600 beds, a unit would represent a clinique in the continental sense, of which there would be five or six major, and as many minor—the former including medicine, surgery, midwifery and gynaecology, psychiatry and neurology, pediatrics, and ophthalmology; the latter, dermatology, dentistry, otology, syphilis, and genito-urinary diseases. There might be, as at Berlin and Vienna, two or three medical and the same number of surgical units. In Vienna, for example, there are the completely equipped midwifery and gynaecological units, each with lecture rooms, laboratories, rooms.
for students, and the whole paraphernalia for teaching and research.

THE ORGANISATION OF THE UNIT.

Let us take medicine, the one with which I am familiar. The components are the professor or director, 60 or more beds, an out-patient department, four or five laboratory rooms, and a staff. Let us deal with these in detail.

The Professor has three duties—to see that the patients are well treated, to investigate disease, and to teach students and nurses. He should be a man with wide sympathies and of trinocular vision. He should have a comprehensive and thoroughly scientific training, and should enter clinical medicine through one of three portals—physiology, chemistry, or bacteriology and pathology. He must be keenly practical, keenly scientific, fond of his patients, fond of his work, and devoted to his students. He should live as much in his wards and laboratories as do his colleagues in their laboratories of anatomy, physiology, chemistry, or physics. The question at once comes up, Is this possible? Can we expect surgeons, physicians, obstetricians, and specialists to devote themselves entirely to University work, and to carry out the three functions of the hospital, without regard to the public outside? Theoretically it sounds feasible, but in practice I do not believe it to be possible. In the case of a successful teacher with a widespread reputation, the public, to say nothing of the profession, could not be kept away from him. But it would not be unreasonable to ask such a man to devote at least one-half, or even more, of his time to the hospital duties. For the heads of the different units the university would go into the open market and seek the best men available. Under our present system the university choice of professors in the practical departments of the faculty of medicine is, as a rule, limited to men who hold hospital positions in a town. It is encouraging to see that the provincial universities are departing from this old practice, and I am very glad that you lost your distinguished townsman, Dr. George Murray, who was appointed to the chair of medicine in Manchester.

But it may be urged—have we not heard that the day of the pure clinician is over?—did not our Opsonator maximus, Sir Almroth Wright, pronounce his funeral oration a few months ago at the Royal Society of Medicine? Do not listen to him—Clinicus perniciosus, to quote Mindererus, with vision so myopic that he can see only applied bacteriology in clinical medicine. As is the twig, so is the tree, and with his upbringing such opinions are perhaps natural; and it is not for me to abuse an old friend, of whose good work no one has a higher appreciation. But in these days so winged are Sir Almroth Wright's words that they fly far and
need a protest from one who has been keenly alive to every bacteriological advance in medicine. Think of what would have happened if a man of Sir William Broadbent’s wide sympathies, clear judgment, and enthusiasm had had a great modern clinique at St. Mary’s Hospital, such as those of Leyden or of Kraus at Berlin—do you suppose the bacteriological tail would have wagged the clinical dog? Far from it! Sir Almroth would have done just as good work as director of the bacteriological laboratory of the clinique, and vaccine therapy would not have received such tardy recognition, and would have been in just as satisfactory, or possibly in a more satisfactory position. A striking comment, too, on Sir Almroth’s claims is the fact that the medical and surgical staff at St. Mary’s are still on duty!

Patients.—In the case of the medical clinique the number will depend upon the size of the medical school. In a hospital to which many acute cases are admitted a male and a female ward with about 30 beds each, and with a few small extra rooms, would furnish ample clinical work for a senior class. There are continental cliniques in which double this number of patients are treated, and the matter is one for organisation. To make the teaching and the work of the unit effective, it is essential to give the professor control of an out-patient department, with proper arrangements for the study of the cases and for the teaching of the junior students. In how many out-patient departments is the university side of the hospital problem considered?

Laboratories.—It is a characteristic of modern medicine that every available science is pressed into her service in the study and treatment of disease. Any sort of rooms will do for laboratories. The very best of work has been done with the meanest sort of equipment. The ideal plan is that followed in certain hospitals, the new cliniques, for example, of the Cochin in Paris, in which each unit consists of two wards on either side of a central building, the lower floor of which is devoted to administration, and the upper to laboratory purposes. In every way it is a convenience to have the laboratory rooms in close proximity to the wards, and when feasible they should form an essential part of the unit, but for the sake of economy they may be merged, and all the pathological work concentrated in one building. Nowadays provision should be made for (1) cardio-respiratory physics and physiology; (2) bacteriology and vaccine therapy; (3) chemistry; (4) microscopy; (5) X ray and electrical work; and (6) a general laboratory for the students. From the standpoint of the teacher the most important of these is the large laboratory for the students, in which the subject of clinical microscopy is systematically taught to the juniors, each man having his own place and his own microscope, and
in which the seniors work while acting as clinical clerks. No benefaction to the Johns Hopkins Hospital was more helpful to the medical school than that in which some kind friends gave the necessary money for a clinical laboratory. The best general clinical and pathological laboratory I have seen lately is that newly opened at the hospital which gave me my start in life, the Montreal General. The arrangements for students are admirable.

_The staff._—The present arrangement existing in almost all English, American, and Dominion hospitals is antiquated, and in every way ill-adapted to modern conditions. A man is given a house physicianship for six months, or possibly a year; there is a senior resident physician, and there are laboratory assistants, who may or may not be at the disposal of the professor. Gradation, permanency, and specialisation should prevail as in any other university department. There should be four groups. First, the senior assistants nominated by the professor, holding university as well as hospital appointments, and who should be prepared to remain for an indefinite number of years. In a clinique of moderate size three such could be utilised. The first assistant, a man of some maturity, should be in full charge of the department in the absence of the professor. The position should be made attractive and ample opportunities should be given for original work and for teaching. From this group the professoriate throughout the country would be recruited. Secondly, the junior assistants, house physicians, appointed annually and by competition from the members of the senior class. Thirdly, laboratory chiefs, who need not be resident; but it is a good plan to have one of the senior assistants in charge of the laboratories, and one who is at the same time responsible for the laboratory teaching of the students. The arrangements vary greatly at different clinics in accordance with the leanings of the professor towards chemistry, physiology and physics, or bacteriology. Fourthly, an out-patient staff made up of assistant professors in the department, as a rule men in private practice, and who are in charge of the junior teaching. At the Johns Hopkins Hospital we had three men in charge of the medical out-patients, each of whom took two days in the week, each with his own staff of assistants and a group of students assigned as clinical clerks. Special out-patient departments may be managed by the clinique; for example, the Tuberculosis Dispensary, which at the Johns Hopkins Hospital, under the care of Dr. Hamman, has grown to be one of the most important departments of the hospital.

