An Address

ON THE

RELATION OF ANGINA PECTORIS

AND

ALLIED CONDITIONS TO AN ARTERIO-CARDIAC
REFLEX HAVING ITS ORIGIN IN THE
ABDOMEN.

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INTRODUCTORY.

ANGINA PECTORIS has attracted the attention, and at times rivetted the thought, of many great clinicians during the past 130 years. That it should exercise the same influence over the clinicians of to-day need not, therefore, cause surprise. Gibson¹ of Edinburgh, Oliver² of New-castle-upon-Tyne, and Mackenzie³ of Burnley have all three shown quite recently that they were the victims of the fascination or attractiveness of the subject. It requires no special courage to avow oneself a fellow-victim, however much courage it may require to attempt the elucidation of a subject which has been dealt with by so many able physicians of the present and the past. Were apology necessary for so doing, I would say that I have found the view which I am to submit of much practical value: that, instead of conflicting with past observations, this view interprets them, and that it gives the clue for the disentanglement of the confused mass of opinion on the subject. My views have emerged from the special attention which I have long given to cardiac and vascular disturbances, and more recently from a fuller knowledge of the processes of primary and secondary digestion, of nutrition, and of elimination. My indebtedness to the work of Pawlow,⁴ Herter,⁵ Chittenden,⁶ and others I gratefully acknowledge. The relations between gastro-intestinal conditions and vasculo-cardiac disturbances and the influence of dietetic substances upon the vasculo-cardiac system have recently been more and more impressed upon me, and this communication contains the conclusions at which I have arrived and indicates the path by which I have reached them.

HISTORICAL AND CRITICAL.

Heberden to Trousseau, 1768-1862.

We do not require to go further back in our historical retrospect than the date when the term "angina pectoris" was introduced into medical nomenclature. Those who wish to examine the earliest records of the symptoms called by this name will find the references in several of the older authors, particularly in Parry's monograph.

Angina pectoris was the name given by William Heberden⁷ in the year 1768 to a disorder of the breast, which he thus described: "Those who are affected with it are seized while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away if it were to increase or to continue." "The seat of it and sense of strangling and anxiety with which it is attended may make it not improperly be called angina pectoris."

As regards the nature of the disease to which he gave this name, he says that "the opinion of its being a convulsion of the part affected will readily present itself to any one" (p. 64).

In a later paper, of date 1785, containing the account of the *post-mortem* examination on the historical "unknown," he reports that there was no lesion found; and in his comments on treatment he advocated the use of medicines which "relieve and quiet convulsive motions" (vol. iii, p. 10).

Here at the fountain-head is found the idea of the nervous nature of the clinical phenomena of angina pectoris, a conception of the condition shared by many subsequent writers.

The next step worthy of note was made by Dr. Caleb Hillier Parry,⁸ a physician in Bath, who published in 1799 a monograph entitled *An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris*. This work is characterized by remarkable clinical acumen, and Parry's views, it seems to me, have not always been accurately presented by subsequent writers. Some of the clinical points dealt with by him are to be specially noted. He

points out, for instance, that the first symptom is "an uneasy sensation (described variously as a stricture, an anxiety, or a pain) about the middle of the sternum, across the left breast, and in certain stages of the disorder usually stretching into the left arm" (p. 42), also that the pain occurs in paroxysms, and that early seizures seldom occurred without apparent cause (p. 43). He also insists, "we shall always find it (the pulse) become more or less feeble according to the violence of the paroxysm" (p. 45). Here we have an early recognition of great differences in the degree of severity in the leading symptom. The pathology of the condition was held by him, and by his friend Jenner, to be disease of the coronary arteries interfering with the blood supply to the heart. His induction was that a heart with such coronary arteries could not bear any extra strain put upon it, that if subjected to extra strain it became surcharged with blood, that its action became enfeebled, and might cease. The extra strain could be induced by mental emotion, by physical effort, and by gastro-intestinal conditions. As the heart condition advanced, even slighter causes than these might produce paroxysms. Thus heart failure and its organic cause formed the central, and to him the essential, factors, in determining the gravity of the condition. According to Cullen's nosology this was assuredly a *syncope*. "The motion of the heart diminished, or even for some time ceasing. *Motus cordis imminutus, vel aliquamdiu quiescens*." It differed from an ordinary syncope "only in being preceded by an unusual degree of anxiety or pain in the region of the heart." He therefore termed the disorder *syncope anginosa*. To make the position of this old Bath physician still more clear let me give his own words regarding a patient who had been long subject to attacks of what he considered a "very pure angina pectoris." He accompanied his patient on a walk up hill in order to witness what occurred during the fit. He says "when the paroxysm was thus excited, I could perceive no symptom of disorder in addition to the uneasiness in the breast, except a gradual and most evident diminution in the strength of the pulse" (p. 59).

Allan Burns,⁹ Professor of Anatomy and Surgery in Glasgow, and practising in that city (1809), accepts Parry's view that syncope anginosa originates "from some organic lesion of the nutrient vessels of the heart" (p. 137). He compares a heart with "cartilaginous or ossified" coronary vessels to a limb with a ligature round it called into active exercise. The limb so bound can only work for a very short time; "it soon fails and sinks into a state of quiescence" (p. 138). He holds that "the essence of this complaint consists in a reduction of the action of the heart" (p. 146).

In 1815 Jurine,¹⁰ in an essay to which the Academy of Medicine awarded a medal, combated Jenner's and Parry's view that angina pectoris was due to ossification of the coronary arteries. To him "sans douleur sternale, il n'y a pas d'angine de poitrine" (p. 72), and he argued that the angina was essentially due to an affection of the pulmonary nerves (p. 123).

Laennec,¹¹ in 1819, had found that in several subjects who had laboured under angina pectoris, and in whom there co-existed either hypertrophy or dilatation of the heart, there was no ossification of the coronary arteries (p. 757). According to him, "angina pectoris in a slight or middling degree is extremely common, and exists very frequently in persons who have no organic affection of the heart or large vessels" (p. 756). Desportes (1811) is quoted by Laennec as holding similar views. Angina pectoris was to Laennec a neuralgia.

