Remarks ON THE MEANING AND MECHANISM OF VISCERAL PAIN

AS SHOWN BY THE STUDY OF VISCERAL AND OTHER
SYMPATHETIC (AUTONOMIC) REFLEXES.

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[Concluded from p. 1454.]

III.—THE MECHANISM BY WHICH VISCERAL PAIN IS PRODUCED.

Insensitiveness of the Viscera to ordinary Stimuli.

WHEN all the facts concerning visceral pain are taken into consideration, it will be found that the usual conception of the manner in which pain is conveyed to the mind fails to explain the phenomena connected with the recognition of pain when it is due to affections of the viscera. To begin with, the viscera are insensitive to those stimuli which produce pain when applied to the external body wall. The visceral tissues can be cut, torn, and burnt, and the individual remain unconscious of any sensation. Nevertheless, we all know that the viscera are capable of giving rise to pain of the most excruciating character, so that the reason the methods employed to produce pain in stimulating the external body wall fail to produce visceral pain is manifestly that they are inadequate; and from this fact alone we are forced to conclude that a satisfactory explanation of how visceral pain arises has yet to be found.

Sensitive Tissues of the External Body Wall.

Before dealing with visceral pain it is necessary to comprehend more fully the sensibility of the tissues forming the external body wall. How profoundly ignorant we are of this subject has recently been shown by the investigations of Head and his colleagues.

For the purpose of this study I shall briefly refer to a few points the misunderstanding of which has so often led astray the clinical observer.

If we take the abdominal wall we find three great layers endowed with exquisite sensibility to pain. The first of these, the skin, I need not dwell upon, save to point out how its sensibility frequently becomes increased in visceral disease, and how this increased sensitiveness is united to an exalted muscular reflex. The second of these sensitive layers is formed by the flat muscles of the abdomen and is the most important, and the one most commonly affected in visceral disease, its sensibility being very readily increased. Muscular hyperalgesia is such a striking phenomenon, is so frequently present, and plays such an important part in the protective mechanism, that it is astonishing to find it almost universally overlooked. One can read elaborate treatises devoted to special organs in which this symptom is the most striking and the most instructive feature, but its presence is nevertheless overlooked or misinterpreted. If any one will take an ordinary case of stomach ulcer, appendicitis, gall stone or renal colic, or enlarged liver, and note the tenderness of the abdominal wall and observe how this deep tenderness extends far beyond the site of the organ affected, they will begin to appreciate the nature and significance of this symptom. With a little care one will be able to distinguish these symptoms from cutaneous hyperalgesia and from hyperalgesia of the deeper tissues. The third sensitive layer is one of which anatomists and physiologists were quite ignorant when I first found it out clinically. It is the layer of loose connective tissue lying immediately outside the peritoneum. I suspected its presence for a long time as I frequently could get exquisite tenderness in pushing my fingers between the recti muscles; for instance, in cases of gastric ulcer, the stomach not being affected by the pressure. Its demonstration occurred first in a case of operation for the radical cure of hernia by Mr. Caird, who had injected cocaine in the site of the incision. The skin and muscles were cut through and the patient experienced no pain. When Mr. Caird gently tore through the loose connective tissue the patient experienced most exquisite pain. After the peritoneum was exposed it was incised and afterwards stitched and

the patient felt no pain. This observation I have verified on several occasions. I consulted several anatomists but found they had no knowledge of any nerve distribution in this region. Subsequently Ramström made a careful histological examination of the abdominal wall of the man and other mammals and showed that this region was richly endowed with nerves and nerve endings, the nerves being derived from those which supply the muscles of the belly wall.

This observation may probably afford a clue to the confused statements that exist in regard to the sensitiveness of the peritoneum. I can only say this, that I have on numerous occasions in the course of operations scratched and cut the serous surface of the peritoneum on conscious subjects, and have never known the slightest sensation elicited. One can understand, however, that the inflamed peritoneum would readily affect this remarkable nervous layer. Peritonitis, however, so readily produces muscular hyperalgesia and tonic muscular contractions (viscero-muscular reflex), that the pain and tenderness is demonstrably, in the majority of cases, of spinal origin.

With the recognition of these sensitive structures—frequently rendered exquisitely sensitive to painful stimuli in visceral disease—it will be understood how impossible it is to judge of the sensitiveness of the viscera by external exploration. When, therefore, we find the surgeon or physician demonstrating the sensitiveness of any viscus, it will be realized that he is in reality stimulating, in his examination, those extremely sensitive structures of the external abdominal wall, and referring the pain he elicits to an organ that is totally insensitive to any such stimulation.

Artificial Production of Visceral Pain.

It is a curious fact that although the belief is so universally held that the viscera are endowed with "sensory" nerves, and that physiologists refer to afferent sympathetic nerves as "sensory" in function, not a single authentic observation has been rendered in proof of these assertions. Of course, a great deal depends on what is considered evidence, many people being perfectly satisfied if they elicit pain by pressing over an organ. Physiologists have interpreted certain movements as an expression of pain after electrically stimulating an afferent sympathetic nerve. But this does not prove that pain was evoked, nor does it prove in what situation the pain was felt—for the location of the pain is the key to the problem. It is because of the absence of the specification of the locus of the pain that Lennander's otherwise important observation is rendered of little value in respect to this investigation.