_Teaching._—Entering one of the clinical units, a student should be made to feel a part of it, having his share in caring for the patients and in studying the disease, and even in the teaching. He should be made to feel that
the hospital is his home. This has been one of the special boons enjoyed by the British medical student in his senior years; the hospital has been in reality the medical school, and there has not been that disassociation between the two which exists too frequently elsewhere. The teaching should be entirely practical. In a few systematic lectures some of the more rare affections may be considered. Brought into daily contact with disease, the students gradually learn to recognise it, and are taught the management of patients. In clinical lessons and in demonstrations the professor and the assistants would in the course of a year cover the ground very thoroughly. In amphitheatre clinics it is easy to keep all senior students in touch with the department, while the special group, acting as clinical clerks, spend the greater part of their time in the wards. The five years of the curriculum should be divided equally, and the 30 months given to the hospital would enable a man to go the rounds of the departments and get a very full insight into the work. Hospitals have no vacations, and the old-time vacations should be done away with, and the school year divided into quarters, during which the work would proceed continuously. A man could take a week or so of vacation when he felt it was necessary, but it is high time a stop was put to an atrocious waste of time on the part of the students. The professor and his assistants should keep in mind the fact that the men entrusted to their care are to become practitioners, and as far as possible the practical work of the wards should be done by them. Upon one thing I would insist—that every assistant connected with the clinique taught. A few exceptional men, like the distinguished physician, the late Professor Rowland, are really too good to teach; but for the majority, daily contact with students, and a little of the routine of teaching, keep us in touch with the common clay and are the best preservatives against that stakeness so apt to come as a blight upon the pure researcher.

Research work.—Just as the big chemical laboratory of the university exists for research, so should the clinical units help to advance our knowledge of the causes of disease and of its treatment. The problems are innumerable, and in a well-organised clinique there will be men working at them in almost every department. A few of the better students may always be utilised for this purpose. The important thing is to keep one section at least of the clinique well in the fighting line, battling with the problems of to-day, in metabolism, immunity, cardio-respiratory physics, &c.

The other units would be organised in the same way, and, as I remarked, in large schools there could be three, or even four, medical and surgical cliniques. The important thing is to organise each unit on university lines. For example, the professor of psychiatry should be ex-officio in charge of
the city asylum, managed as one of the departments of the university, and into which, as a matter of routine, each medical student would pass for one of his ten hospital trimestres.

The truth is, we need an active invasion of the hospitals by the universities. But—and here comes the rub—the universities must be willing to undertake their share of the expenses, and the men in charge of the units must be paid salaries sufficient to enable them to devote one half at least of their time to hospital work—to give it the first place in their lives. At present in many places, indeed one may say everywhere, the university does not pay enough for its hospital privileges. How much, I would like to know, does the University of Durham pay to the Royal Victoria Infirmary? Nowadays the physicians and surgeons give their time, but the public, through university channels, should pay this group of men, not only to care for their sick poor, but to train their doctors and nurses, and to study for them the problems of disease. It is impossible to separate the three primary functions of a large hospital, of the machinery of which the medical student is just as much a part as the nurse, and so involved are the new methods of diagnosis, of laboratory treatment, and of research with the utilisation of skilled scientific assistants that it would puzzle anyone to assign the proportion of cost to A, B, or C. A good rule is that followed at the Johns Hopkins Medical School—the University paid half of the salaries of those members of the staff who were directly engaged in teaching and one half, I think it was, of the laboratory expenses.

As the old order changes new developments must be met, and here in the provinces, less hampered by traditions and not tied in the chains of vested rights, you can reorganise the hospitals on these lines through the universities. I have not been discussing Utopia; it is being done elsewhere. In the United States and the Dominions these changes are rapidly progressing, and the hospitals, for example as in Toronto, are being built on a modern plan, with units such as I have described. It will take time and much money, but it can be done; in fact, it has to be done if British medicine is to adjust itself to the new conditions, and so maintain its splendid traditions. Nor is it a matter which concerns the mother country alone. In former days the over-seas students came here for all their work; now the majority of them take their qualifying studies at home and come abroad for post-graduate work. The students from various parts of the Empire should come to our universities and hospitals and find thoroughly organised departments, with laboratories as well equipped for the study of the problems of disease as are those of chemistry and of physics for the study of these subjects.
SULLE TELANGIECTASIE EMORRAGICHE EREDITARIE

Estratto della "Riforma Medica", Napoli, 1911 - N. 3
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In tutti questi casi, la malattia fu ritenuta associata, in qualche modo, all'emofilia. Chauuffard
propone, per essa, il nome di emofilia cutanea. Rendu la chiama « pseudo-emofilia ».

I primi casi da me osservati furono due fratelli, nella cui famiglia erano stati affetti sette membri. I pazienti avevano sofferto di epistassi sin dalla fanciullezza, ed oltre a ciò erano andati soggetti ripetutamente ad emorragie da numerosi piccoli angiomi cutanei. Nel terzo caso da me riferito, trattavasi di un uomo di 49 anni, il quale, sin dal suo decimo anno d'età, andava soggetto di quando in quando ad epistassi, ed oltre a ciò ad altre emorragie, da telangiectasie della lingua, delle labbra, della faccia e delle mani. Le emorragie nasali provenivano da numerosi angiomi disseminati. All'epoca in cui pubblicai questo caso, io riferii, giusta le informazioni avute, che nessun altro membro della famiglia era affetto; appresi però posteriormente, che uno dei figli del paziente presentava dei nevi vascolari. Il quarto caso da me osservato, concerneva un uomo di 53 anni, nella cui famiglia avevansi delle epistassi e delle telangiectasie già da tre generazioni. Questi quattro casi trovarsi riferiti per esteso nel Quarterly Journal of Medicine, Oxford 1907.


Recentemente ho osservato un altro caso, in cui erano affetti un padre ed una figlia.

Quest'ultima era una signora trentacinquennne, la quale fu da me osservata insieme col D.r Anderson, di Londra. Il padre della paziente soffriva già da molti anni di gravi epistassi ricorrenti, ed avea la faccia coperta di telangiectasie. Oltre a lui, non vi fu nella famiglia alcun altro caso, se non
quello di sua figlia. Questa soffre, sin dall'infanzia, di epistassi ricorrenti, e non è passato quasi mai un intero mese, senza che essa vi sia andata soggetta. La paziente ha presentato, sin dall'infanzia, alcune telangiectasie; ma il numero di queste è considerevolmente aumentato negli ultimi sei anni, massime sulle labbra. Essa è stata sottoposta, con notevole vantaggio, alla cauterizzazione delle telangiectasie nasali e delle linguali. Oltreché da queste due sedi, la paziente ha presentato emorragie anche dalle labbra.