Forbes¹² (1832), the translator into English of Laennec's great work, divided cases of angina pectoris into organic and functional. The pain to him was the result of "some unknown temporary condition of the nerves of the part," and he believed that "such a morbid condition of the nerves may be produced in a heart in all other respects sound."

Bertin¹³ (1833) also repudiated the view that angina pectoris was due to ossification of the coronary arteries. He had seen the symptoms "accompanied with a multitude of organic affections, either of the heart or of the large vessels, or even of the lungs" (p. 410).

Hope¹⁴ (1839) held that "the nature and variability of the symptoms of angina pectoris confirm the opinion of Laennec that it is a neuralgic affection" (p. 499).

Latham¹⁵ (1846), along with a vivid portrayal of angina pectoris, considered the disease to be "a certain assemblage of symptoms, and not any constant pathological condition belonging to the structure of the organ" (p. 399). The paroxysm was to him "plainly a compound of pain and of something else" (p. 385). It was a "spasm of the heart," as it had been to Heberden. But then note his conception of spasm. He says: "Spasm is always accompanied with pain. And pain and spasm, wherever they are, disable the part which they befall" (p. 385). "In spasms of smaller degree the heart fails to close freely upon the blood and to impel it freely into the arteries. In its spasm of greater degree it fails to project it altogether" (p. 386).

Walshe¹⁶ (1851) looked upon angina pectoris as a "paroxysmal neurosis in which the heart is essentially concerned" (p. 190). He thought genuine angina pectoris to be a very rare disease, while on the other hand he "very frequently met with a form of complaint combining in a minor degree many of the characters of angina." To this "imitation of the true disease" he gave the name of

"pseudo-angina." He thought that this was the explanation of Laennec's notion that the condition was of very frequent occurrence (p. 203-4).

Stokes¹⁷ (1854) agreed with Parry, and says "that the symptoms of angina arise from a temporary increase of weakness in an organ already weakened" (p. 486). He disputes Latham's idea of spasm, and thinks the idea of syncope more in accordance with observed fact.

Trousseau¹⁸ refers to the variety of pathological change which had been described as having been present in the hearts of persons who had suffered from angina pectoris. On the other hand, he has of necessity to admit that angina pectoris, even when most intense, need not be a symptom of an organic lesion. He therefore regarded the organic changes, when they were present, as non-essential, as coincidences, and held that the real affection was a neurosis—in fact a neuralgia. That there might be no ambiguity in the minds of his hearers he again repeated his opinion that it might be only "an idiopathic neuralgia."

THREE VIEWS.

At this point we may draw a line and say that up to it there were, if we exclude Jurine's view—which we may safely do—three views regarding angina pectoris: the first, that it was a convulsion of the heart, or a spasm of it; the second, that it was due to interference with its blood supply; and the third, that it was a neurosis—the word having no subtle or obscure significance, but meaning, simply and plainly, a neuralgia—an idiopathic pain in nerves. Up to this time, it is also important to note that the new term was not limited to a relatively rare malady, characterized by a few dramatic incidents and a suddenly tragic conclusion. Its use had become so extended that Walshe deemed it desirable to introduce the term pseudo-angina, under which to range the cases that, in spite of the correspondence in symptoms, had not the grave significance that the fatal angina of Heberden presented.

ANGINA PECTORIS VASOMOTORIA.

With the year 1867 a new factor was introduced into the discussion on the nature of angina pectoris. Nothnagel¹⁹ in that year wrote a paper which he entitled "Angina Pectoris Vasomotoria." In this condition the symptoms closely resembled those of angina pectoris, and would certainly, I think, have been placed under that designation by some of the distinguished clinicians whose works have been referred to. The essential point in this paper was the recognition of the important clinical fact that in this group of cases narrowing of the arteries preceded and caused the cardiac embarrassment and other symptoms; that, in fact, the anginous symptoms were secondary to the vasomotor ones; and that by relieving the cramp in the vessels the anginous symptoms were prevented coming on.

EARLIER OBSERVATIONS ON BLOOD SUPPLY AND MUSCLE PAIN.

Before Nothnagel, Latham when dealing with the treatment of persons the victims of angina pectoris had not failed to note the guidance to be obtained from a true estimate of the condition of the vascular system. He says:

"The paroxysm is often put off and its severity mitigated and life prolonged by no means more surely than by keeping the vascular system in a just balance between fullness and emptiness, between rich blood and poor blood" (p. 405).

Three years before the date of the publication of Latham's lectures Sir Benjamin C. Brodie's *Lectures on Pathology and Surgery*²⁰ were published (1846). In them there is a lecture on senile gangrene which I doubt if any living surgeon could add to. He notes that the condition might be due to ossification of arteries or to obliteration without ossification. He enlarges upon the prodromal symptoms of gangrene—he had noted the numbness, pain, and loss of muscular power—which may precede its visible manifestation. With the clinical instinct of the great men of his time he applied these observations to interpret the much discussed question as to the nature and causes of the pain in angina. The paroxysm of pain in angina varied in intensity, so it did in gangrene, greatly. The pain in gangrene might become excruciating—the pain of angina might fail to find words adequate to convey a true sense of its anguish. "The disease," he says, "is called neuralgia, which," he adds, "means nothing" (p. 356). A clear thinker, as well as a masterly observer, this surgeon of sixty years ago!

A still earlier recorded observation bearing upon the relation between lessened blood supply and pain must not be omitted were it only for the sake of historical accuracy. At the Académie Royale de Médecine, the *Séance* of October 4th, 1831, Boullay²¹ submitted an observation he had made on a mare of

a condition to which he gave the name of "intermittent claudication." The symptoms came on brusquely after some minutes' exercise, and they were found to be due to obliteration of the femoral arteries. When the animal was in repose the blood was able to reach the limbs by the collaterals, but when it trotted the compressed collaterals did not furnish enough blood and the limbs were seized by *engourdissement* and by severe pain which made the animal fall.

Charcot²² twenty-six years later revived this subject, and drew attention to a corresponding condition as it occurred in the limbs in man as causing severe pain, and as a premonitory sign indicative of a tendency to senile gangrene. So far as I have found, he did not apply his observations to the elucidation of the symptoms of angina pectoris as the English surgeon Brodie had done ten years earlier.