That pain can be produced by visceral stimulation is easily demonstrated if one employs an adequate stimulus. It is now many years since I pointed out that the most violent pains of which we are conscious are associated with hollow muscular organs, and that by producing violent contraction of a hollow viscus pain can be elicited. The easiest way to do this is to give a distending enema of warm water and to retain the enema until painful peristalsis results. That the pain is really due to the contraction of the muscle wall of the bowel is evident from the fact that with the relaxation of the sphincter during the pain the contents of the bowel are expelled with considerable force, and the pain at the same time subsides. Here it is evident that a considerable portion of the descending colon and rectum must have contracted, but the pain is not felt along the position occupied by these structures, but, in the majority of people, it is referred across the middle line immediately above the pubis.

The following observation demonstrates an exactly similar series of facts:

I had occasion to resect a portion of the small intestine in a conscious subject whose abdominal cavity had been laid open. There were numerous peritoneal adhesions, and while I cut and tore these the patient was unconscious of any sensation. I cut and stitched the serous surfaces of parietal and visceral peritoneum; I tore adhesions from the liver; I cut and sutured the bowel and mesentery, and no sensation was felt. After preparing the upper part of the bowel it was wrapped in a warm cloth and laid on one side. During the subsequent steps the patient frequently moaned. I asked him if he felt pain, and he replied that he did. I asked him where he felt the pain, and he indicated with his hand that it was across the middle line at the level of the umbilicus. I at first felt that it might be due to the part that I was manipulating, but the

pain was intermittent. Chancing to look at the prepared upper part of the bowel that lay on the left side of the abdomen, I observed that every few minutes a peristaltic wave passed over the lower portion of it, and when this occurred the patient moaned in pain. I made certain that the pain was connected with the peristaltic wave, and also made sure the patient had no doubt as to the place in which it was felt, with the result that here before my eyes was the cause of the pain which the patient felt, and yet the patient referred the site of the pain with precision to an area 10 in. or 12 in. away from the contracting bowel.

The Relationship of the Site of Pain to the Site of the Lesion.

For many years I have kept notes of the position in which pain was felt in a great variety of diseases, and in course of time I have been able to identify the exact site of the lesion in cases that come to operation, or to *post-mortem* examination. The conclusion arrived at from the consideration of these cases was that the situation of the pain did not as a rule directly afford any clue to the situation of the lesion, but when the situation of the pain was immediately over the lesion, other evidences showed that the pain was not felt in the organ, but referred to the sensory nerves in the external body wall. I shall quote the proofs for these conclusions presently, but here I will describe the theory which explains the peculiar nature of visceral pain.

The Mechanism by which Pain is produced in Visceral Disease.

When a nerve that terminates in a sense organ is stimulated in any part of its course from the periphery to the brain, a stimulation is given to the brain of a kind similar to what would have happened if the peripheral end-organ had been stimulated. Thus the stimulation of any part of the optic nerve or auditory nerve gives rise to the sensation of light or of sound. In the same manner, if a sensory nerve be stimulated in any part of its course through the brain, spinal cord, or trunk of the nerve, the resultant sensation is referred to the peripheral distribution of the nerve in the external body wall. In the normal processes of life, a stream of energy is continually passing by the afferent nerves to the spinal cord, and continuously playing upon the efferent nerves that run to muscles, blood vessels, and so forth, maintaining what we call "tone" in muscles and blood vessels. These processes are conducted so that they give rise to no appreciable sensation.

If, however, a morbid process in a viscus gives rise to an increased stimulus of the nerves passing from the viscus to the spinal cord, this increased stimulation affects neighbouring centres, and so stimulates sensory, motor, and other nerves that issue from this part of the cord. Such stimulation of a sensory nerve will result in the production of pain referred to the peripheral distribution of the nerve whose spinal centre is stimulated, so that visceral pain is really a viscerosensory reflex. If the increased stimulus affects a motor centre, then a contraction of the skeletal muscle results, and thus is produced the visceromotor reflex.

The reasons why the pain is referred to portions of the body so far apart is because in the course of development the tissues that in a low scale of life immediately covered the organ have been displaced. Thus, the pain felt in the testicle in renal colic is due to the fact that in its journey down to the scrotum the coverings of the testicle receive a twig from the first lumbar nerve, and when the root of this nerve is stimulated, as in renal colic, the pain radiates to the testicle. In renal colic one never finds the skin of the scrotum hyperalgesic, but always the deep covering of the testicle, because the scrotum is supplied by the sacral nerves.

Referred Pain.

This view is the one practically adopted by all to account for what is called "referred pain." Ross described visceral pains as of two sorts, "splanchnic" pain in the organ and "somatic" pain referred to some part of the body wall remote from the organ. But when the so-called "splanchnic pains" are critically examined they will be found to be of the same nature as somatic pains. Thus Ross said that the pain in the back in cases of gastric ulcer was referred or somatic, while the localized epigastric pain was local—in the organ itself—splanchnic. So confident was he that he said that if he passed a needle

through the site of the latter pain he would penetrate the ulcer. This view is one so commonly held that I shall deal in some detail with the pain of gastric ulcer, but here I may simply point out that in the movements of respiration and in the movements of the walls of the stomach the ulcer is bound to shift its position, and if the pain were in the ulcer the situation of the pain should be constantly varying, whereas it remains fixed even on very deep inspiration and expiration.

