THE PHENOMENA OF ANGINA PECTORIS, AND THEIR BEARING UPON THE THEORY OF COUNTER-IRRITATION.

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In the following paper I shall endeavour to make an analysis of certain of the phenomena met with in cases of angina pectoris, with a view not only to throw some light if possible upon the explanation of angina, but also to carry the process somewhat further, and through it to seek for a solution to certain other problems in nerve physiology.

I will take as my text the description of his attacks given me by a patient who suffered from very typical attacks of angina. This patient, who had no perceptible organic heart-disease, described his attacks as consisting of a sudden severe gnawing pain at the apex of the heart, shooting through to the bladebone and down the left arm to the tips of the fingers, where he felt a strong tingling sensation. The pains were accompanied by palpitations of the heart, and towards the end of the attack by the belching of a quantity of wind. After the attack, there was soreness and tenderness of the skin of the left side corresponding to the seat of the pain. The attacks lasted from five to fifteen minutes. Let us endeavour to ascertain the meaning of this series of phenomena.

In the first place, what in physiological language is the explanation of a sudden severe attack of pain? It means that the person is conscious of an abnormal commotion in the grey matter of the sensory region of the brain, corresponding to the region of the body where the pain is felt. This commotion may have arisen spontaneously in this part of the brain, as is probably the case in most cases of epilepsy associated with sensory phenomena; or it may be due to the transmission

upwards of the results of a commotion in the grey matter of one of the subordinate parts of the nervous system, as for instance in the sensory grey matter of the spinal cord, or, lower still, in the grey matter of the sympathetic ganglia. With these, again, it is also possible that the commotion began spontaneously; or, on the other hand, they in their turn may only be passing onwards a commotion which has begun still further away from the centre, viz. (1) in the trunks of the sensory nerves, or (2), far more frequently at their peripheral extremities, in both of which situations the commotion is rarely spontaneous, but is almost invariably due to something outside the nerve or its extremity, which has brought the nerve into action.

Thus, then, an attack of pain localising itself in the heart may be due in theory to any one of the following causes:—

(1.) Some structural change in the heart substance, or some change in the heart contents, acting upon the peripheral terminations of the cardiac nerves.

(2.) A commotion arising spontaneously in the grey nervous ganglia of the cardiac plexuses of the sympathetic, the commotion being transmitted upwards to the spinal cord and brain.

(3.) A commotion arising spontaneously in the cervical ganglia of the sympathetic which give off branches to the cardiac plexus; or in the ganglia of the pneumogastric.

(4.) A spontaneous commotion in those portions of the grey matter of the spinal cord which are connected with these ganglia by the bands of communication passing from the cord to the sympathetic.

(5.) A spontaneous commotion in that part of the grey matter of the brain which in any of the previous cases would be the recipient of the nervous impulses passed up from below.

These, then, are the various conditions upon which the pain may in theory depend It would be interesting to inquire whether all of them are in practice met with. This, however, would require a long and difficult examination of the evidence derived from a large number of carefully observed cases. I am not sure whether in the present state of our knowledge

such an inquiry is likely to lead to satisfactory results. At any rate, for my present purpose it will be enough to say that in most cases of angina associated with structural disease of the heart or large vessels the primary irritation probably takes place at the peripheral extremities of the cardiac nerves; but that there is some evidence to show that there are varieties of angina beginning spontaneously in the nerve-centres. I shall say little, moreover, as to the *nature* of the commotion set up in the grey matter, beyond stating that it is probably closely allied to that met with in cases of epilepsy, and still more so to that present in the so-called epileptiform neuralgia. The relationship between epilepsy and angina pectoris was long ago pointed out and insisted upon by Trousseau.

Let us pass on to consider another feature of the attack: the pain having begun suddenly in the neighbourhood of the apex of the heart, shot through to the left bladebone, to the left shoulder, and down the left arm to the tips of the fingers.