After this, however, intermittent claudication as a cause of painful muscle spasm took its place in medical literature, and has been much used by later writers when discussing the cause of the pain in anginous paroxysms.

Arterial Spasm.

Standing alone, and forming an epoch by itself, is Lauder Brunton's²³ discovery (1867) that dilatation of the peripheral vessels by the inhalation of nitrite of amyl removed or relieved anginous symptoms. From this date vasomotor spasm became a still more definite factor in the consideration and the interpretation of the "syndrome of symptoms" known as angina pectoris.

WHAT IS INCLUDED UNDER "ANGINA PECTORIS"?

What, then, are we to regard as angina pectoris? Are we to confine the use of the term to that type of case, described with such dramatic force by some of the earlier writers that there still lingers round its earlier records a glamour of picturesqueness? Are its symptoms to be bounded by the *dolor pectoris* and the *angor animi* of its early recognition, and is its close to be characterized by the same tragic suddenness? I think not. From the time of Parry the term "angina pectoris" has been applied to symptoms of less gravity, and to conditions where the outlook was not necessarily grave. Let me clinch this statement by quoting the words of Professor W. T. Gairdner,²⁴ who, writing in 1877, says:

We now know that this typical angina is only the culminating form of a group of symptoms, which in their less pronounced, less definitely painful, and more complicated forms, are found to permeate the whole field of cardiac pathology and diagnosis (p. 570).

From his own personal experience he says:

There is often an element of subjective abnormal sensation present in cardiac diseases which, when it is not localized through the coincidence of pain, is a specially indefinable and undescribable sensation (p. 565).

A sensation which can only be called anxiety or cardiac oppression (p. 566).

To this group of symptoms he gave the special title *angina sine dolore*, recognizing thereby what he believed to be "its true diagnostic and pathological significance and its alliance with the painful angina of Heberden." Even the *dolor pectoris* is, then, no longer an essential symptom; and the *angor animi* is whittled down to a sensation of anxiety. This shrewd clinical pathologist saw, however, that there were phenomena which, while different to, were of the same kind as those of the classical angina. This is what Walshe recognized when he used the much-abused term "pseudo-angina." While Gairdner places angina amongst the neuroses he hardly, I think, attaches the same meaning to the word as the earlier writers did; and I am not sure that he does not somewhat grudgingly stick to the term. He was clearly impressed by the possible importance of vasomotor spasm.

Sansom²⁵ on the other hand (1892) thinks it probable that a paroxysm of angina pectoris is an intense nerve storm, provoked by impulses conveyed to certain cerebro-spinal centres (p. 13). He refuses to adopt the theory of cardio-arterial spasm as the indispensable cause of the pain (p. 438).

Osler,²⁶ in his erudite lectures on the subject (1897), after defining angina pectoris, proceeds to say that the term is employed generically to define paroxysmal attacks of pain in the chest, qualifying the varieties by such names as true, false, hysterical, and vasomotor (p. 8).

Douglas Powell,²⁷ in a very admirable article, classifies angina pectoris under (1) a vasomotor group, and (2) angina pectoris gravior, which includes secondary and primary cardiac angina. Forbes,¹² Bramwell,²⁸ Gibson,²⁹ Oliver,³ and others recognize two classes, organic and functional. Huchard³⁵ insists that there is only one angina pectoris, and that it is always due to ischaemia of the heart muscle.

From all this it is quite clear that the term is no longer confined to the severe and fatal malady described by Heberden. It has been extended to a great variety of conditions, characterized by some sense of precordial discomfort, or of discomfort referred directly to the heart, which may or may not be accompanied by pain, and which may or may not be associated with coarse anatomical lesion. It was to cover these lesser manifestations that such terms as pseudo-angina, angina notha, false angina, and functional, as distinct from organic, have been used and applied.

Angina is indeed, to use Osler's words, "a syndrome or symptom group," the clinical pathology of which has to be determined in each separate instance. The term angina pectoris has in fact the same nosological significance as the term dropsy formerly had. It is not a disease; it is a symptom or a group of symptoms, and it is no longer the symptom but the cause behind and determining it which is the problem presented to the clinical pathologist of to-day.

CONSTRUCTIVE AND CRITICAL.

In turning to the constructive side of this communication I ask you to look with me at certain vasomotor phenomena which are to be observed in the body; and I hope to make it clear what I regard to be the relations of those phenomena to the phenomena of angina pectoris.

The presence of arterial spasm has been recognized by many who have written on angina pectoris since the date of Lauder Brunton's epoch-making discovery. Views as to the causation of the spasm have been various, and the relation of the spasm to the symptoms has also been variously interpreted.

AN ARTERIAL ABDOMINAL REFLEX.

I ask you to consider with me certain observations regarding an arterial reflex having its origin in the abdomen, the importance of which I think has not hitherto been fully appreciated. After taking food, and for at least part of the time during which the active processes of digestion are under way, there is an influx of blood to the whole splanchnic area, leading to hyperaemia, such a hyperaemia as considerably reduces the volume of blood in parts outside this area. This drainage into the abdominal vessels is balanced in the general circulation by a systemic arterial contraction. This is evidently a reflex phenomenon originating in the splanchnic system, passing to the vasomotor centre in the medulla, and thence transmitted to the systemic arteries. The existence of this set of phenomena has been carefully worked out by Dr. George Oliver³⁰ in the average healthy person. The arterial contraction is shown by a definite diminution in the diameter of the vessels. It is to be regarded as a physiological hypertonic contraction, which in the average person is accompanied by a rise of blood pressure. However that may be, there is no doubt about arterial narrowing; and I am content to build upon this one certain and positive fact. The fact, then, with which I begin is the change in the systemic arteries during active digestion—whether it be called narrowing, contraction, or hypertonus, signifies not, so long as the word we use expresses our thought. The change will be found to be a reduction in size and an apparent thickening of the arterial wall. The degree of these arterial changes depends upon the kind of meal which has been taken. In the big eater and the wine-drinker the arterial contraction is associated with a rise of blood pressure and a true increase of arterial tension.