Radiation of Pain.

Perhaps the best evidence to be found as to the true nature of visceral pain is to observe the manner in which pain spreads. No attempt is ever made by writers to appreciate the meaning and significance of the spreading of pain. Thus in an elaborate monograph on gall stones, a "hypersensitive" gall bladder and a "tender liver" is spoken of, and the pain of an inflamed gall bladder is "diffused over a large area along and below the margin of the liver." In what tissues was this widely diffused pain felt? If the pain were in the gall bladder, how comes it that it was felt in a region more extensive than that occupied by the gall bladder?

The diffusion of pain over a wider area than that occupied by the lesion originating the pain can be proved in many ways to be due to an extension of the irritation affecting the roots of sensory nerves in the spinal cord. Thus the extension of the painful area is frequently associated with hyperalgesia of the tissues of the external body wall. The pain is often found to radiate along peculiar areas, inexplicable unless we recognize the relationship in the spinal cord of the nerves supplying these areas, as, for instance, when the pain of angina pectoris passes from the front of the chest into the axilla and down the arm—that is to say, into areas supplied by contiguous nerve roots (third, second, and first dorsal nerves); or the pain may appear at a distance from the affected organ and gradually approach it till the pain is felt in the tissues covering the organ, as when in angina pectoris the pain may at first be confined to the arm, but with increasing severity radiates to the front of the chest.

The radiation of pain that occurs in a skin lesion is also due to the strong stimulus spreading in the spinal cord, exactly in the manner I showed in the spreading of the autonomic sensation and pilomotor reflex. This is brought out very clearly in the following experience related by Sherrington: "By applying a mustard leaf over the front of the upper part of the sternum I can produce in myself the sensation of a patch of hot, unpleasantly tingling character, referred to the inner side of each upper arm, just above the inner condyle." "In this case the associated sensations and the stimulus applied lie, as shown by delimitations of the cutaneous segmental-nerve areas, both of them within the same segmental area." I have on several occasions been able to produce a goose-skin reflex in a limited area on the front of the chest over the second and third costal cartilages by stroking the inner and anterior surface of the arm above the elbow, and a limited area of goose-skin in the arm by stroking the front of the chest in the place just mentioned. From such facts as these we are justified in drawing the conclusion that Sherrington's experience demonstrates that the radiation of pain from a local cutaneous stimulus is due to an excitation of sensory cells in the spinal cord.

The Pain in Gastric Ulcer.

The study of the pain in affections of the stomach and bowels affords evidence for demonstrating the nature of visceral pain, and at the same time shows the practical importance of this method of observing visceral pains. In this part of Lancashire gastric ulcer is a disease of extraordinary frequency. For many years it has been my habit to note the situation in which pain was felt most severely in the hundreds of cases that have consulted me. I soon found that the great majority of these patients referred the pain with great precision to some region in the epigastrium. Now the fact that gastric ulcers are situated frequently in parts of the stomach at a distance from the epigastrium, raised my suspicion that the pain could not be felt in the ulcer. Examining these cases more carefully, I found this localized pain was frequently associated with great tenderness of the skin,

and of a limited portion of the upper part of the recti muscles. Watching cases that ultimately came to operation, or that died and on which I had a *post-mortem* examination, I found that the site of the ulcer bore no relation to the site of the pain, but that when the ulcer was near the cardiac end of the stomach the localized pain and cutaneous and muscular hyperalgesia was situated high in the epigastrium, whereas when the ulcer was near the pylorus the pain, etc., was situated low down in the epigastric region. The following typical instances not only illustrate this point, but demonstrate the practical utility of this way of examining cases.

I was called into consultation to see a patient twenty hours after perforation of a gastric ulcer had taken place; she had suffered for many months from pain after food, and located the site of this pain with great precision in the upper part of the epigastrium, over the xiphisternum (area A, Fig. 4).

Fig. 4.—The area A shows the site of pain when the ulcer was at the cardiac end of the stomach, A'; B, when the ulcer was in the middle of the lesser curvature, B'; C, when the ulcer was at the pylorus, C'.

I reasoned from this that the perforated ulcer would be near the cardiac end of the stomach. The incision opening the abdominal cavity was, therefore, made as far to the left as possible. On opening the abdomen there was found a large quantity of fluid, and a considerable quantity of flaky lymph covered most of the exposed organs. The stomach was carefully searched, and we found it rather firmly adherent at the cardiac end to the posterior wall of the abdomen. We inspected the whole of the stomach except the adherent part, and there can be no doubt that the adhesions surrounded the ulcer. We resolved not to break down these adhesions, as it would have been impossible to drag this portion of the organ sufficiently far out to enable it to be stitched. The abdomen was very efficiently flushed, the wound stitched up, and the patient made a good recovery. Approximately the site of the ulcer corresponded to the area A', Fig. 4.

A girl, aged 14 years, who had had pain after food for many months, referred the situation of the pain, with great precision, to a spot in the middle of the epigastric region (area B, Fig. 4). She was suddenly seized with collapse and severe pain over the upper part of the abdomen. There could be no doubt that it was a case of perforation of the stomach. Within eight hours we opened the abdomen, and, as I had noted the situation of the pain previous to the rupture, I suggested that the ulcer would probably be found in the middle of the stomach. The incision was therefore made well to the left of the middle line. The perforation was at once met with in the middle of the stomach (area B', Fig. 4), on the lesser curvature. It was stitched up, and the patient made an excellent recovery.