L'inferma è una donna di statura media, con viso spiccatamente pallido. Essa presenta piccole telangiectasie disseminate sulle due guance, ed una di circa 3 millimetri di diametro sul dorso del naso. Sulle labbra si osservano molte piccole telangiectasie di color rosso vivo, di 2-4 millimetri di larghezza, ed oltre a ciò, tre o quattro angiomi più grandi, rosso-bluastri, di 10-15 millimetri d'estensione, che sporgono come tumoretti emisferici. Questi erano da prima assai più piccoli, e sono man mano aumentati di volume negli ultimi anni. Sulla lingua vi sono una mezza dozzina di nevi, due dei quali di 5 millimetri di diametro, e forse più. Nessuno se ne osserva alle guance, né alla faringe. Sulle dita vi sono alcune telangiectasie puntiformi disseminate, una delle quali sotto l'unghia dell'indice sinistro. La pelle del resto del corpo ne è libera. La mucosa del setto nasale è coperta d'una spessa crosta di sangue, per effetto d'un'emorragia verificatasi due giorni prima.

Il solo fattore etiologico di qualche importanza è l'eredità. Nella letteratura son ricordate, sinora, 14 o 15 famiglie, nelle quali la malattia occorse per due a cinque generazioni. L'affezione sembra manifestarsi con ugual frequenza nei due sessi. Talvolta essa incomincia sin dall'infanzia, tal'altra le telangiectasie non appaiono che nell'età adulta. La pelle della faccia e la mucosa del naso, delle labbra e della bocca, sono prevalentemente affette; meno spesso la pelle delle mani; di rado quella delle altre parti del corpo.

Le emorragie provengono sempre dalle telan-


Molto può farsi per la cura della malattia, di-struggendo le piccole telangiectasie con la cauteriz- zazione. Solo così può sperarsi di ottenere una ces-sazione od un’attenuazione delle epistassi ricorrenti. In un caso riferito da C o e, si ricavò gran giova-mento dalla somministrazione di lattato di calcio. Allorchè si tratta di angiomi di una certa grandezza, come quelli delle labbra nel caso su riferito, è cons-igliabile di sperimentare il radio.
HEADACHE, vertigo, convulsions, aphasia, paralyses, and a progressive dementia are among the cerebral manifestations of arterio-sclerosis. Death "at the top" may be slow, as in the old oak with which Dean Swift compared himself; or it may be sudden, when a vessel ruptures; or more gradual, if thrombosis occurs. These may be called the major manifestations, but there are others less serious though of great importance, as their significance may be overlooked or misinterpreted. To headache and vertigo I shall not refer, since every one now recognizes how common they are as early symptoms of arterio-sclerosis in the young, and more constant features in the aged. It is more particularly to the transient aphasias and paralyses, cerebral crises as they have been called, occurring in states of high blood pressure and in arterio-sclerosis, to which I wish to call attention. Within a few weeks of each other I have recently seen two cases which illustrate the character of the attacks, and the first case is unusual since, so far as could be determined, only high blood pressure existed.

A well-built, active man of forty-three, who had driven his engines at a maximum speed for twenty-five years—keenly occupied in business, using tobacco freely, and intensely devoted to Bacchus, Venus, and Vulcan,—returned on the afternoon of March 1st, 1910, to his hotel, rang the bell for the servant and found that he could not speak. Perfectly conscious, he could not say a word, and was very much upset, and still more so when he found he could
not write. He was a little dazed mentally, as he could not tell the time. He became emotional, and the doctor found him crying and still unable to speak. In a few hours he could say a few words, but incoherently. The next day he could talk, but not quite freely. There was no paralysis, no disturbance of vision, and no headache. Within three or four days he was quite well, and could talk perfectly. The blood pressure was found to be 212 mm., and the attack was regarded as possibly a slight haemorrhage. I saw him on May 18th, 1911, nearly fifteen months after the attack. Very well, except that he has become very nervous and apprehensive, he has given up work, and has wandered about, and has been under the care of a great many doctors.

On examination he was a very healthy-looking man of good colour and good physique. The radial arteries, with the blood current flowing, could easily be rolled under the fingers. The pulse was recurrent, with practically no difference in the fulness of pulsation beyond the point where the artery was compressed. In a section of the emptied radial, no arterial wall could be differentiated with the finger. It was the same with the temporals and the brachials. With the blood current flowing they could be rolled under the fingers. Emptied they were not palpable. Neither the brachials nor the femorals were sclerotic. The retinal arteries looked prominent. The apex beat was inside the nipple line. There was no evidence of enlargement of the heart; the second aortic sound was ringing. The blood pressure was 220 mm. in spite of the fact that he had been taking for more than a year nitrites and potassium iodide. He has had no other cerebral attack. His general condition was very good, but he was morbidly apprehensive about his condition.

A very different picture was presented by Mr. ———, aged sixty-two, seen June 6th, 1911; a man who had worked hard in many parts of the world, but had not been a heavy drinker. A wiry, tough-fibred man, he had always kept himself in very good condition, but had used tobacco to excess. One afternoon, just a year ago, while waiting for tea, he went out to say something to the gardener, and to his surprise found it impossible. He did not feel giddy or dazed, and five minutes later he could speak quite well. He returned to the house, and about an hour afterwards some people came in, and to his surprise he could not say how-do-you-do, could only nod and give a grimace. He could see the people were very much upset, and he was mortified to feel that perhaps they thought he had been drinking. He was greatly embarrassed as he could not say a word. The gentlemen urged him to see a doctor at once.
He walked out to the garden gate with them but could not say good-bye. He then went across the road to the doctor, but could tell him nothing. He had no headache and he felt quite clear in his mind. In the course of ten to fifteen minutes he began to say a few words, though not quite clearly; in a day or two he could say everything. He remained in bed for a couple of days on low diet. His blood pressure was found to be 200 mm., and for the first time it was found that his arteries were sclerosed. He was a healthy-looking man of good colour. He had lost more than twenty-five pounds in weight, and had been very much worried about the high blood pressure. There was an extreme degree of arterio-sclerosis. Brachials, radials, and ulnars were visible in their entire course, with forcible pulsation. The pulse was recurrent; the radial wall, very much thickened. There was not much difference in the sensation given to the finger between the vessel full and empty. Blood pressure, 130 mm. He had worried incessantly about the high blood pressure and had become greatly depressed. This is not the first instance in which I have known worry and loss in weight to be the most effective means of lowering high tension.