Putting extreme cases aside, the general statement will be found to be correct that narrowing of the arteries is present, but in many people, both middle-aged and old, I do not believe that there is any constant rise of blood pressure along with it. To the educated finger and the analytical mind there is no doubt about this, whatever our instruments show. The point is not a purely academic one, for the small and constricted vessel, with its slight systolic expansion, is very commonly regarded as an indication of heart debility and treated as such. In other cases the mistaking of hypertonus for a rise of blood pressure may be of no practical consequence, for the treatment applicable to the one is suitable for the other.

The point here insisted on is the existence of this arrangement for adapting the circulation to the normal processes of ingestion and digestion of food. It is a

normal reflex process. It is a systemic vessel constriction originating in a normal stimulus in the abdomen, and brought about through the anatomical connexions in the nervous system. It is not, therefore, a "neurosis"; nor is the exaggeration of this reflex, prompted by a large meal of stimulating foods, a neurosis. It is necessary to lay emphasis on this distinction. This reflex, like all normal reflexes, is a beneficent arrangement, and could be easily shown to be one of the means of protecting the body from injury. It varies in delicacy in different persons; it exists in all.

Exaggeration of Normal Abdominal Reflex.

Granting the existence of this normal reflex, it will not be questioned that high feeding and alcoholic liquors accentuate it; and that under such circumstances there is marked raising of arterial tension in addition to arterial contraction.

It must, however, be here recognized that in the latter conditions there are two elements present—one, the vascular reflex; and second, the passage into the circulation of substances produced in the processes of digestion, and of substances derived from the alcoholic liquors drunk. It is not possible at present to give these two factors their precise place in bringing about the accentuation of vessel constriction and of stimulating the increased tension. The existence of the reflex is granted, and there is really little room for doubt that certain substances present in the blood can directly stimulate vessel contraction. Contraction so induced is not a neurosis; it may, indeed, be induced when the connexion with nerve centres is destroyed.

Action of Reflex on Sclerosed Arteries.

This recurring arterial contraction, especially if associated with increased arterial tension, is, as I pointed out some time ago,³¹ the efficient cause, and the usual cause, of arterio-sclerosis in the sense in which this term ought to be used.³² At the same time I dwell upon the importance of recognizing that sclerosed vessels are not, because of the change in their walls, incapable of contraction, but that, on the contrary, they are prone to become hypertonic from causes which are commonly regarded as trifling. Professor Pal,³³ as seen in his recently-published work, *Gefässkrisen*, has also observed that sclerosed vessels are particularly sensitive, and specially liable to hypertonic contraction.

Let us bear in mind then, *first*, the existence of this systemic arterial reflex having its origin in the digestive organs or processes. Secondly, the influence of the character of the material introduced into the digestive system in accentuating the reflex. Thirdly, the effect of the reflex in producing arterio-sclerosis. Fourthly, that sclerosed vessels are particularly liable to hypertonic contraction.

RELATION OF REFLEX TO ANGINA PECTORIS.

The relation between the phenomena just dealt with and angina pectoris is not at first sight apparent; and, in order to demonstrate the close connexion between the two, I must now as briefly as possible refer to several illustrative cases which I have recently observed.

CASE I.—*Angina Pectoris Gravior, showing Extreme Sensitiveness of Arterio-cardiac Reflex and its Production by Taking Nourishment.*

The patient was a professional man, aged 67, whom I saw for the first time on September 6th, 1904. He had suffered for years from definite and characteristic attacks of angina pectoris. Lately he had been much worse, and had been confined to bed for weeks. When I first saw him he was having many attacks daily, and circumstances of the most ordinary kind evidently brought them on—such circumstances as the taking of nourishment, the presence of flatulence, slight excitement, and even the changing of his position in bed. The severity of the attacks varied within considerable limits; the more severe, and therefore the more typical, attacks began with pain in the left elbow, which soon extended upwards to the shoulder and then downwards over the precordia. During an attack the hands became cold and painful. The medical attendant, who was watching the patient with the greatest assiduity, informed me that the pulse always indicated the advent of an attack; the radial artery became more definite, firmer, and smaller, the

pulse-rate increased, while the wave became small, and might almost disappear. In some of the worst attacks the patient had become unconscious and was thought to be dying. The treatment followed was the administration of nitrite of amyl and nitroglycerine for the more pronounced of the attacks and the relief afforded by them had always been prompt. Under the influence of one or other the pulse wave became larger, the vessel wall softer, the hands became warm, and the pain passed off. The patient took a remarkably calm and intelligent interest in his attacks, and the foregoing is gleaned from his own and the medical attendant's description. The diet for some time had consisted almost exclusively of raw eggs and weak broth, while sedatives of various kinds had been employed medicinally. The patient had always been a large flesh eater. There was no albumen or sugar in the urine. The medical attendant had been impressed by the fact that the patient more recently almost always had a seizure after being given nourishment, even when that consisted of some raw egg. I had the opportunity of confirming the observations on the change in the pulse during a paroxysm soon after my arrival at the patient's bedside. During the time I spent talking quietly to him the pulse was small, soft, and feeble. I examined the heart without causing any discomfort. The sounds were so faint that they were occasionally inaudible, although the chest wall was thin. I next proceeded to examine the abdomen, and had my hand placed quite lightly on the epigastrium when the patient told us that an attack was coming on. I at once removed my hand. He took his attack with remarkable composure, and it was evidently not accompanied by the distressing sensations which so frequently accompany anginal seizures. Pain began in the left elbow, which, as has already been noted, was always the premonitory symptom of the more severe seizures, and extended to the shoulder and then to the precordia. The face became pale. The radial artery became quite abruptly hypertonic, that is, it became firmer and smaller, the rate rose to 100 and 110, and the tension was sensibly raised. He was at once given a tabloid of nitroglycerine, and in a very short time the artery relaxed and the pulse became large, strong, and rather thumping, the pallor of the face was exchanged for a ruddy tint, and the pain ceased. The thumping soon passed off, and the pulse resumed the small feeble character it had presented at the outset.

Comments.