The radiation of the pain from the limited district in which it began until it extended over a wide area is an evidence of an extension of commotion from one small patch of grey nerve-substance to other parts of grey matter more or less intimately associated with it. An extension of nerve commotion of this kind is a very familiar phenomenon to those who have carefully studied the phenomena of epilepsy. When an epileptic fit instead of destroying consciousness at once begins slowly with the production of an elaborate warning, it is no rare thing to find that a nerve commotion giving rise (say) to a tingling in one finger, gradually spreads until the tingling passes to the other fingers, and thence up the arm to the trunk; thence it may pass up the neck to the tongue, face and head, or downwards to the thigh, leg and foot; bright sparks may perhaps next be seen, or a noise heard in the ears. Then a few muscles may begin to twitch in the hand; the arm may next be drawn up, and at this point perhaps the patient loses consciousness, the commotion extending rapidly until the whole body is convulsed. These various sensations and phenomena following in regular succession do but mark

the extension of the original commotion over a wider and ever more widely extending area of grey nerve substance. This extension depends upon the intimate association of one nerve-cell with others, either in its immediate neighbourhood, or closely connected with it in function, an association established by means of commissural interpolar fibres passing from cell to cell. When one of these cells becomes abnormally excited, as in the case of a slowly beginning fit, the commotion makes itself rapidly felt in the other cells connected with it. These will tend in consequence to become similarly excited; and they, in their turn, will excite others still further removed from the first cell, and so on in endless progression.

So it is in angina pectoris. Whatever may have been the original cause of the commotion, the excitement having once been started in some one of the situations above mentioned, will tend to overflow from the original focus into neighbouring parts. But now comes the question, Why does the excitement adopt the peculiar mode of extension seen in the case of angina pectoris I have quoted, and which, as all will allow who have seen much of the disease, is a very common form of attack? Why, in other words, does the pain extend from the heart region down the arms, and down the left arm rather than the right? What possible nervous communication can there be between the heart and the arms, since the heart is supplied by the pneumogastric and sympathetic nerves, and the arms by the lower cervical spinal nerves?

It is at once evident that no intercommunication between these two regions can take place through the cardiac ganglia, nor through the ganglia of the sympathetic giving origin to the nerves which pass to the cardiac ganglia. Neither do the pneumogastric nerves or their nuclei of origin offer a means of intercommunication. Does the spinal cord offer a more probable solution to the problem? The cardiac nerves of the sympathetic come from the three cervical ganglia on both sides. Of these, the largest nerves are the two nerves which come from the middle cervical ganglia. The strands passing from these ganglia to the spinal cord pass in the trunk of the fifth and sixth cervical nerves; those passing from the inferior cervical ganglia, in the trunks of the seventh and eighth

cervical nerves. It is these four nerves which, in conjunction with the first dorsal, form the brachial plexus. We thus see that the region of the spinal cord which gives origin to the brachial plexus gives origin also to the greater part of the fibres which eventually find their way to the heart. Wherever, then, the original commotion may have taken place in an attack of angina pectoris; whether in the cardiac ganglia, or in the cervical ganglia of the sympathetic, or in the spinal cord; and whether the commotion be due to some peripheral irritation from disease of the heart's substance, or be a spontaneous outburst on the part of the nerve-cells implicated, it is evident that it is only when the commotion has begun in the cord, or has passed up to the grey matter of the spinal cord from the sympathetic, that any great extension in its area can take place such as that of which I am speaking. The next question is: Since the cardiac nerves come from the sympathetic on both sides of the body, and must consequently be connected with both sides of the spinal cord in an equal or nearly equal degree, how is it that in the vast majority of cases of angina pectoris, as in the case mentioned above, it is the left arm alone that is affected, and that affection of the right arm alone is so exceedingly rare, although in some few cases both arms may be involved? The answer to this question is of very great interest, for on it, I believe, depends the explanation of the theory of counter-irritation.