My introduction to this condition occurred under peculiar circumstances: As a young man in Montreal there were two doors I never passed,—47 and 49 Union Avenue; going up I called on Dr. Palmer Howard, and if he was not in or was engaged I called on Dr. George Ross; going down, the reverse. Any growth in virtue as a practical clinician I owe to an intimate association with these two men, in whom were combined in rare measure enthusiasm and clear vision. One morning I had a shock, the first of the kind I had ever felt—I realized that my dear friend George Ross was seriously ill. He had always seemed well and strong, though one hot day, in 1878, at the old Savile Club in London, he had an attack of shortness of breath. This day he told a strange story: he had been awakened by the night bell, and, attempting to put out his right hand to get the match-box, he found he had lost power in it. With his left hand he struck a match and rang the bell. When the servant came he could not speak. He realized perfectly what had happened—that he had had a stroke; but to his surprise in a few hours power had returned to his arm, and he could speak, but not quite clearly. When I saw him he was quite himself—no trace of paralysis, and the speech was clear. Arteries like whip-cord—apex beat out—the usual story that we now know so well. This was the first of a series of transient attacks of aphasia, monoplegia, and hemiplegia extending over four or five years, with intervals of good health during
which he lectured and carried on his practice. Once, on his return from Europe with Dr. Roddick and Dr. Alloway, he had an attack of partial paraplegia and had to be helped off the steamer, but it disappeared in the course of a couple of days.

These not uncommon features of arterio-sclerosis have had an abiding interest ever since. In the first edition of my textbook, 1892, I mentioned that: "transient hemiplegia, monoplegia, or aphasia may occur in advanced arterio-sclerosis. Recovery may be perfect. It is difficult to say upon what these attacks depend. Spasm of the arteries has been suggested, but the condition of the smaller arteries is not very favourable to this view. Peabody has recently called attention to these cases, which are more common than indicated in the literature." The subject had been brought before the Association of American Physicians by Dr. George Peabody at our meeting in 1891, in a very thorough study of the relation of arterial and visceral changes (Transactions of the Association of American Physicians, Vol. VI, p. 170). In one of his cases a man aged fifty-six, with well-marked arterio-sclerosis had an attack of transient hemiplegia without loss of consciousness. Then, in the course of ten days, he had four or five attacks in which he lost the power of speech, and had incomplete paralysis of the right side. He died in a very severe attack in which he had complete right hemiplegia with unconsciousness. Extensive arterio-sclerosis was found in the cerebral vessels, but there was no local lesion, no areas of special oedema, or any foci of haemorrhage or softening. So far as I know Dr. Peabody was the first to offer a reasonable explanation of the condition:

"It seemed to me that there might perhaps have been a spasm of the muscular coat of the middle cerebral artery, or of several of its branches; which, in addition to the encroachment upon its lumen, produced by the new growth, was sufficient to cut off blood supply to the parts to which it was distributed; that this had occurred several times, causing each time temporary ischaemia of important brain centres; and that in the final attack it had lasted long enough to produce death, but that it was not complete enough, or of long enough duration, to cause softening."

Peabody urged that as spasm could be seen in the retinal vessels, with transient loss of vision, the same very probably occurred in local vascular areas in the brain causing ischaemia and loss of function. No one has stated the case more clearly, and I am glad to refer to this important, initial bit of work which has not received
recognize except in my text-book. Since then I have seen a score or more cases, which fall into three categories: (a) Healthy individuals with high blood pressure, but without signs of arterial disease. The first case mentioned in this paper had no obvious sclerosis of the palpable or visible arteries. I have seen only two other patients in which hyperpiesis existed alone—one a man aged fifty-one, who had numbness of the left side and hemianopia, which passed away in the course of a day; the other a young man aged thirty-one, who had not had syphilis but who had high pressure and angina and in several attacks loss of power in the left hand with numbness. (b) Patients with well-marked arterio-sclerosis, in whom the cerebral attacks have come on without warning, sometimes as the signal symptom. A majority of my cases come in this group. (c) In advanced sclerosis with cerebral changes, manifested by progressive mental and muscular weakness, all possible types of these transient seizures, including convulsions, may occur. The attacks are most frequent in the aged, but men in the fifth and sixth decades are also affected.

The symptoms are extraordinarily varied, but tend in individual cases to repeat themselves in the attacks. Transient aphasia is one of the most common. The account given by the two patients whose cases are here reported is singularly accurate—inability to talk, consciousness of it, no paralysis, emotional disturbance, and, within a few hours, complete recovery. One patient had at least twenty attacks, all of very much the same type. Loss of the power to write and hemianopia may be present.

Sensory disturbances rarely occur alone, but one patient had day-long attacks of numbness of the face and right hand with loss of the finer movements of the fingers. Paræsthesiae may exist with the aphasia.

Motor paralysis is the most common symptom, and may be hemiplegia, or only the face and hand or arm may be involved. The paralysis, rarely complete, has a transient character, which with the recurrences gives it a peculiar stamp. Complete recovery is of course seen in monoplegias and hemiplegias of organic origin, but not in a few hours or in a day. I have had a letter in the evening from a man who at 9 a.m. could not button his shirt collar. In one instance paraplegia of brief duration occurred.

The mental features are interesting. Confusion of thought is common and emotional disturbances, which are very natural under the circumstances. The transient attacks of mental aberration—forgetfulness or slight delirium—seen sometimes in arterio-sclerosis,
may be the psychical counterparts of the motor attacks, and cases have been reported by Edgeworth and William Russell. Loss of consciousness has not been common in my experience. I saw a patient in Washington whose attacks were always ushered in by a short cry, followed by fainting, and on recovery the right arm and face were paretic and there was transient loss of the power of speech. He had a pulse of sixty, and the question of Stokes-Adams disease was considered.

The mode of origin of these cerebral crises in arterio-sclerosis has been much discussed of late years by William Russell in his work on Arterial Hypertonous, etc., (1907); by Edgeworth,¹ Parker,² Langwill³ and Heard,⁴ Allan⁵, and others.

An interesting discussion in the British Medical Journal, 1909, II, followed a paper by William Russell on "Intermittent Closing of the Cerebral Arteries." When Peabody brought forward the view that in these cases a transient arterial spasm occurred, I was doubtful how far this was possible in sclerotic vessels; but I have since come round to his view, and I do not think any other explanation is more plausible than that these attacks represent vascular crises.

We have plenty of evidence that arteries may pass into a state of spasm with obliteration of the lumen and loss of function in the parts supplied. In the peripheral arteries in Raynaud's disease we can sometimes feel the spastic, cord-like vessel; in the retina we can sometimes see the arteries contracted. Both in Raynaud's disease and in the remarkable thrombo-angitis described by Buerger the obliteration may persist until necrosis occurs, but in many instances it is only transient and the circulation is restored. A case of Raynaud's disease,⁶ with recurring attacks of aphasia, hemiplegia, and loss of consciousness, some occurring coincidently with the local asphyxia and necrosis, convinced me that intermittent closing of the cerebral vessels could occur, and the transient nature of the attacks with the complete recovery seems to offer no other explanation so satisfactory. And we know now that there are neither anatomical nor physiological objections to this view as applied to the cerebral arteries.