There could, of course, be no doubt that the patient was the victim of a severe form of angina pectoris, and that the attacks were accompanied by marked arterial constriction. It was fortunately easy to arrest the seizures. They had, however, persisted for weeks without any lessening in their frequency or in their severity. The question which awaited solution was, Could anything be done to lessen the tendency to the attacks? Palliation was easily achieved; but could any measures be adopted which might be more curative? The point in the history which was most uncommon was the fact that the taking of nourishment was almost always followed by an attack, although the amount taken was small and could hardly be further reduced. Acting, however, on this indication, it was decided to materially alter the diet, the nature of which has been already stated. The dietary now advised was to consist of peptonized milk, peptonized oatmeal gruel, etc., and one raw egg well beaten up daily. The patient was moreover given small doses of arsenic, tincture of strophanthus, and spirits of nitrous ether. This line of treatment was persisted in, and when I saw the patient a fortnight later the report of his condition showed considerable and satisfactory improvement; the attacks had greatly diminished in number and severity, and the taking of nourishment was only occasionally followed by an attack; the pulse was stronger. The tongue, however, had become coated, and small doses of rhubarb and soda were advised.

A week later the report was that the attacks had still further lessened in severity and frequency, and that when an attack was threatened it could be averted by a dose of aromatic spirit of ammonia or even by a dose of rhubarb and soda mixture. When pain in the elbow came on, it was accepted as an indication that the threatened attack would be somewhat severe and the patient was at once given a small dose of nitrite of amyl. The sensations

which now more ordinarily preceded an attack were a sense of discomfort and distension in the stomach, and attacks were often associated with definite flatulent distension, the escape of gas being followed by relief. The medical attendant noted that when the abdominal discomfort supervened the pulse rate increased, and there was distinct hypertonus; as the attack passed the vessel wall relaxed and the rate returned to normal. Within the next two months the patient's condition varied a good deal, but it was noted that any gastro-intestinal or digestive upset at once threatened to induce an increased number of attacks. The management of the diet and of the digestive system required much tact and judgement and was admirably conducted by the doctor in charge. In some weeks the patient had improved so much that he was able to be out of bed and to move about the house for the greater part of each day, and to interest himself in outside affairs. His clinical history for the succeeding months was chequered, for as his condition improved, it became impossible to continuously diet him as was desirable; still, at the end of June, 1905, he had not had a seizure for six weeks and was in fact freer of attacks than he had been for years.

Soon after this, however, albuminuria made its appearance with anasarca. The anasarca was got rid of, but the albuminuria persisted; Cheyne-Stokes breathing developed and became extreme. The patient finally developed gangrene of his entire right lower limb, of which he died in the end of October, 1905, a proof that his arterial system was more degenerate than the radials indicated. I submit the case as illustrating an extreme exaggeration of the normal reflex which has been described. The angina in this patient was associated with a weak and somewhat enlarged heart. The hypertonic spasm of systemic arterioles, resulting from gastro-intestinal stimulus, proved so severe a strain on the weakened heart as frequently to lead to a syncope, during the anginal paroxysm, which was only short of being fatal. If we had had the opportunity of seeing the heart, it is probable that atheromatous or ossified coronary arteries would have explained its debility. The instructive feature was the extraordinary sensitiveness of the peripheral organs from which the reflex started, and the success in allaying that sensitiveness by changing the kind of nourishment given, and its partial predigestion. The remedial effect was so pronounced that it gave me the impression that in this patient the normal reflex had been irritated beyond control by the free use of the more pure proteid foods, until it responded to the most trifling local irritation. That the anginous symptoms were always determined by the vascular hypertonic contraction was undoubted, and, so long as the latter could be restrained, there was no angina, and, as has been indicated, the key of the position was in the alimentary system.

CASE II.—Condition allied to Angina, showing the Stimulation of the Arterio-Cardiac Reflex by Digestive Disturbance.

This case might be classed as angina sine dolore, but I use it as an example of conditions allied to angina pectoris, in which hypersensitiveness of the abdominal reflex gave rise to phenomena of considerable interest and importance.

The patient, aged 68, was a member of one of the learned professions, and had spent many years in the tropics. I saw him in November, 1904, during convalescence from an influenzal bronchopneumonia, and was informed that on the afternoon of the previous day between 4 and 5 o'clock he had had a "fainting attack," in which his face became pale, he broke out into a cold perspiration, and the pulse was imperceptible. The nurse had been greatly alarmed, as she thought the patient was dying. He soon, however, rallied from this, and I saw him in the afternoon of the following day. It is unnecessary to enter into the details of this case at this stage, beyond saying that when the medical attendant and I inquired fully into his dietary it was found to be very faulty, and he was forthwith put upon a comparatively austere regimen.

He had no similar attacks during the following days, but he often spoke of an attack as impending, and he apparently expected an attack about 4 o'clock in the afternoon. It was thought by his family and the nurse that this was pure nervousness, and an expression

of the pessimistic view he tended to take of his condition. Five days later I again saw the patient and obtained from the nurse the statements on which the preceding account is based. I was waiting by the patient's bedside for his medical attendant to arrive, and thought the patient, who was sitting propped up in bed, looking well, for his colour was good. His pulse was 80, soft, small, and feeble. The respiration, however, showed a long deep breath often with a little sigh, followed by several quiet inspirations; it was distinctly "cerebral" in type, and it made me somewhat anxious. I asked the nurse to give him half an ounce of whisky in water, although he had had a couple of drachms shortly before, as I wanted to see the effect it would have upon the pulse and respiration. The effect was unexpectedly striking and even alarming. In a few seconds the pulse altered greatly in character: the artery very plainly tightened up, becoming smaller and harder, and the rate varied within very wide limits. It was frequently 88 per minute, then it would run up to over 100, and again sometimes fall even to 40. Along with this sudden irregularity in the pulse rate there were equally pronounced respiratory and cerebral phenomena. Lying with his eyes shut the patient seemed to fall asleep, and the breathing was so quiet and shallow that only on close attention could it be seen that he was breathing at all. Then he awakened up with a start and a groan, followed by some deep breaths. This series of phenomena went on with perfect regularity as long as he was not spoken to, each part of the cycle only lasting some seconds. When asked on awakening what was the matter, he explained that he had become unconscious, and had been dreaming that he was falling down a precipice, or passing through some equally unpleasant experience, often in company with some old friend long dead. He spoke of these attacks as "delirium," and insisted that the present was only an exaggeration of what he had experienced every afternoon, and to which reference has already been made. He could be kept awake by speaking to him, and he preferred being spoken to, because, as he said, it helped him to fight against the attacks. When he woke up from the nightmare his words were sometimes incoherent and unintelligible for a few seconds, but he rapidly regained complete self-possession.