It is well known that in the grey matter of the spinal cord, in addition to the fibres passing from the nerve-cells to form the roots of the spinal nerves, and to those which pass upwards to the brain, there is an extensive system of commissural fibres connecting the two sides of the cord, and connecting also cells and groups of cells together. It is, indeed, upon this intercommunication of groups of cells that reflex action depends. It is no less certain that cells endowed with a similar function, as, for instance, that of motion, but connected with different regions of the body, are more or less intimately associated together. It might at first sight be thought that this grouping of cells of similar function would coincide strictly with the grouping of the fibres emanating from these cells to form nerve trunks, e.g. that the cells whose emerging fibres go to

form the motor part of the musculo-spiral nerve in the arm would form one group; and that those whose fibres go to form the motor part of the musculo-cutaneous nerve would form another. Similarly it might be expected that the cells governing the sensation of those parts of the skin of the arm supplied by the musculo-spiral nerve would be associated together; and that the same would be true of those governing the sensation in the skin supplied by the musculo-cutaneous nerve.

Now it has been shown by Remak, Erb, Ferrier, and others that this is not the case with the *motor* nerves, but, on the contrary, that the grouping of motor cells in the spinal cord depends on the physiological association of muscles in the production of certain movements, and not on the mere anatomical fact of their being supplied by the same nerve-trunk. In a paper I read before the Medical Society of London,[1] I narrated two cases proving the truth of this proposition most conclusively. The patients were suffering from progressive muscular atrophy, involving the arms. In one of them the only muscles involved were the biceps, the brachialis anticus, and the supinator longus—the three muscles, in fact, which are associated together in bending the elbow. In the other case, all the muscles of the arm and hand were completely atrophied *except* these three muscles. Now two of these muscles are supplied by the musculo-cutaneous nerve, and the other by the musculo-spiral.

If this is so with the *motor* functions of the cord, we might expect to find somewhat the same variety of association in connection with the *sensory* functions. In this case we should expect that the cells connected with the sensation of the upper arm, taken as a whole, would be associated in a group together, in spite of the variety of nerves distributed to the skin of this part; whilst, on the contrary, the cells governing the sensation of a scattered district supplied by one nerve, such, for instance, as that supplied by the musculo-spiral nerve, would belong to several groups, rather than be collected into one group. In other words, cells governing the sensation of neighbouring parts of skin would be more or less closely

[1] 'Proceedings of Medical Society,' vol. v.

connected with one another; whilst those supplying patches of skin at a distance from one another would be correspondingly separated.

But if this be true of neighbouring parts on the *surface*, we may expect that it will be equally true of parts which, like the skin and the structures lying *beneath* it, are contiguous to one another; thus we should expect to find that the centres controlling the sensation of any particular portion of skin would be intimately associated with those governing the sensation of the subcutaneous fat, of the muscles, and even possibly of the bones, lying beneath that area of skin. Similarly, that the sensory centres for the skin over any particular organ would be associated more or less closely with those controlling the sensory functions of the subjacent organ.

This theory of association of cells in the spinal cord, according to physiological requirement and correlation, has, as I have already said, been proved to be true in the case of the motor functions, and I believe that the distribution of the pain in angina pectoris proves its truth in connection with the sensory functions.

Let us for the moment grant that it *is* true, and let us inquire what method of distribution of pain we might expect to find in connection with an attack starting in the nervous apparatus of the heart or great vessels, and spreading.

It would first attack the part, superficial to the heart, or great vessels; that is to say, the sternum and ribs and their muscles, and the skin covering them. Having reached the skin, it would tend to spread from one part to another, but this spreading would be more likely to follow certain directions than certain others; for instance, as we have followed the majority of the sympathetic cardiac nerves into the cervical enlargement of the spinal cord, we may expect to find that the pain will be confined to the upper extremities, rather than that it would pass from the skin of the thorax to that of the abdomen. Again, the skin of the left side of the thorax would be much more affected than the right side, because of the situation of the heart to the left, and therefore as the extension of the pain goes on, it will be the left arm that will be chiefly involved. At the same time we should expect that in

severe cases the radiations would cross to the right side, and that the right arm might be affected, though rarely, if ever, to such an extent as the left. We should also expect that it would be exceedingly rare to find that the pain passed to the right arm without affecting the left.