In another patient, who had suffered for years from gastric ulcer (pain and vomiting of blood) the pain was always felt at the lower part of the epigastrium. She died of phthisis. At the *post-mortem* examination there was the mark of a small blister at the lower part of the epigastrium (area C, Fig. 4). She was wont to apply a small blister over the place she felt the pain. I asked my colleague to note this area and its relation to the ulcer. The ulcer was found in the pylorus corresponding to the area marked C', Fig. 4.

Another case had been under my care for ten years for repeated attacks of epigastric pain. She consulted an eminent surgeon, who wrote to me stating that the patient had an ulcer in the middle of the stomach and on the posterior wall, and he recommended an operation for her relief. I re-examined the patient, and made the following note: That, inasmuch as the pain is situated at the lowest part of the epigastric region, and as there is also here a limited area of tenderness of the skin, the ulcer should be found at the pyloric orifice. This view was verified at the operation subsequently performed.

Pain in Peristalsis of the Bowel.

From the observation of a large number of cases of obstruction of the bowel, I found that if the obstruction were in the small intestine the violent pain was usually referred across the middle of the abdomen, but never felt below the umbilical region. When, however, the pain descended to the hypogastric region then the obstruction was found in some part of the great intestine. This is simply due to the fact that above the seat of obstruction the violent peristalsis produces pain, whereas below the seat of obstruction the bowel does not contract. This view is corroborated by the observation I have already cited where the small intestine was seen to contract in the course of an operation for resection, and by the results of a distending enema. It can also be observed in certain

cases of diarrhoea. Thus, in a patient to whom I had given large doses of digitalis pain of great severity was felt in the lower part of the epigastrium, with intervals of relief. This pain gradually descended in the middle line until it was felt immediately above the pubis. Coincident with the pain reaching this situation, an urgent desire for defaecation was experienced, and relief was at once obtained by the evacuation of a large and loose motion. It is to be justifiably inferred from this case that violent peristalsis had been set up in the upper part of the small intestine, that the peristalsis had swept over the small intestine and large intestine, and that although different parts of the bowel must have contracted in different parts of the abdomen, the pain was felt in the central areas of the abdomen, descending as the stimulus, from descending coils of intestine, reached successively lower portions of the spinal cord.

From the foregoing considerations the legitimate conclusion to be drawn is that all pains arising from viscera supplied only from the autonomic nervous system are part of a reflex mechanism (viscero-sensory reflex).

IV.—THE MECHANISM BY WHICH THE SYMPTOMS IN ANGINA PECTORIS ARE PRODUCED.

In seeking an illustration of the application of the principles I have endeavoured to enunciate, I have chosen the subject of angina pectoris, as the symptoms can be referred with certainty to the organ at fault, and because the complex of symptoms that are included in an attack of angina pectoris are capable of being analysed with great precision on account of the peculiar distribution of the sensory nerves, whose centres in the spinal cord and medulla are in close relationship to the centres of the autonomic nerves of the heart. The cases I cite demonstrate that the term "angina pectoris" includes a number of reflexes, and I give in detail the more conspicuous of these, namely, sensory reflexes, when the pain and hyperalgesia affect the chest, arm, head, and neck; motor reflexes, resulting in spasm of the intercostal muscles; secretory reflexes, as shown by profuse secretion of saliva and urine. Some cases show other reflexes, as the respiratory, but the discussion of these would lead me beyond the object I had in writing this series of papers.

Notwithstanding the numerous papers devoted to the consideration of angina pectoris, practically none have dealt with the analysis of the symptoms present during an attack. Angina pectoris is so often surrounded by such tragic circumstances, that it forms a suitable theme for turgid eloquence, and on that account we too often get the lurid description of an impressionist artist instead of the plain, matter-of-fact description of an accurate observer. A careful sifting of all the details brings out the fact that the essential principles underlying the pains associated with affections of the heart, differ in no way from those of any other hollow muscular organ. So terrifying is the attack to the patient that his perception of the details of his suffering are generally confused, so that often no clear account can be obtained from his description; but if he is intelligent and is asked to note particulars in future attacks, he will often be able to throw a very valuable light on the onset and character of the sensations he experiences. The observations of patients made by the physician during an attack also afford great help in this respect.

The Viscero-Sensory Reflex in Dilatation of the Heart and Liver.

Before dealing with the more characteristic attacks of heart pain which go by the name of angina pectoris, I wish first to draw attention to the very distinct sensory evidences that arise from the dilatation of the heart. These sensory symptoms are practically identical with those that arise in cases of distension of any other viscus, as the stomach, bladder, or liver. To illustrate this I select cases where the dilatation of the heart occurs rapidly, and is followed by a rapid distension of the liver such as we find in certain forms of paroxysmal tachycardia. In certain of these cases the rhythm of the heart starts suddenly at the ventricle (or more probably at the fibres joining auricle and ventricle). The heart at once beats with great rapidity, but fails to maintain the circulation, and in consequence we have very rapidly-developed great dilatation of the heart, fullness of the

veins, enlargement of the liver, and dropsy. I have seen a number of these cases, and in two particularly I had observed some fifteen to twenty attacks, of which the following is a good example:

Observation 1.—The patient may be in good health and no abnormality be detected save, in some cases, the occurrence of a ventricular extra systole. Suddenly the heart's rate becomes greatly accelerated—to 180 beats and more per minute. In a few hours' time the patient's face becomes dusky, the lips become swollen and livid, and there appear great shortness of breath on exertion, marked increase in the size of the heart, distension and pulsation of the veins of the neck, and enlargement and pulsation of the liver. The jugular and liver pulsation are of the ventricular type. Pain and oppression may be felt over the chest. The skin and deeper tissues of the left chest are extremely tender on pressure in the area shaded in Fig. 5. If the pectoralis major muscle be grasped where it forms the axillary fold it will be found extremely tender. The skin of the left side of the neck may also be tender, and if the left sterno-mastoid muscle and the left trapezius muscle above the middle of the scapula are lightly grasped they may be found exquisitely sensitive. The skin and muscle over the liver will also be found extremely sensitive to pressure, and the parts hyperalgesic extend over a much larger area than the enlarged liver. If the heart's rate revert to the normal the patient at once experiences great relief and in a few hours all signs of the circulatory disturbances disappear.

Fig. 5.—The shaded areas show the regions of cutaneous and deeper hyperalgesia in case of acute dilatation of the heart and acute distension of the liver.

The tenderness of the skin and muscles in the regions described above may be found in patients during the early stages of dilatation of the heart or of the liver from any cause and is very common in heart failure secondary to mitral disease. In some, one can tell when improvement is taking place by noting the diminution of this tenderness.

There can be little doubt as to the mechanism by which the hyperalgesia of the tissues in the three regions is brought about. The relationship of the dilatation of the heart and liver with these sensory phenomena was undoubtedly that of cause and effect. The tenderness to pressure of the tissues in the left chest was due to the stimulation of the afferent sympathetic nerves by the dilated heart. These nerves stimulated the sensory centres of the third and fourth dorsal nerves in the spinal cord, so that a stimulus reaching them from their peripheral distribution gave rise to a painful impression. The tenderness of the left sterno-mastoid and trapezius muscle and of the skin of the neck was due to the afferent fibres of the "bulbar autonomic" system—that is, the vagus conveying a stimulus to the sensory roots of the second and third cervical nerves. The hyperalgesia of the tissues covering the liver was due to a stimulation of the sensory centres in the spinal cord by the afferent sympathetic fibres from the engorged liver.

The Manner in which the Distribution of the Upper Dorsal Nerves are Modified in the Course of Development.

In order to appreciate the mechanism of the pain felt in affections of the heart, the manner in which the upper dorsal nerves come to be distributed should be borne in mind. Ross has pointed out that in the primitive vertebrates, before the development of the limbs, each spinal nerve is distributed segmentally round one half of the body. The upper dorsal nerves are therefore entirely distributed over the thorax and to the tissues covering the heart. The upper limbs, as they bud out from the trunk in their development, drag with them away from the chest portions of the upper dorsal nerves, so that parts of the first and second dorsal nerves are distributed to the ulnar border of the forearm and inner surface of the upper arm. Thus, suppose a pain originating in the heart and stimulating the first and second dorsal nerve, it would be felt in the lowly vertebrate over the heart, whereas in man it would be felt in the upper arm or in the forearm.

The Pain of Angina Pectoris is a Viscero-sensory Reflex.

The usual description given of the pain in angina pectoris is that it is felt in the heart and shoots into the arm, or that there are two pains, a local pain in the heart and a referred pain in the arm. If, however, a careful analysis be made of all the symptoms present, facts will be found that practically demonstrate that in angina pectoris there is but one kind of pain, and that its production is in accordance with the law I have attempted to establish in this series of papers, namely, that it is a visco-sensory reflex. It is not in every case one is able to demonstrate the proofs of this hypothesis, but the facts derived from suitable cases afford legitimate conclusions applicable to all cases. Shortly, these facts are, that the pain in the very gravest cases may be felt in regions distant from the heart, that this pain is identical in character with that felt over the heart, that the pain may originally start in parts distant from the heart and gradually approach and settle over the heart, and lastly, that the tissues of the external body wall over the heart may be found extremely hyperalgesic after the pain has passed away. From this last fact it is inferred that, inasmuch as the seat of pain corresponds to the region of hyperalgesia, therefore the pain was felt by the hyperalgesic nerves. To assume otherwise is to ignore a principle that explains satisfactorily the sensation of pain wherever arising.

The following observations illustrate this point. They are examples taken from a large number of cases that demonstrate the same feature. For the sake of brevity only those points bearing upon this argument are referred to.

Observation 2.—Female, aged 30, suffering from stenosis of the aortic, mitral, and tricuspid valves, was seized with a violent pain referred to the outer part of the chest wall. The pain passed off, but a sense of soreness and smarting remained over the part in which the pain was felt. On examining her I found a portion of the skin of the chest extremely tender to touch, corresponding to the area shaded in Fig. 6.

Fig. 6.—The shaded area shows the distribution of the cutaneous hyperalgesia and the site of pain in the earlier attacks of angina pectoris.

Fig. 7.—The shaded areas show the distribution of the cutaneous hyperalgesia and the regions in which pain was felt in the later and more severe attacks of angina pectoris.