This alarming and curious condition lasted about an hour. It had come on about three hours after lunch, which had consisted of a chop, spinach, and one potato. Some time after that he had had milk and hot water and two drachms of whisky in lieu of afternoon tea. When the patient's condition was as has been described, I asked the nurse to make some tea, and he was given two-thirds of a moderate-sized cupful of this. The attack had completely passed off by the time the doctor arrived; the pulse had returned to the condition it was in before the attack, the breathing was regular, and he was quite calm and self-possessed. When the next meal came he was quite able to feed himself. I had no doubt the patient's view was correct that the symptoms shown this particular afternoon were only an exaggeration of the afternoon symptoms which he had spoken of, but which no one with more understanding than a nurse had witnessed. The doctor had called at various hours for the purpose of observing the attacks, but the stimulus of his visit appeared to be sufficient to postpone them.

Comments.

The phenomena in this case were very striking, and the symptoms could only be regarded as indicating an anxious condition of matters. The fact that they threatened chiefly in the afternoon, and had so far lasted only for a comparatively short time each day, was reassuring; while the time of their occurrence suggested a connexion between the symptoms and the stage of the digestion of the mid-day meal, which was the principal meal of the day. Suspicion was all the more strongly directed to this view by the fact, of which I had no doubt, that the attack I witnessed was precipitated or intensified by the half ounce of whisky given at my suggestion. The immediateness of the effect was startling: it gave the impression of being produced the moment the draught reached the stomach. It was indeed impossible to escape from the conviction that the phenomena pointed to the production by the alcohol of a peripheral gastric irritation or stimulation, which precipitated or intensified a condition which was already threatening. On this

assumption the local irritation must have been followed by a very prompt reflex effect. The impulse communicated by the sensory nerves of the stomach to the vasomotor centre must have stimulated that centre so as to increase its vaso-constrictor action, the result being the hypertonic contraction of the radial artery which was so markedly present. This stimulation of the vasomotor centre must have also told in some way upon the cerebral blood supply, judging from the cerebral manifestations which were present. That even the motor area of the brain was involved was shown by the occurrence of muscular twitching and starting of the limbs, especially on the right side, when the other cerebral manifestations were at their maximum. The respiratory centre, as shown by the great respiratory irregularity shared in the perturbation; this association, however, is of common occurrence. That the attack should have passed off as it did, without any therapeutic measures being taken beyond the administration of a few ounces of tea infusion, only strengthened the view that it had been determined by peripheral conditions of a temporary kind; that, indeed, the true cause of the trouble lay in an error in the digestive process at this particular period after the mid-day meal.

The diet was altered in some of its details, and for an afternoon or two the patient was given a dose of one of the commonly-used vaso-dilators as soon as the symptoms threatened to come on. He had no more attacks; his recovery was complete, and he has returned to his duties in the East.

CASE III.—*Anginous Paroxysms First Appearing on Physical Effort: Influence of Emotion and of Diet.*

This patient was a public servant, whom I saw in July, 1905. He had suffered for some time from attacks of typical angina pectoris, commencing with pain referred to the heart. This symptom first appeared when he was hunting, and so constantly reappeared when he attempted to follow the hounds that he had to stop doing so. He had had one or two attacks of great severity during the night, in at least one of which it was thought he was to die. He had been treated with considerable success by an eminent Irish physician. He carried about tabloids of trinitrin in his waistcoat pocket, and had been taking potassium iodide for some time. His condition had become practically stationary; hunting was out of the question; he could only walk on level ground; the slightest hill, or the slightest hurry, produced a breast pang, which arrested further activity. When I first saw him he was having a slight attack almost nightly, soon after midnight, and at that time he usually took a tabloid of trinitrin. He claimed to be a small eater, and his daily wine consumption did not exceed two or three glasses. His dietetic habits had not been seriously inquired into, and had not been altered. He had been told that the artery of his heart was rigid, and he and his family were dwelling under the shadow of the fear of sudden death at any moment. On examining him I found that the heart was slightly enlarged, the dullness reaching to the nipple line, but the sounds were clear and pure at apex and base. The pulse was regular, of good strength, and gave no indication of an enfeebled myocardium. The vessel wall was not much thickened for a man of his age, and in view of the arduous life he had led at home and abroad; there was no albumen or sugar in the urine, but it tended to be scanty and high-coloured. The bowels were acting, but not freely, and his conjunctivae indicated "sluggish liver." The question here, as in Case I, was whether anything could be done to carry improvement beyond the point it had reached. I was again fortunate in witnessing a slight attack in this patient. When I was examining into the state of his heart and vessels, which under the circumstances I was doing with great care, and not hurriedly, he became a little agitated and informed me that he was having a slight attack, with the usual feeling in his heart. On putting my finger on the radial pulse it had become markedly hypertonic. In a very short time the hypertonic spasm relaxed, and the heart sensation vanished.

Here was another instance of the pain and anxiety of angina, assuredly due to arterial spasm. There seemed to me, further, no doubt that the measure of emotional disturbance aroused by the medical examination, had induced the attack. There was, therefore, no doubt as to the

existence of marked vasomotor sensitiveness. As the patient was already taking potassium iodide more or less regularly, and trinitrin when threatened with a paroxysm, I felt that not much benefit could be expected from further medicinal measures. Guided again, therefore, by the theory that this vasomotor sensitiveness owed its origin to the circumstances and conditions of the alimentary system, I strongly advised that all wine should be stopped, and that other changes be made in the diet. The progress of the case need not be enlarged upon; it is sufficient to say that the patient made marked improvement in every respect.