But the original commotion in the cardiac centres will not only affect those parts of the nerve-centres governing the sensations of the front part of the heart or its large vessels, but all parts will partake in it. Consequently the radiation to contiguous districts will take place not only towards the front, but in other directions also. Hence we may expect to find that the patient will complain of pain in the left scapular region, whence also the way is not far to the left arm. The radiation downwards would tend to pass to the stomach, and we might thus look for some affection of that organ. We should be prepared to find also that there would be more or less affection of the lungs.

Let me narrate very briefly the account given of their attacks by a number of different patients. It will be remembered that the patient who has served as the text for this paper experienced a severe gnawing pain near the heart's apex, which extended over the whole cardiac region, shot through to the left bladebone, and down the left arm to the fingers. At the end of the attack he belched a quantity of wind. The attack left a soreness and tenderness in the skin of the left side of the chest. Another patient of mine, suffering from severe aortic valvular disease, had attacks in which he felt a sudden pain beginning over the heart, going back to the left shoulder-blade, and down the left arm to the finger tips, where he had a strong sense of tingling. Sometimes the pain extended also to the right shoulder and down the right arm, but here it never extended farther than the elbow. At the end of the attack, he belched a quantity of wind for a few minutes. Occasionally he suffered from shortness of breath during the attacks

Another of my patients suffering from aortic valvular disease had attacks which began with a sudden choking sensation at the top of the sternum, with intense dyspnœa; this was followed by intensely sharp pain over the apex of the

heart, which extended to the left shoulder and down the left arm, which felt numb and heavy. After a few minutes he began to belch a large quantity of wind, the eructation continuing for some time.

A fourth patient of mine, a woman without evident organic heart-disease, had attacks, almost always at night, in which she had sudden severe pain in the cardiac region of a shooting or dragging kind, which went down both arms, but down the left more than the right. The hands tingled, and she felt as though the nails were being dragged out. Towards the end of the attacks she belched a quantity of wind. After the attacks the arms felt numb and weak.

Trousseau describes the following cases:[1]—

(1.) A woman with aneurism of the aorta had attacks of sharp sudden pain in the precordial region, radiating to the base of the chest, where it produced an intense sensation of constriction; thence to the loins, up to the neck, down the left arm to the extremities of the fingers, leaving the arm numb and heavy.

(2.) Another woman, without organic heart-disease, had pain behind the sternum, passing to the left shoulder and down the left arm, causing numbness.

(3.) A man without organic disease had pain behind the lower part of the sternum, passing to the base of the neck and down both arms equally, giving rise to painful numbness.

(4.) A man with aneurism of the aorta had attacks beginning with a sense of suffocation, followed by severe pains behind the sternum, passing to the left shoulder and down the left arm.

(5.) A man without organic disease had attacks of pain behind the sternum, accompanied by intense dyspnœa. The pain passed to both arms, but chiefly to the left, giving rise to numbness. The attack ended with a strong desire to pass water, and with a sense of congestion in the nasal mucous membrane.

In the majority of these cases, then, we see that the attack, beginning in the cardiac region, passed to the left arm alone; in three it passed to both arms; but in only one was the right arm affected equally with the left. In no case was the right

[1] 'Clinique Médicale,' 4ème édition, vol. ii. p. 527 *et seq.*

arm alone affected. Trousseau mentions a remarkable case in which the right arm was affected and not the left; but in this case the pain was entirely confined to the *right* side of the chest, and hence can scarcely be looked upon as one of ordinary angina pectoris, the case being probably one of epileptiform neuralgia of certain intercostal nerves of that side.

What evidence is there to show that the pain in the cardiac region was situated in the skin, covering that region as well as in the heart or its dependencies? An answer to this question is of importance, if my theory be correct that the pain passes from the deep organ to the superficial structures over it, and thence spreads along the skin to the arm, &c.