Transient paralyses in uræmia may be due to oedema, as suggested by Traube (Gesammelte Beiträge, Bd. 2, p. 551); but the condition is rarely transient and is more often a terminal event. It is possible that there are cerebro-spinal manifestations in angio-neurotic oedema, as in the extraordinary case which I reported⁷ of a physician aged twenty-nine, who had right hemiplegia and aphasia at the age of
nine, and, within a year, five or six attacks of transient hemiplegia, subsequently migraine, and well-marked attacks of angio-neurotic oedema. Howland has recorded a case of this disease with spinal symptoms. The association of migraine with cerebral symptoms is well known, and Mitchell Clarke has reported a familial form with which hemiplegia occurred in three generations.

The diagnosis, usually easy, is based on the existing conditions of high tension or sclerosis or both, the slight and transient character of the attacks, and the recurrences. Slight paralyses due to haemorrhage or softening rarely pass away so quickly, and it may be weeks before a patient speaks clearly or uses the hand freely. Numbness, tingling, and slight weakness of one side with headache may be precursors of a "stroke" in which case the symptoms are not transient but progressive. In sclerosis of the cerebral arteries small foci of softening are not rare and some of these may produce symptoms. An artist friend motoring in the neighbourhood of Oxford felt so badly in his head that his wife insisted upon coming at once to my house. He was a bit dazed and could not sit at luncheon, but there did not seem very much the matter. He said, however, that he felt "queer in his head," and could not see properly. I could find nothing wrong with the retinae but I did not take the fields. Two days later he consulted Mr. Lawford who found a quadrantic hemianopia, which has never cleared up. Here no doubt was a definite lesion.

The prognosis is largely that of the sclerosis. Patients may live for years and be very comfortable in the intervals. While writing this I had a visit from my old friend, Dr. Litchfield, of Pittsburgh, who happened to mention the case of a Mr. L., whom I had seen with him eleven years ago with transient attacks of what Dr. L. called "mutism," often associated with numbness and tingling on the right side. The patient had arterio-sclerosis, and I remember we discussed the possibility of the condition being due to angiospasm. He has had in all ten or twelve of such attacks: they pass off in a few days; associated with the feeling of fulness and headache. Once or twice he has had a transient diplopia. In the intervals he is pretty well, though it is difficult to keep down his blood pressure. An interesting point of which Dr. Litchfield reminded me was that this man's father had had similar attacks, beginning when he was a comparatively young man, and lasting until he was over seventy. Of considerable moment, as illustrating the necessity for a more widespread recognition of this condition, is the fact that Mr. L. a few years ago consulted a well-known heart
specialist, who said that he had chronic meningitis, gave him bromides, and his friends a hopeless prognosis.

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THE PATHOLOGICAL INSTITUTE OF A GENERAL HOSPITAL.

By Sir William Osler, Bart., M.D., F.R.S.,
Regius Professor of Medicine, Oxford.

(Reprinted from the "Glasgow Medical Journal," November, 1911.)
THE PATHOLOGICAL INSTITUTE OF A GENERAL HOSPITAL.
THE PATHOLOGICAL INSTITUTE OF A GENERAL HOSPITAL.¹

BY SIR WILLIAM OSLER, BART., M.D., F.R.S.,
Regius Professor of Medicine, Oxford.

I wonder if you appreciate in Glasgow the delightful memories that crowd the mind of a man familiar with the story of medicine in Great Britain, when the name of your Royal Infirmary is mentioned—memories of men who, by their life and doctrine, have set forth not alone the very best the profession has had to offer, but the very best that man has ever offered to his fellowmen. I am fully aware of the presence of those who will regard my memories as illustrating how slight and imperfect my knowledge is of this old foundation; but I have an abiding faith in the general ignorance of an audience, and I know that I know many things that many of you know that you should know, but don't know, about the men who have made the Royal Infirmary famous. In any case I am going to use this knowledge, imperfect as it may be, as a setting for the main theme of my address—the place of the pathological institute in a general hospital.

I am glad you have adopted the name institute—for the first time, I believe, in Scotland. Plato made the curious remark that while it was a slow and arduous process to get men to change their ideas, it was an easy matter to get them to accept new names, and under these gradually to bring about wished-for changes. I am sure that the name indicates that you feel the infirmary should bear its share with the University in advancing scientific medicine; for an institute is something more than a dead-house, and very much more than an ordinary pathological laboratory. It is the cerebrum of the infirmary, the place where the thinking

¹ An address at the opening of the new Pathological Institute of the Royal Infirmary, Glasgow, 4th October, 1911.
is done, where ideas are nurtured, where men dream dreams, and thoughts are materialised into researches upon the one great problem that confronts the profession in each generation—*the nature of disease*.

Ask the editors of the hundreds of archives, journals, annals, magazines, bulletins, Comptes Rendus, Centralblätter, Zeitschriften, devoted to the service of medicine, what is their chief aim—their *raison d’être*? Ask the professors of all the medical schools of Europe and America, the directors of all the laboratories, what is their chief problem? Go back in history and ask Hippocrates, Galen, and all the great students of medicine down to Cullen and the Hunters, what one thing influenced their lifework? Ask the surgeon why, a few minutes ago, he removed an appendix in the operating-room near by, or the doctor why he has just given a dose of antitoxin to an infant with diphtheria? From all come the one monotonous answer—*the nature of the disease*. There is not a page of this month’s number of the *Glasgow Medical Journal* but reflects this all-powerful, all-pervading influence: the whole life of the profession, whether moving in the units or expressed in its great institutions, is controlled to-day, as it ever has been controlled, by what we think of the nature of disease. Why is a right judgment on this one point the aim of medical education and of research—the be-all and the end-all of our efforts? Because upon correct knowledge depends the possibility of the control of disease, and upon our views of its nature the measures for its prevention or cure.

This institute itself is a witness to the wonderful transformation in our knowledge of the nature of disease, in which this old infirmary has played a splendid part. Let me indicate briefly the steps in this transformation. Hippocrates looked at patients with keen eyes and a clear brain, and, as a product of his period, when the nature of nature began to be recognised, he could not but deduce from his observations the naturalness of disease—that it was not an entity apart from man, but a part of man himself. And this was the great contribution of the Greeks, who first taught us to look with the seeing eye at disease. Observation plus thinking has given us the vast stores of knowledge we now possess of the structure of the bodies of living creatures in health and disease. There have been two inherent difficulties—to get men to see straight and to get men to think clearly: but in spite of the frailty of the instrument, the method has been one of the most powerful ever placed in the hands of
man. It gave us Vesalius and the new anatomy, Newton and a new universe, Morgagni and the new morbid anatomy, Laennec and the new medicine, Virchow and the new pathology, Darwin and a new outlook for man on the world.