CASE IV.—*Angina with Gastric Disorder: Attack caused by Palpation of Epigastrium.*

This was a female patient in the Royal Infirmary, who complained of attacks of pain in the chest, over the precordia, with shortness of breath and of indigestion and pain in the epigastrium. The heart was weak and somewhat dilated. I mention this case on account of an experience I had in connexion with it somewhat resembling an incident noted in Case I. On examining the patient's abdomen I found that there was surface hyperaesthesia in the epigastrium, as is common in some forms of gastric disturbance; and as I was gently palpating the region, I noticed the patient's face become pale and expressive of anxiety and discomfort. I at once put my finger on the pulse, and asked her what was the matter; she replied that the pain in her chest had come on. The radial artery had become markedly contracted and hardened. As the hypertonic spasm of the artery relaxed the heart discomfort passed off.

CLINICAL PATHOLOGY.

We are now in a position by gathering up our separate points to explain the whole clinical pathology of angina pectoris. In the first place there may or may not be a morbid anatomy. When present, it varies so widely in different instances that, from the earliest times to the present, many physicians have refused to accept any anatomical change as sufficient to explain the phenomena. Whenever this is the state of knowledge it will be found that men turn to that scapegoat of all that is unknown—the nervous system.

In attempting to interpret the phenomena of angina pectoris, I shall begin with an illustration of the simplest form of angina. Tobacco poisoning, from smoking, is commonly recognized as the cause of a form of angina pectoris—a toxic angina. It so happens that I can give myself tobacco angina by smoking strong tobacco. The symptom which arouses me to the fact that I must put my pipe aside is a sense of constriction at the heart, a feeling as if it were grasped; it is not a very unpleasant sensation, but it gives one the impression that it only required to be much intensified to make the dolor pectoris to be avoided, whatever self-denial it required. The heart discomfort is accompanied by a hypertonic contraction of the radial arteries. Discomfort and hypertonus disappear together. If I am told this is a neurosis, all I can reply is, if it is, opium poisoning, or tetanus, is also a neurosis; and that I do not look at morbid processes from that standpoint.

Whatever the degree of intensity the heart-pang may reach, it is, I believe, always the result of a sudden embarrassment of the myocardium, and usually and chiefly that of the left ventricle. This sudden heart embarrassment, varying widely in degree and intensity, is the one fact common to all the divisions or subdivisions under which all cases have been classed. The *angor animi*, which also varies in degree, is, I think, common to all sudden heart embarrassments, which fall short of being so immediately fatal that there is no time for the development of such sensations.

This heart embarrassment is, as Mackenzie³ has quite recently pointed out, an "impairment of the function of contractility." This is the modern expression of what Parry meant by "syncope." It is to this condition, associated with pain, that Rosenbach³⁴ confines the term "stenocardia."

The impaired contractility is due either, in the first place, to the sudden strain put upon a feeble myocardium by general arterial contraction, or by a call for increased work; or, in the second place, to a myocardium feeble, not from anatomical changes in it, but temporarily so, from deficient blood supply, the result of

the participation of the coronary arteries in a general arterial spasm.

The cause of the sudden heart embarrassment in the vast majority of cases is the arterial hypertonic contraction, or spasm contraction, we have been considering; the latter term indicating the more sudden and severe degrees of contraction. Even toxic conditions only give rise to angina when the factors indicated are present.

By means of the cases reported I have endeavoured to illustrate the remarkable relations which exist between the digestive system, or the materials introduced into it, and the general arterial system. I have also shown that conditions having their origin in the abdomen can induce a hypersensitiveness of a normal vasomotor reflex, which becomes apparent as arterial spasm, whenever the exciting conditions are sufficiently pronounced; and that when those conditions are altered the arterial spasm disappears.

As I have already indicated, the arterial spasm may be determined by direct irritation of the stomach. In two of my cases the spasm and its accompanying paroxysm of angina, were caused by palpation of the epigastrium. This illustrates the well-known nerve connexion between surface and viscera—gastric disorders frequently causing surface hyperaesthesia, while in these two cases the surface stimulus produced the same effect as if a stimulus had been applied directly to that viscus.

The stimulus is also provided by the substances taken into or absorbed from the alimentary tract. These substances exercise a supreme influence in producing and in maintaining the hypersensitiveness of the vasomotor reflex, and therefore of the vasomotor centre.

This hypersensitiveness of the vasomotor centre will explain what has long been recognized—that paroxysms of angina have as their main determining cause physical effort, mental emotion, or digestive disturbances.

THE UNIFYING PRINCIPLE.

By the application of the facts I have brought under review the group of symptoms known as angina pectoris can be satisfactorily explained, no matter how diverse the conditions may be in which it occurs. It explains the occurrence of the symptoms without anatomical change, while at the same time it shows the importance of the presence of anatomical changes in the coronary arteries, or in the myocardium. In this connexion it must always be borne in mind that even calcareous coronary arteries are not calcareous to their finer subdivisions, so that in this condition, as well as in the varying degrees of arteriosclerosis, the power of spasm contraction is not lost—may indeed be intensified. It gives to the nervous system its due place in the production of the phenomena.

To illustrate the production of a paroxysm by mental emotion or physical effort, let me refer to Case III, in which slight emotional disturbance or slight exertion had the effect of inducing a paroxysm. The connexion between the emotional centres and the vasomotor centre is a normal one, and constantly active. In this case a normal emotional impulse acted upon the hypersensitive vasomotor centre, resulting in the arterio-cardiac phenomena of angina. This was no abnormal excitation of any emotional centre. It was a normal excitation passing on to an abnormally sensitive vasomotor centre. The vasomotor centre in this same patient was equally influenced by slight physical effort; it was not the voluntary muscle centres, nor the nerve endings in the muscles, which were unduly sensitive, but the vasomotor centre to the normal influences resulting from voluntary muscular effort.

By the cases reported I have also shown that this hypersensitiveness of the vasomotor centre, even in grave angina pectoris, can be reduced, controlled, or even removed by dietetic measures, with the result that the anginous seizures are removed or greatly modified. In cases where the arterial spasm is associated with great anatomical change, either in the myocardium or in the coronary vessels, absolute cure can hardly be looked for; but in all cases the symptoms of angina pectoris may be much ameliorated by accepting, as a working hypothesis, the propositions I have ventured to submit, and conducting the treatment in accordance with what they indicate.