A few days later she began to suffer from attacks of pain in the left breast and down the inside of the left arm, and on examination I found that the hyperalgesia had extended and occupied an area similar to that shaded in Fig. 7.

These attacks of pain became so severe on the slightest exertion that she was obliged to keep to her bed. She partially recovered from these attacks, but they recurred with increased severity. When suffering the most severe attacks the hyperalgesia embraced nearly the whole of the left chest and inside of the left arm and also a portion of the right chest. The left sterno-mastoid muscle and trapezius also became very tender, and the patient would sometimes complain of pain on the inner surface of the right forearm, and here also I found a patch of cutaneous hyperalgesia. Two years and a half after the first attack of pain she was recovering from a series of violent attacks, when, on getting out of bed she fell forward and died immediately.

The fact that in this case the attacks of pain were followed by a hyperalgesia of the skin in the region where the pain was felt, and the further fact that the pain started at a distance from the cardiac region, and was often felt most severely at a distance from the heart,

proves that the cardiac pain in this case was a viscerosensory reflex, and that the pain felt over the precordia was presumably of the same nature.

Observation 3.—Female, aged 56, with high blood pressure, suffers from pain in the foot (? gout), and has had several severe attacks of true angina pectoris, when the pain is referred over the left chest and through to the shoulder. After the attack she passes a large quantity of clear urine, and the skin and deeper tissues of the left breast and the left sterno-mastoid and trapezius muscles become very painful on pressure. In one attack the pain was felt in a limited area over the second left interspace, and next morning I found the skin and deeper tissues were extremely tender to the slightest pressure.

In this instance, also, the hyperalgesia of the skin corresponded to the region where pain was felt, and is a presumptive evidence in favour of the view that the pain as well as the hyperalgesia is the result of the viscerosensory reflex.

Observation 4.—Male, aged 42. Consulted me on October 18th, 1905, complaining of pain in the left little finger and ulnar border of the left hand and forearm, coming on when he exerts himself at his work and in going up hill. For some months he suffered from a dull aching pain at the back of his shoulder-blades. I found dilatation of the aorta, slight incompetence of the aortic valves, and slight enlargement of the heart (verified by subsequent *post-mortem* examination). The blood pressure was 150 mm. Hg. During the following weeks the pain in the left arm increased in severity, and gradually extended up the arm till it was felt in the axilla, and finally it invaded the left chest. At first the pain was wont to start at the little finger, and to pass rapidly up the arm, but latterly it seized him with such severity and suddenness that he could not tell where it began. It usually held him with the greatest severity either over the heart or in the inner surface of the left arm immediately above the internal condyle. I particularly asked him to note in his frequent attacks if there was a difference between the chest pain and the arm pain, and his reply was that there was no difference in the character of the pain, but, if anything, the arm pain was the worst. Sometimes the pain was very severe over the left side of the neck and behind the left ear. Under treatment he seemed to make considerable improvement. During January these attacks again came on. During some of the attacks the pain was so agonizing that he felt he was dying, and wished to die. The pain was equally severe in chest and arm, and saliva sometimes dribbled from his mouth. On February 12th the least exertion was sufficient to induce a severe attack of pain, and from 3 p.m. till 7 p.m. he was scarcely free, and ultimately became unconscious. When I saw him next day he was having another series of attacks. He was keeping his left arm very still, and helping himself to food entirely with his right hand. He told me he dare not move his left arm, as even the act of lifting the left hand to his mouth was sufficient to induce an attack. His pulse was extremely soft and weak. His blood pressure had fallen to 95 mm. Hg. During the following night he had another series of attacks, became unconscious, and expired.

I omit many details in this case on purpose to emphasize the regions in which the pain was felt in a case of the most severe form of angina pectoris. It seems to me that no other explanation save that of the viscerosensory reflex can satisfactorily account for the pains in this case, and to attempt to distinguish the chest pain as a heart pain and the pain in the arm as a referred pain would be arbitrary, illogical, and opposed to the evidence. The radiation of the pain from the hand to the chest was practically of the same nature as the more common radiation of the pain from the chest to the hand. The pain in the neck and behind the ear on the same hypothesis would be induced by the stimulus passing from the heart by the vagus, a view that would also explain the increased flow of saliva during some of the attacks—points to which I shall afterwards revert.

The two following observations of attacks of angina pectoris which I witnessed, illustrate true heart pains of the most severe type at places remote from the heart.

Observation 5.—Male, aged 14, suffering from adhesive mediastinitis, with enormous enlargement of the heart; seen with Dr. Agnew. The patient lay propped up in bed. As I was gently testing the sensibility of the skin outside and under the left nipple the patient was suddenly seized with an attack of severe pain. He gave a great sob and leant forward with his left upper arm across his chest and his right hand pressing it gently. He rocked backwards and forwards with deep sobs while tears streamed down his cheeks. His pulse became very soft and his face pale, with beads of perspiration on his forehead. In a few minutes the pain subsided and he lay back exhausted. Afterwards he said that, on my touching a certain spot, a pain shot from his chest into his arm, and during the whole time the awful

pain remained in his arm—and he put his hand over the fleshy part of the upper arm. Dr. Agnew told me he had given up examining him because on a few occasions, on applying the stethoscope, attacks similar to those I witnessed were induced.