Let me give you an illustration of the application of this method and of its limitation. The ancients knew aneurysm of the external vessels, and if I had my choice in time of witnessing an operation I would ask to see Antyllus in the third century tackle a femoral aneurysm. Vesalius and Ferrellius, Lancisi, John and William Hunter, Scarpa, and some hundreds of keen observers have taught us the structure of aneurysm and the means by which it may be recognised. Take the work of Allan Burns on *The Heart*, published in 1809, one of the best contributions to clinical medicine of the Glasgow school, and by the best, the very best member of one of your best families, distinguished alike in science and commerce. Read his chapters on the arteries, and you will see how far a shrewd observer could go in the recognition of anatomical changes and clinical features. And no Scotsman ever used his favourite instrument to better effect than did Allan Burns in the clear logical reasoning with which he lays down what is now known as the intermittent claudication theory of angina pectoris. Or take a work of another famous Glasgow teacher, whom I was proud to know as a friend, Professor Gairdner, whose *Clinical Medicine* is a storehouse of valuable facts. Turn to the chapter on aneurysm, and see how the art of observation may be made to lighten the dark corners of disease, in the wards and in the dead-house. These two men may be taken as types of the workers who, between Morgagni and Virchow, revealed to us the seats of disease. You remember the title of the great Morgagni’s work, *De sedibus et causis morborum*, and while he did illumine the structure of disease and correlate the symptoms in life with the appearances after death, the work had the limitation expressed by the sub-title, *per anatomem indagatis*. This was the *organon* with which was built the broad and deep foundations of clinical medicine and morbid anatomy. Observation alone could give a complete knowledge *de sedibus*, but never *de causis morborum*. Other men with other minds and other methods had to give us this through the long and painful experience of many centuries. Seeing and thinking have done much for human progress; in the sphere of mind and morals everything, and could the world have been saved by armchair philosophy, the Greeks would
have done it; but only a novum organon could do this, the powerful possibilities of which were only revealed when man began to search out the secrets of nature by way of experiment, to use the words of Harvey.

The ancients thought as clearly as we do, had greater skill in the arts and in architecture, but they had never learned the use of the great instrument which has given man control over nature—experiment. Dumas, in a letter to Pasteur, made a clear statement of the two methods: "The art of observation and that of experiment are very distinct. In the first case, the fact may either proceed from logical reasons or be mere good fortune; it is sufficient to have some penetration and the sense of truth in order to profit by it. But the art of experimentation leads from the first to the last link of the chain, without hesitation and without a blank, making successive use of Reason, which suggests an alternative, and of Experience, which decides on it, until, starting from a faint glimmer, the full blaze of light is reached." There is not one of us to-day who has not benefited by the countless thousands of experiments which have made modern life what it is; the physical sciences, with the practical application of which you have had so much to do, have re-made Glasgow as they have re-made the world. And there has not been a single advance of the first importance which is not a fruit of this scientific modernism. This institute which we open to-day is a manifestation of the new spirit. We did not get very far in our knowledge of the workings of the animal body in health and in disease until we began to use experiment. A few of the old Greeks appreciated its value, Galen in particular, but it was not until the Renaissance, until the advent of Galileo and of Sanctorius, that men realised how powerful it might be. Harvey made use of it in a golden discovery, and his monograph, Exercitatio anatomica de motu cordis, is the first great product of the experimental method applied to medicine. Thousands of men with keen eyes had watched the heart beat, had seen arteries spurt red blood, had seen the black blood flow from the veins, and they had thought and thought and thought of how the heart beat and how the blood flowed, but all in vain until, in a few simple experiments, the problem of its circulation was demonstrated. Since the days of Harvey we have gained extraordinary insight into the processes of the animal body, and in almost every department by the use of his methods. Take, for example, the work of one of your
infirmary surgeons, in which I was always deeply interested. From the days of Cain man had seen that “ganz besonderer Saft,” the blood clot, and from Galen on speculation had been rife as to its cause. Much had been demonstrated by John Hunter, and much more by Hewson, but it was not until 1831 that the problem was approached in a productive way by Andrew Buchanan, a young surgeon, for long years connected with this infirmary, and for thirty-seven years Professor of the Institutes of Medicine in the University. By a series of ingenious experiments he showed that the act of coagulation was not the effect of any spontaneous property existing in the fibrin, but that it only occurred under the influence of suitable re-agents, and he compared it very properly with the action of rennet in effecting the coagulation of milk. We have travelled far from Buchanan’s “washed blood-clot,” with which we older teachers of physiology used to work and do our class experiments, but the fundamental facts as demonstrated by him remain, fully substantiated by the subsequent work of Schmidt, Hammarstini, and others. Buried in the new terminology you students may find it hard to recognise the merit of the old Glasgow physiologist and surgeon, and I doubt if he himself could make very much of the following definition of the nature of coagulation which I quote from a recently issued work on Physiology:—“The activator thrombokinase, in the presence of free calcium ions, activates the thrombogen, or prothrombin, with the result that the active thrombin-thrombosin, fibrin enzyme, is formed. Fibrin enzyme so produced acts upon soluble fibrinogen and converts it into insoluble fibrin.”

What, you may ask, has all this to do with the nature of disease? The new method has at last put in our hands a means to obtain certain knowledge of the nature of some of the most important of diseases; and what is more important, the methods for their effective control. Morgagni to-day would make the title of his book De sedibus et causis morborum per anatomien et experimentum indagatis. At the middle of the last century we did not know much more of the actual causes of the great scourges of the race—the plagues, the fever, and the pestilences—than did the Greeks. The facts that fevers were catching, that epidemics spread, that infection could remain attached to particles of clothing, &c., all gave support to the view that the actual cause was something alive—a contagium vivum. This was really a very old view, the germs of which may be found in the
Fathers, but which was first clearly expressed, so far as I know, by Fracastorius, a Veronese physician in the sixteenth century, who spoke of the seeds of contagion passing from one person to another; and he first drew a parallel between the processes of contagion and the fermentation of wine. This was more than one hundred years before Kircher, Leeuwenhoek, and others began to use the microscope and to see animalcule, &c., in water, and so gave a basis for the "infinitely little" view of the nature of disease germs.

It was a shrewd, but very characteristic, remark of Robert Boyle that he who could discover the nature of ferments would be able to explain the nature of certain diseases. In August, 1857, a young teacher of the University of Lille read a paper at the Scientific Society on "Sour Milk Fermentation." Louis Pasteur was a chemist, accustomed to accurate observation and careful experiment. At Lille the opportunity offered to study the problems of fermentation in the making of beetroot alcohol, and this led to a comprehensive study of the whole question, from which he concluded that the transformation of sugar into alcohol, and carbonic acid, the souring of milk—in fact, all the processes which we know as fermentation—are co-relevant to a phenomenon of life. A parallel between fermentation and an acute infection had been drawn centuries before, but it came practically home to Pasteur's mind with the suggestion, in his own words, "what would be most desirable would be to push those studies far enough to prepare the road for a serious research into the origin of various diseases." If the changes in fermentation are due to minute living organisms, why should not the same tiny creatures make the changes which occur in the body in putrid or suppurative diseases? And the great Frenchman took an early opportunity that offered to test the truth of this suggestion, and the unravelling of the nature of the silkworm disease was the first great victory of the experimental method applied to a biological problem of disease.