In the first place, I would quote the statement of my first-mentioned patient, who said that the skin over the heart was sore and tender to the touch after the attacks. Does not this recall at once the tenderness over a neuralgic district; and, on the other hand, if the intercostal nerves had been unaffected, why should the skin be tender? Again, Trousseau describes the case of a man with an aortic aneurism subject to attacks of angina pectoris, who after his attacks had *numbness of those parts of the chest where he had experienced the pain.*

In the next place, I would quote the description so often given by patients, and upon which Trousseau and other authors lay so much stress, viz. that even where there is no actual dyspnœa, the patient ceases at the height of the attack to draw breath, or draws it as lightly as possible, on account of the pain that it gives him to do so. Does this description not resemble exactly the condition of a patient with severe intercostal neuralgia—severe pleurodynia?

It will be seen that in several cases the patients made special mention of pain in the bladebone following on that in the precordial region. Walshe speaks of "pain shooting to the mid-dorsal spine" as not infrequent. This would appear, therefore, to be a radiation independent of that over the præcordium, and if this be so we should be justified in referring it to an extension from that portion of the cardiac centres of the spinal cord governing sensation in the back of the heart.

In all my cases gaseous eructation from the stomach

was a marked feature, and most authors who have treated of angina mention it as a frequent symptom. This is the most usual form of implication of the stomach, and it is one of very great interest, as proving conclusively the influence of the nerves of the stomach over the secretion of gas in that organ. Just as the connection between the heart and its superficial neighbours is evidently established through the sympathetic nerves passing to the cervical spinal cord, so here the connection between the heart and its deep neighbour the stomach is probably made through the pneumogastric nerves. If therefore there be a form of angina having its origin in the cardiac ganglia, and another form having its origin in the centre in the spinal cord controlling the heart, as is by no means improbable, we should expect that the former variety would be accompanied by stomach disturbance, and that the latter would not. The materials at my disposal are not sufficient to enable me to treat further of this point; but it will be an interesting question to ascertain whether the anomalous cases of angina not connected with organic cardiac disease, and not following the more usual methods of distribution (some of which cases may be supposed to arise in the spinal centres), present the symptom of eructation as frequently as the more typical cases of angina, or those definitely connected with heart-disease. This symptom of gas secretion under purely nervous influence is a most curious one, and worthy of further examination. I do not propose, however, to go further into this branch of the subject on the present occasion, beyond calling attention to the probable connection between the gaseous eructation of angina pectoris and the remarkable eructations of gas from the stomach in certain hysterical patients; the well-known phenomena of phantom tumour; the eructations which follow the fit in some cases of epilepsy; and probably also certain forms of flatulent dyspepsia in neurotic subjects.

As regards radiation of the nerve-commotion from the cardiac nerve-centres to those of the lungs, this is less frequently seen. In one of my cases dyspnœa was a prominent feature, and it was so, moreover, in two of the cases narrated by Trousseau. Walshe says that real dyspnœa is exceedingly

rare in angina pectoris; but in my own case above mentioned, there could be no doubt of the presence of real and most urgent dyspnœa.

The infrequency of lung symptoms may be taken, I think, as an indication that the affection of the stomach in angina pectoris is due to the close connection of the cerebro-spinal centres controlling the heart with those controlling the stomach, and that this connection depends upon the close proximity of the organs in the body, rather than upon the fact that both organs are supplied by the same nerves. Were it true that the almost constant association of symptoms in the heart and stomach in angina pectoris is due to the latter cause, it would be difficult to explain why the lungs, which also derive a large part of their nerve supply from the same nerves, are so very much less frequently affected. If, however, the explanation I have offered be the true one, then we should expect that, as by far the larger part of the lungs is situated at a distance from the heart, the communication between the heart and lung centres in the cerebro-spinal axis would not be particularly close, and that the lungs would only be affected in special cases.