Observation 6.—Female, aged 60, complained of pain of agonizing severity limited to the ulnar border of the left forearm. Coming to see me one day she hurried to catch the train, and when she reached my consulting-room she sat down. In a few minutes the pain seized her and she took up her left arm and nursed it across her breast with evidences of great suffering. The pain subsided in a few minutes and she said she felt as if she would have died. The pain was felt nowhere but in the left forearm. Three months afterwards the patient died from heart failure. At the *post-mortem* examination there was found marked atheroma and calcification of the coronary arteries and extensive chronic fibrous myocarditis.

In this last case the pain was doubtless limited to the highest sensory nerve centre (first dorsal) whose fibres are associated with the sympathetic supply of the heart.

Evidences of the Viscero-motor Reflex.

So far I have dealt with the viscerosensory reflex, and no less striking evidence can be found of the visceromotor reflex among the group of symptoms included in the term "angina pectoris." Some would limit the term "angina pectoris" to that class of cases where, in addition to the pain, there is a sense of contraction in the chest, amounting to the sensation at times as if the chest were gripped in a vice, or as if the breast bone would break. I am convinced that these sensations arise from spasm of the intercostal muscles, and correspond to the hard contraction of the flat abdominal muscles in affections of the abdominal viscera. If one watch a case of what is called "muscular rheumatism," where the intercostal muscles are affected, and where these muscles are stimulated by the slightest movement to violent cramp-like contractions, one cannot but be struck by the resemblance to the description given by the "gripping" sensation experienced by patients suffering from certain affections of the heart. I have watched the attacks in such cases, and could find no difference between them and those where the sense of contraction was the chief symptom in heart disease.

The visceromotor reflex may be present alone, or, as is more commonly the case, it may be associated with the pain. The purely visceromotor reflex is seen best in the elderly, where it may be considered as a symptom of one form of the terminal affections of the heart due to arteriosclerosis or old age. I have found it a precursor of steadily-advancing cardiac weakness, and although for a time considerable relief may be afforded, the changes in the heart are so advanced that in the nature of things only one end can be looked for. The three following observations illustrate this view:

Observation 7.—Male, aged 82, with large tortuous arteries, was seized while walking with a sense of constriction across the chest that compelled him to stand still. These attacks became so frequent and so severe that he could scarcely walk 50 yards before he had to stop and lean against the wall. He described the sensation as one not of pain, but as if somebody gripped the upper part of the chest with a strong hand. With rest and suitable treatment these attacks gradually disappeared. Three months after the heart suddenly became irregular (ventricular rhythm), dropsy set in, and he died after seven weeks of heart failure.

Observation 8.—Male, aged 74, had suffered for several years from attacks of intermittent high blood pressure. Latterly these attacks were accompanied by a feeling of tightness across the chest when he walked. This sense of constriction disappeared after a course of iodide of potassium. During one of these periods he suddenly died while reading his newspaper after breakfast.

Observation 9.—Female, aged 78. Two years before her death she experienced attacks of breathlessness with a sensation of constriction across the chest. This feeling of tightness was so readily set up that she was obliged to stay in bed. The attacks disappeared but recurred again shortly before her death. These latter attacks were accompanied by slight precordial pain. She became gradually weaker and died. At the *post-mortem* examination the coronary arteries were markedly thickened, with calcareous patches in their walls.

The following recent experience illustrates the fact that the visceromotor reflex is a symptom distinct from the pain.

Observation 10.—Male, aged 48, consulted me on November 25th, 1905, for a pain he felt across the middle of his chest. He had felt a slight pain here for some months on walking up a hill. He is a master builder, and on this day

while watching his men at work, feeling cold, he started to help them dig up some earth to make himself warm. He did this for a quarter of an hour with a good deal of energy. He then examined several partially-built houses, running up and down a good many steps. On his way home he became conscious of this pain, and as it continued to increase in severity he called on me. I examined him carefully, and found a slight dilatation of the heart with an impure first sound. The blood pressure was 130 mm. Hg. On his way home the pain increased in severity, and after he reached home it became very violent, and my colleague saw him and prescribed opium, which relieved him. When I saw him next morning he gave a graphic account of his sufferings. He said: "In the tram coming home the pain got worse, and after getting home it became so severe that I felt I was going to die. The pain spread from my chest down my left arm to my little finger. You asked me, when I saw you yesterday, if I felt any gripping sensation, and I did not know what you meant; but, by George, I know now. When the pain was at its worst, I felt my chest suddenly seized as if in a vice, and I rolled on the floor in agony. The pain and the gripping eased off for a time and then came on again. This continued till I got the morphine. This morning I awoke all right, but at 10.30 that gripping sensation came on and held me tight for ten minutes. I dare not move for fear the awful pain should come, and I felt every moment that it was about to come, and I was in such terror of it that the sweat poured off me."

With rest and treatment these attacks got less, till now he only feels a slight pain when he over-exerts himself.

So far the symptoms I have dealt with have been mainly concerned with the reflexes connected with the sympathetic nerve supply. Equally instructive though less frequent symptoms can be shown to arise from stimulation of the vagus. At its centre in the medulla this nerve is in near relationship to the upper cervical nerves, and it would seem more particularly the sensory nerves supplying the sterno-mastoid and trapezius muscles. Not only can these muscles become extremely tender in various heart affections (see Observations 1 and 2), but the pain from heart affection may be felt in the region of distribution of the cervical nerves, as noted in Observation 4. The following observations also show the same thing.