There was need of a principle which, when applied, would explain the phenomena in each of the various groups into which the disorder has been divided. There was abundant clinical experience showing the remedial effect on the paroxysm of vaso-dilators; but the only

explanation of the arterial spasm which up to the present time has been offered has been, to use Sir R. Douglas Powell's words, that of "a pure neurosis of the cardiovascular system."

Owing to the varying degree of intensity of the symptoms I would suggest that in classifying the cases the simplest distinction might be found in the terms "angina pectoris major" and "angina pectoris minor," the former being confined to those cases in which there was believed to be permanent anatomical change in the heart or its vessels.

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FOOD FEVER IN CHILDREN.

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I PROPOSE to describe, under the name of "food fever," a class of cases with which I have been familiar for a number of years. The disorder consists of an attack of fever which comes on suddenly, is accompanied by signs, more or less pronounced, of digestive disturbance, lasts in its acute form for several days, and may linger on in a modified degree for some weeks. If these attacks occur frequently—once a month or so, as they sometimes do—their effect upon the nutrition of the patient is highly injurious.

Dr. H. Davy, in the *Lancet* of September 24th, 1904, published an interesting paper on this subject, which he called "Fever in children caused by the indigestion of certain kinds of carbo-hydrate foods." Dr. Davy instanced the case of his own son, who suffered repeatedly from this form of pyrexia. The attacks came on quite suddenly and without discoverable cause, and by their frequency interfered greatly with the boy's nutrition, brought his education to a standstill, and kept his parents in a state of continual perplexity and alarm. I have already described these cases in my large work on *Disease in Children*, under the name of "acute recurring gastric catarrh." It is not, however, every case of recurring gastric catarrh which presents these symptoms, but those which do exhibit them are so characteristic, and to many practitioners so puzzling, that I have thought it might be convenient to group them together under a distinctive name.

SYMPTOMS.

The subjects of the complaint are usually neurotic children of either sex between the ages of 3 or 4 and 10 or 12 years, although I have seen cases in children still younger. The attack begins with headache, accompanied sometimes by vomiting, sometimes by looseness of the bowels, but always by signs of some kind of digestive upset. The child lies about with a dull, pasty face, a dirty tongue; he does not care to play, and has a temperature of from 101° to 105°. If the abdomen is examined, the stomach resonance is found to reach higher than normal—often nearly as high as the left nipple—and the edge of the liver can be felt one or two fingerbreadths below the costal margin. The water is very high-coloured, or thick, and very acid.

If the case is left alone, or not treated properly, the temperature remains for some days continuously higher than the normal level, and at some period of the twenty-four hours shoots up still higher. It generally rises a little after food, and is almost always 102° or more in the evening. At the end of a few days it subsides, either suddenly after a profuse sweat or gradually, but is often moderately raised (99° to 101°) for a week, or even longer.

If, when the fever begins, the child is given a good dose of calomel or blue-pill, the temperature generally falls directly the medicine has acted; but instead of remaining low, as it would in an ordinary case of upset, it rises again in the course of a few hours, and in the evening generally reaches 100°, or a little higher. For several days afterwards the temperature may remain uncertain—not continuously higher than normal, but rising above a healthy level at some period of the 24 hours, in the evening or after a meal. The children, as a rule, make no complaint of bodily discomfort, but are fretful and irritable in the day, and at night lie awake or sleep badly, grunting and groaning and tossing from side to side. Often they do not care about their food, and sometimes it is most difficult to make them eat anything at all. The younger children, when forced to take food into their mouths, roll it about from one cheek to another, as if they could not bring themselves to swallow it.

Children who have these attacks only at rare intervals soon recover their health after the temperature has fallen to its normal level, and seem in no way the worse for their indisposition. If, however, as sometimes happens, the attacks are repeated every five or six weeks, nutrition soon begins to suffer. Such children are pale and thin, have little or no appetite, and even if lively and active, have habitually a languid circulation and cold feet. These signs of malnutrition are generally accepted by the parents as the necessary outcome of a supposed delicacy of constitution. It is the frequent recurrence of fever which causes so much anxiety, especially as the attacks come on quite suddenly and without obvious cause. A child seems in his usual health, is perhaps going to school regularly and joining briskly in all the school games. One morning he leaves his home apparently as well as usual, but returns in the evening flushed in the face and complaining of headache. His temperature is taken and found to be 102° or higher. The complaint then follows its usual course. Some of the attacks, however, have special symptoms. A highly neurotic child may have a fit of convulsions, which may be repeated again and again. In a certain proportion of the cases a prominent symptom is vomiting, which may persist for several days, and I am strongly of opinion that many of the cases described as "periodic" or "cyclical" vomiting are of this nature. The vomiting is sometimes accompanied by pain in the epigastrium or umbilical region.

A little girl of 6 was subject to sudden attacks of fever, with vomiting and pain in the pit of the stomach. The febrile condition always lasted five days, during which time she looked yellow without being actually jaundiced, and her bowels were very confined. The child, when I saw her, was supposed to be well, but she looked very pale and thin and her feet were bitterly cold. She was brought to me that I might suggest some means of preventing the attacks.

The pain is sometimes the most distressing symptom.

A child of 8 was brought to me because she had suffered for more than a year with attacks of acute abdominal pain and high fever which came on every three or four weeks. The child, after a restless night, would complain in the course of the morning of severe abdominal pain. Her temperature would be 103° or 104°. The pain always went on continuously for three days, the child sitting or lying with her knees drawn up and crying with the pain. Sometimes she was sick. At the end of the three days the pain and pyrexia would subside, leaving the patient much weakened, with a poor appetite and dislike to any exertion. I put the child upon a strict diet and enjoined special precautions against chills. I heard two years afterwards that the attacks had ceased from the time the treatment was begun.

In other cases there are signs of intestinal catarrh, generally of the descending colon, and the child passes loose stools containing mucus and blood.

Not long ago I saw a boy 11 years old, who was brought with the history of many attacks of this kind during the last four years. In these attacks the temperature would rise as high as 105° or 106° for the first few hours, then on the second day it would fall, but remain higher than normal in the