In some of the more severe cases the radiation is much more extensive than to the back, left arm, stomach or lungs. We have already seen that it may go to the right arm; but it may also go up to the head down the trunk, and even to the lower limbs. In one of Trousseau's cases above described it affected the bladder and the nasal mucous membrane.

One of the most instructive phenomena of radiation to my mind is the sense of intense constriction, which is occasionally complained of. One of Trousseau's patients described it as being like a bar of iron violently tightened round the chest. Does this not at once recall to our minds the *douleur-en-ceinture* of spinal-cord disease which we have every reason to believe to be due to the involvement of the sensory centres of the intercostal or lumbo-cutaneous nerves? Its presence in angina pectoris is a fresh proof, if such be required, that wherever in angina pectoris the original irritation may have been, the seat of the commotion of which the patient is conscious is in the spinal cord. It shows, moreover, the inti-

mate connection between the spinal centres of deep organs, and those for the superficial structures covering them; for there is much less evident nervous connection between the cardiac ganglia and the intercostal nerves, than between these ganglia and the arms; yet we see that a commotion in the spinal cardiac centres may propagate itself rapidly to the centres from which emanate the sensory fibres of the intercostal nerves.

And now for the bearing of these remarks upon the theory of counter-irritation. When we use the actual cautery to the skin over the spines of the vertebræ in a case of disease of the spinal cord, what are we doing? We are producing a profound impression upon the peripheral endings of the nerves of the skin over the diseased part. We produce, moreover, a moderate degree of inflammation in the skin and parts immediately subjacent to it; but considering the depth at which the diseased part lies below the surface, we cannot imagine that the direct effect of the burn reaches so far inwards. Neither is it possible to suppose that the small amount of blood drawn to the skin by this slight degree of circumscribed inflammation can modify in any perceptible degree the amount of blood in the deeply subjacent organ. Can we not from our knowledge of what takes place in angina pectoris deduce an explanation of the effect of counter-irritation? An analysis of the phenomena of angina pectoris showed the intimate relationship which exists between the nerve-centres governing adjacent structures. We saw there how an irritation at the periphery of the nerves governing the deeply-seated organ, an irritation arising from heart-disease, aneurism of the aorta, &c., caused a commotion in the spinal centres with which those nerves are connected; that this commotion rapidly passed to centres from which emanate the nerves supplying the parts superficial to that organ, and that in these centres also a violent storm arose.

Can we not easily imagine the reverse; that an irritation of the peripheral endings of the nerves supplying the superficial structure, should set up a commotion in the spinal centres to which these nerves pass; that this commotion should extend to the closely-related centres governing the

subjacent organ or structure, and should profoundly affect this structure?

As a matter of strict fact this reverse process does sometimes take place in connection with the heart. Let us refer once more to Trousseau's masterly lecture on angina pectoris. One of his patients without organic heart-disease, but with a gouty history, had attacks *beginning in the left arm* and passing upwards to the chest, where the pain was chiefly confined to the cardiac region; here the pain was so intense that it seemed to the patient himself as though he must die. The attacks lasted three minutes, and in all points resembled attacks of angina pectoris, with the exception that the radiation of the pain took place in an inverse order. Another patient, without organic heart-disease, but who was also gouty, had attacks beginning in the muscles of the left arm, and thence passing to the cardiac region. In a third case the pain began in the two shoulders, and rapidly passed to the neck, to the tongue, to the arm, and to the chest. Here also there was gout, but no organic heart-disease.

In a fourth case the pain began in one of the dorsal vertebræ. It passed thence to the left arm, and finally to the cardiac region. No organic heart-disease was discovered in this case.

These cases, it is true, were not traceable to any morbid condition of the peripheral ends of the nerves of the arm, the primary commotion probably taking place in the spinal centres governing the sensation of the arm; but they serve to show how readily a commotion in these centres passes to the centres governing the cardiac region; and if a commotion beginning spontaneously in the spinal centres for the arm can thus so readily be transmitted, a similar commotion of these centres, induced by peripheral irritation, would do so with equal ease.