Judging from the history of science, it could scarcely have been expected that these brilliant studies of Pasteur would have borne fruit so soon; but the spirit of research was abroad, and there were keen men everywhere trying to solve the ever present problem of the nature of disease.

You had the singular good fortune in 1860 to appoint to your Chair of Surgery a young Englishman, named Joseph Lister, already with, for his age, a reputation as a strong surgeon; but he was much more than this, he was a trained experimental physiologist and a good microscopist. His
early important studies were on inflammation, on various disturbances of the circulation, and on the coagulation of the blood. In this infirmary, as early as 1865, his attention was turned to the question which, above all others, disturbed the practical surgeon—how to prevent the appalling mortality in surgical cases after wounds and operations. It had long been recognised that now and again a wound healed without the formation of pus, that is, without suppuration, but both spontaneous and operative wounds were almost invariably associated with that change; and, moreover, they frequently became putrid (as it was then called—infected, as we should say), the general system became involved, and the patient died of blood poisoning. So common was this, particularly in old, ill-equipped hospitals, that many surgeons feared to operate, and the general mortality in all surgical cases was very high.

Believing that from outside the germs came which caused the decomposition of wound, just as from the atmosphere the sugar solution got the germs which caused the fermentation, Professor Lister applied the principles of Pasteur's experiments to their treatment. It may be well here to quote from his original paper in the *Lancet*, 1867:—“Turning now to the question how the atmosphere produces decomposition of organic substances, we find that a flood of light has been thrown upon this most important subject by the philosophic researches of M. Pasteur, who has demonstrated by thoroughly convincing evidence that it is not to its oxygen or to any of its gaseous constituents that the air owes this property, but to minute particles suspended in it, which are the germs of various low forms of life, long since revealed by the microscope, and regarded as merely accidental concomitants of putrescence, but now shown by Pasteur to be its essential cause, resolving the now complex compounds into substances of simpler chemical constitution, just as the yeast plant converts sugar into alcohol and carbonic acid.”

From these beginnings modern surgery took its rise, and the whole subject of wound infection, not only in relation to surgical diseases, but to childbed fever, forms one of the most brilliant chapters in the history of preventive medicine. Brilliant researches, helpful to our fellows, and a source of pride to your city, will come from the University laboratories and the hospitals, but it is difficult to imagine the possibility of another such revolution as that which Joseph Lister effected from the wards of the old infirmary—a revolution so far-reaching that we, blessed still by the presence of the
Master, while keenly appreciating can scarcely realise its true greatness.

The institute, an integral part of the infirmary, the director and staff co-ordinate with the physicians and surgeons and their staffs, the other departments should be dovetailed in such a way that every member has an interest in its work. I have often remarked that the secret of the success of the Johns Hopkins Hospital lay in the dominating influence of the pathological department. Everything depends upon the organisation. With two modern hospitals the problem of the relation of their pathological departments to the pathological department of the University has been settled in the only way. In the interest of both institutions the union has been made organic, and the professors of the subject at the University have the same relation to the pathological laboratories of the hospital as the professors of surgery have to their wards in the infirmaries. Only with this type of organisation can a great institute as a university unit fulfil its threefold mission, to the students, to the staff, and to the public. Here, after passing the Vesalian stage in anatomy and the Harveian in physiology, the student learns with Morgagni and Laennec the structural changes wrought by disease. Here he recognises the correlation between the symptoms in life and the post-mortem appearances, which is the bed-rock in the art of diagnosis. And here he reaches the stage in which Virchow and Koch teach the true nature of the intimate processes of disease, de causis as well as de sedibus morborum. All this before in the final stage he sees in the wards the marvellous benefits which have followed the practical application by Pasteur and Lister of the methods of science. Just as the embryo passes through life of lower grade, before resulting in the thinking man—the ontogeny reproducing the phylogeny—so the career of the medical student follows the evolution of the marvellous knowledge that has made our profession the most helpful of all to humanity. And do let him feel that he is at home in the institute, a part of it in work and in teaching. Let me urge you not to neglect the Morgagni side, not that it is likely in a thoroughly practical school like this; but so deep may be the absorption in the problems of disease that the virtue of teaching, the labour of drilling the students in the technique of post-mortems, the patient line-upon-line, precept-upon-precept method becomes a burden, and the priceless lessons of the dead-house are not enforced by the voice of a master. Only in one way lies redemption for the director
of any institute or laboratory, he must have associates who know more about certain subjects than he does himself. An Admirable Crichton in these days is a quack, and in the art of delegation, in the subdivision of labour, in specialisation among his subordinates, the director will find safety. The patient demonstrator who spends two hours with a group of students at a section has a place of equal importance with the man who is chasing the secret of anaphylaxis. In the hurly-burly of to-day, when the competition is so keen, and there are so many seeking the bubble reputation at the eye-piece and the test-tube, it is well for young men to remember that no bubble is so iridescent or floats longer than that blown by the successful teacher. A man who is not fond of students and who does not suffer their foibles gladly misses the greatest zest in life; and the teacher who wraps himself in the cloak of his researches, and lives apart from the bright spirits of the coming generation, is very apt to find his garment the shirt of Nessus. Encourage the students to help in the teaching, and arrange the time of sections not for your own convenience, but for the students and staff. I had a practice of making the clinical clerk tell the story of the case, not read an abstract, but speak it out and tell its difficulties and the diagnosis, right or wrong. It was good for us all, the teacher and the taught, and we met on the same levels as seekers for truth. How far should students be encouraged to do original work? As much as possible, though in the present congested state of the curriculum the possibility is not a very big one. A keen man who has had a good chemical training may find time to work out a small problem in metabolism suggested by one of his cases in the ward. A student in physics may have ideas on blood-pressure, an advanced student in physiology may wish to test the teaching of the laboratory in a problem suggested by a cardiac case. A laboratory without a few undergraduate research students is scarcely fulfilling its mission. There are difficulties, I know, but let them be on the side of the student, not on the part of the institute.