Observation 11.—A man aged 62 years complained of great pain striking into his chest and behind his ears when walking. Thus in going to his work he was wont to allow seven or eight minutes to walk to the station, but now it took him over half an hour, as he had to stop on account of the pain every fifty yards. After accurately noting the pain, he described it as arising in the left breast, extending across to the right breast, seizing him in the neck, and extending up behind the ears, where it held him with great severity. In showing me the situation, he laid the fingers of both hands over the insertion of the sterno-mastoid into the mastoid process. On one occasion the pain extended from the breasts to the armpits and down the side of each arm to the elbow.

This patient dropped dead while at his work, and on *post-mortem* examination I found extreme calcareous degeneration of the coronary arteries.

Other Reflexes occurring during an Attack of Angina Pectoris.

Other very striking phenomena are sometimes met with during an attack of angina pectoris. During or after an attack an abundant flow of saliva and the secretion of large quantities of pale urine may occur; both symptoms I suggest are due to reflex stimulation of the floor of the fourth ventricle (see Observations 3 and 4).

Observation 12.—A man, aged 58 years, of gouty diathesis, complained of pain, induced often by the slightest exertion, which arose in the left breast, passed up the armpit, and extended down the inner surface of the left arm to the little finger. During an attack an abundant flow of saliva took place into the left side of the mouth. These attacks became so frequent that he could only walk a very short distance without inducing an attack. He dropped dead while sitting at his desk, and at the *post-mortem* examination I found that the heart had ruptured and the coronary artery was very atheromatous. Where the rupture had taken place the myocardia had nearly disappeared.

Observation 13.—Male, aged 46, whose work (a tackler) entailed periods of great muscular exertion, complained of pain striking into the chest when walking up a bill. If he stops as soon as he feels the pain coming on it passes off, but if he persists the pain increases to an agonizing severity and radiates into both arms, but worst into the left, as far down as the little finger. At the same time the chest is gripped so that he is forced to straighten himself and to breathe deeply, and at the same time his mouth fills with saliva and an aching pain is felt in the throat. A few minutes after each attack he has to pass urine, which is always abundant and very clear. These details I had from him after he had carefully noted a number of attacks.

Summation of Stimuli as a Cause of Angina Pectoris.

The fundamental functions of the heart muscle correspond to those of other involuntary muscles that form the walls of hollow organs; these functions being modified to suit its special work. Like the other viscera, the heart is insensitive when stimulated in a manner that provokes pain when applied to the tissues of the external body wall. I have pointed out that a prolonged strong contraction of a hollow organ can produce pain, and that this is undoubtedly the cause of the severe pain of renal gall stone and bowel colic. Can the heart give rise to pain in a similar manner? On account of the modification of its functions the heart cannot pass into a prolonged state of contraction. Immediately it contracts the function of contractility is at once abolished, and the muscle passes at once into a state of relaxation, and for this reason the pain cannot be produced by a "spasm of the heart." But I suggest that the heart muscle may produce pain when it is confronted with work greater than what it can readily overcome—a cause which produces strong peristalsis and pain in other hollow viscera. But the pain in the heart arises by a slightly different mechanism. A skeletal muscle will contract if a stimulus of sufficient strength be applied to its nerve. If the stimulus is too weak no contraction follows, but if this weak stimulus be frequently and rapidly repeated then the muscle contracts in accordance with the law of the summation of stimuli. I suggest that the heart muscle induces pain on the principle of the summation of stimuli. If we study such a case as is recorded in Observation 10 the pain was manifestly due to the man's unwonted exertion, but it did not attain its maximum severity till a couple of hours after the strain had been put on the heart. I could quote a large number of parallel instances, but for lack of space must be content here with throwing out the suggestion. I may say, however, that I submitted this view to Professor Sherrington, who replied that he thought my argument exactly right, and suggested several other cogent reasons in its support.

Presidential Address

ON

THE PAST, PRESENT, AND FUTURE OF THE BRITISH MEDICAL ASSOCIATION.

DELIVERED AT THE ANNUAL MEETING OF THE
METROPOLITAN COUNTIES BRANCH.

By HUGH R. KER, F.R.C.S. EDIN.,

PRESIDENT OF THE BRANCH.

THE first pleasing duty I have to perform on taking this chair is to thank you most sincerely for the great honour you have conferred upon me by electing me as your President. I can assure you I thoroughly appreciate the distinction; and though I feel how impossible it is for me to hope to compare favourably with my eminent predecessors, I gladly promise, trusting to your indulgence to overlook my shortcomings, that I will do my best to sustain the credit and high position of this Branch and to promote the interests of the great Association of which it forms so important a part.

I found it a most difficult task to select a subject suitable for my address to you, and I must ask your forgiveness if I succeed in bringing before you little that is fresh, and have again to traverse the paths which have been trod by many of my predecessors. After much thought and many doubts, I have taken for the title of my address, The Past, Present, and Future of the British Medical Association.

I did not know this title had ever been taken before, until I was looking up the records of former addresses, and found that it was the subject of Sir R. Quain's address in 1873.

On making this discovery I was at first inclined to choose another subject, but on second thoughts it seemed to me it would not be inopportune, considering the vast changes which have taken place in the position and constitution of the Association in the last thirty years, to review our position both past and present, and see if we