But if I am correct in believing that the evidence derived from an analysis of the phenomena of angina pectoris and allied conditions points to an intimate communion in the spinal cord between the sensory centres for superficial parts, and those for subjacent organs or other structures, ought we

not to find corroborative evidence of this fact in other physiological or pathological conditions?

It is less easy than might at first sight appear to get such evidence; since severe superficial localised lesions, not in themselves affecting deeper structures, are not very common. The following conditions must not, however, be passed over, as at any rate suggestive of an action of this nature.

(1.) The occurrence of deep-seated inflammations from surface chills. In most instances of this kind it is quite impossible to believe that the organs themselves are affected by the chill, as for instance when the kidneys, wrapped up as they are in their thick coatings of fat, &c., take on acute inflammatory action after exposure to a cold wind. In these cases the inflammation is probably produced through the mediation of the nervous system; and there can be little doubt that the peripheral sensory nerves take an important part in the process. As, however, it is very exceptional for the initial chill to be local in its action, it is difficult to be sure that the deeply-seated inflammation has been induced by the action of the chill on the overlying parts. We, nevertheless, tacitly by our action, admit the probability of this connection; for the man with a weak chest is warned to take especial precaution in wrapping up his chest; the man with delicate kidneys is made to wear a flannel-band round the abdomen; "cholera-belts" are served out to troops in warm climates, and so on.

There is one instance of a deep-seated inflammation occurring from a local chill which is, I believe, quite confirmatory of the views above stated. I refer to the inflammation of the facial nerve in the Aquæductus Fallopii resulting from exposure of the cheek to a draught. It is difficult to believe that the nerve lying deeply in and beneath the parotid gland can itself be sufficiently affected by the chill to induce inflammation. The inflammation is probably strictly analogous to that met with in other deeply-seated structures as the result of chill; and in this case the relation between the seat of the chill and the inflammatory effect is a very definite one. I am aware that I am entering upon debatable

ground, as to the respective parts taken by the central nervous system and sympathetic and vaso-motor systems in the production of inflammation. Nevertheless, I would offer these as suggestions, in the hope that those more competent than myself to deal with these difficult questions may be able to throw further light upon the subject.

There is one curious fact which I would mention in this connection, viz. the occurrence of duodenal ulcers in cases of surface burns. Having no good medical library within reach, I am unable to ascertain in what class of burns these ulcers have been chiefly met with. If, as I believe it has been stated, they occur in connection especially with superficial burns of the abdomen, may we not look upon their occurrence as another instance of the effect upon deeply-seated organs of superficial lesions over the organ?

(2.) I now pass to another point. If the radiation of the pain in angina pectoris, and the effects of counter-irritation over deeply-seated morbid structures, be both of them due to sensory co-ordination in the spinal cord, can we, by a study of the former, ascertain any facts which may help us in understanding the modus operandi of the latter?

It is exceedingly common to find in patients subject to attacks of angina pectoris extending to the left arm, that the arm remains in an abnormal condition for a longer or shorter time after the attack is apparently over. The most usual description is that the arm is numb—the sensation to touch is dulled. Frequently also there is a subjective "numb-feeling," i.e. not only is the sensation to touch dulled, but the patient has the positive sense of numbness—as one of my patients described it, a "buzzing" in the limb, because it seemed to him so allied to buzzing in the ears. Then again there may be the opposite condition to loss of sensation, viz. pain; either spontaneous, or developed by pressure—soreness. Thus, one of Trousseau's patients suffered from "painful numbness" in the left arm after the attacks; and on the other hand the patient who served as my text, had soreness and tenderness in the left side after his attacks.

(3.) A third variety of after-effect is the sense of weakness or "heaviness" that some patients experience in the arm. And

lastly, in one of Trousseau's patients the left arm became pallid, and subsequently of a bluish tinge.