I said the institute of pathology was the cerebrum of the infirmary, the thought-centre for the staff; but let it be remembered that the institute exists for the infirmary, not vice versa. In many ways it may be helpful to every man working in the wards and in the out-patient departments. The best doctor, like the successful general, is the one who makes the fewest mistakes. In the dead-house, by the contemplation of the mistakes of nature and of our
own, we glean a wisdom which enables us to correct the one and to avoid the other. No man becomes a sound physician or a good surgeon who does not frequent the dead-house. Fortunately the infirmary has splendid traditions of the co-operation between the laboratory and the wards. At my first visit to Glasgow in 1872 I recall with pleasure the acquaintance I then made with Joseph Coats, whom I saw at work in the dead-house, and whose studies in pathology brought so much credit to the Glasgow school. And Professor Macewen's masterly researches upon Infective Diseases of the Brain and Spinal Cord, a magnum opus worthy of a successor of Lister, tells on every page of the benefits a surgeon may derive from an intimate association with pathological laboratory. Every one of the young men on the staff should be workers in the institute, each with his place, each with his problem. Even the older men when not over-burdened with practice will find mental refreshment and stimulation in a few hours of laboratory work. It is a sad day when the world is so much with the clinician that he cannot spend part of it in the pathological or the clinical laboratory. Here comes the question of the relation which should exist between these two important parts of a hospital. Often they may be combined, but a special advantage of a separate clinical laboratory is that each student in his senior years can have his own place to work, his own microscope and apparatus, a place at which he can keep up the laboratory habit acquired in the medical school. Then, too, we must look forward in this country to the organisation of our clinics on University lines, when the professors are not simply attending physicians but directors of hospital units which have the same relation to the University as the other scientific departments, with subdivisions in the clinical laboratory of bio-chemistry, cardo-respiratory physics, and bacteriology, each under the guidance of a skilled assistant. When this can be carried out the pathological institute does not suffice, and other arrangements must be made. Here, too, the practical man comes for inspiration, for new ideas, and here he finds the touchstone by which he can tell the true in the new. That is to say, if he has sense. A very practical man, dependent for his large clientèle on a winning smile and a smooth tongue, reported to a medical society a new tip which, he said, was derived from a visit to a well-known pathological laboratory. He had seen brains hardened in bichromate of potash and chloric acid. Happy
idea! He began the treatment of cases of softening of the brain with these drugs with, he assured us, the most excellent results!

Oliver Wendell Holmes remarked that knowledge and timber should not be used until they are seasoned, and here should be found for the staff and for all members of the profession in the city and district that seasoned knowledge which alone can make us wise unto the salvation of our fellowmen.

And, lastly, this institute exists for the benefit of the public. There is not a patient in the wards who will not be helped by the work done here. Nowadays laboratory methods of treatment and diagnosis are more and more in vogue. This will be the routine of service, but the larger public that pays the piper has the right to call the tune; and the demand which they make, and with just right, is that the resources of the institute should be requisitioned in the fight which science is making against unnecessary disease and untimely death. From laboratories have come not alone the war cries of modern medicine, but the chief weapons against the acute infections. The incentive, the intense conviction of the necessity of the fight, and of its hopefulness, has come from the men who realised that the general infections, whether endemic in cities or widespread epidemics, were preventible could we but get a knowledge of their causes, could we but know their nature. Even before this knowledge was complete we had recognised the association of disease with dirt, and of fevers with overcrowding and with poverty. And Glasgow was early in the field. The sanitary story of your city in the last half century is one of which you may be justly proud. Under the intelligent direction of Professor Gairdner, Dr. Russell, and of your present efficient health officer, Dr. Chalmers, from the worst, or one of the very worst, you have become the best, or one of the very best. To wipe out typhus, to have made typhoid a last ditcher, to have cut in half the mortality from tuberculosis, are among the peace victories in which you citizens have shared. Given to pessimism, the Briton loves to look on the dark side of things. There is no such medicine for the malady as a study of the health records of our great towns—a story of marvellous progress, better housing, better feeding, better drinking, better health, and, as a consequence, better citizens.

Two problems remain. First, to make effective the knowledge we now possess, and this is largely a question of
intelligent organisation. When the public awaken, what has been done for typhus and typhoid will be done for tuberculosis, malaria and plague, as well as for a host of minor maladies, the causes of which we know.

But in a vast field we need new knowledge and seasoned knowledge, and this the other great problem directly concerns the institute. Four riddles of the first rank await solution. Cancer killed in this city, in 1910, 845; in 1909, 34,053 in England and Wales. Literally thousands of workers are struggling to unravel its mystery, and while much has been done, its heart has not been unlocked—the nature of the disease is unknown. The key may be in other hands; and so interlaced are these biological problems, so conditioned by our knowledge of chemistry and physics, that some young Pasteur at Lille or a young Helmholtz at Königsberg may be twirling it in his fingers all unconscious of its use. The exanthems are still with us, still killing thousands, and we await the researches which will reveal the causes of measles, scarlet fever, and small-pox. Perversions of metabolism are every day yielding up their fascinating secrets, but we lack the sure and certain studies that alone can give us control of such common diseases as diabetes, gout, and arthritis.

And, fourthly, we are entering a new chapter in the researches upon the internal secretions, on the functions of those mysterious glands, so insignificant anatomically but so potent in their influence upon growth and nutrition. These and scores of minor problems are to be solved by this generation, and there is much knowledge to be seasoned before it can be used to the best advantage. For example, the whole vaccine problem is being kiln-dried; and the rapid seasoning of Professor Ehrlich's great discovery will give an immense impetus to the study of specifics, torpedo remedies, as Huxley called them, agents that will kill the enemy and spare the host. I told you in illustration of the value of observation the story of aneurysm, how we had known the external from since the days of Galen, and the internal from Vesalius. Twenty years ago we felt we had a very full knowledge of the subject, and even ten years ago had any one suggested that we might some day be able to prevent aneurysm he would have been thought a dreamer. The clear vision of Francis Welch had given an inspiration de causis, but it was not till the demonstration by Schaudrim of the germ of syphilis that we recognised fully its enormous importance in the causation of arterial disease in general,
and aneurysm in particular. And now Ehrlich comes forward with a remedy which, widely and carefully used, should prevent these specific lesions of blood-vessels, and cut in half the incidence of aneurysm in the community.

The most vivid recollections of my boyhood in Canada cluster about the happy spring days when we went off to the bush to make maple sugar—the bright sunny days, the delicious cold nights, the camp fires, the log cabins, and the fascinating work tapping trees, putting in the birch-bark spouts, arranging the troughs, and then going from tree to tree collecting in pails the clear, sweet sap. One memory stands out above all others, the astonishment that so little sugar was left after boiling down so great a cauldron of liquid. And yet the sap was so abundant and so sweet. The workers of my generation in the bush of science have collected a vaster quantity of sap than ever before known; much has already been boiled down, and it is for you of the younger generation while completing the job to tap your own trees. Considering the enormous quantity of sap we have collected, you may feel disappointed at the comparatively small bulk of sugar left after complete boiling, but sweeter or better sugar has never been tasted than that of our making; and among all samples in the market, no brand ranks higher than that from the old Glasgow Royal Infirmary. It is for you in this new infirmary and in this splendid institute to see that the quality is maintained.