Let us translate these various descriptions into their physiological equivalents. Loss of sensation in such a case means that the sensory nerve-centre implicated (in all probability the spinal centre) is exhausted by the nerve storm through which it has passed to such an extent, that it is no longer capable of acting fully under ordinary stimuli; and observe, that in not a few cases it is the centres to which the storm has passed from the primary source of commotion which give evidence of this great exhaustion.

The positive sensation of numbness, the "buzzing" in the limb, must mean that the commotion does not at once die down, but goes on for a time in a quiet manner; sufficient to attract the patient's attention, but not enough in most cases to amount to actual pain. The positive sensation is generally accompanied by more or less exhaustion of the centre, as is shown by the presence simultaneously of dulness of sensation to touch.

Soreness or tenderness mean that the centre is not exhausted, but is, on the contrary, left by the attack in an irritable condition, so that ordinary stimuli produce an over-action in the centre. The sense of weakness or heaviness in the limb shows that in those cases in which it is present, the whole nervous apparatus of the limb is for the time disorganised, the motor functions suffering as well as the sensory. It would be interesting to enquire into the mechanism by which this modification in the motor functions is produced, but I do not propose to do so in the present paper.

In order to apply the above rough accounts of what probably takes place, so as to give a full explanation of the action of counter-irritants, it would be necessary to have a clear knowledge of the influence of the central nervous system upon the production of inflammation. My knowledge of this branch of the pathology of inflammation is insufficient to enable me to do this, but I would make the following suggestions:—

(1.) Whatever may be the influence of the nervous system over inflammation, it is probably an *over-active* or *irritative* influence rather than the contrary.

(2.) Two methods suggest themselves to combat this over-activity. In the first place, we endeavour to soothe it; but failing in this, we endeavour to tire out the centre more rapidly than it would tire of itself.

(3.) Since in the case of deeply-seated inflammations we cannot act directly on the centre by stimuli, we act upon those centres with which it is intimately associated; stimulating these powerfully, in the expectation that they will pass on the commotion induced in them to the centre upon which we wish to act.

(4.) The centres most intimately associated with that governing the inflamed part are those controlling the tissues in the neighbourhood of the part, and hence we stimulate the surface with a view to acting on the organ lying beneath it.

(5.) The centre controlling the inflamed part being thus goaded to further action, rapidly becomes exhausted, and thus its irritative action ceases.

(6.) We have seen that even after so severe a commotion as that present in an attack of angina pectoris the centres are not always exhausted to the point of ceasing to be excitable, but on the contrary are occasionally left in a state of irritation. I believe that the analogue of this is now and then met with in counter-irritation. A patient was under my care suffering from long-standing spastic paraplegia, associated with spinal pachymeningitis. The actual cautery was freely applied on several occasions with great ultimate benefit, but the first effect of the cautery was to increase markedly the spastic symptoms (i.e. the symptoms of irritation), which after a few days diminished, until they were considerably less than before the application.

(7.) I have hitherto said nothing of the vaso-motor nerves, which play so important a part in inflammation. Without pretending to have much knowledge of the mechanism by which the vaso-motor nervous system acts in inflammation, I believe I shall be correct in saying that the influence of the central nervous system on this process is largely exerted through the mediation of the vaso-motor nerves, and the action of the latter has to some extent been presupposed in speaking

of that of the former. The profoundly intimate connection between the central nervous system and the vaso-motor system is a matter of every-day observation. In this connection the observation of Trousseau already mentioned is of great interest, viz. that in one case where the pain of angina pectoris radiated to the left arm, the arm became first pallid and then bluish. This indicates most clearly the close connection that subsists between the spinal sensory centres for a region, and the vaso-motor centres for the same region.

In the above account I am aware that I have touched upon much debatable ground; where, moreover, a far greater knowledge than I possess would be necessary to do full justice to the subject. It is quite certain that much has yet to be learnt in connection with many of the points I have mentioned, and my object in writing this paper has been to offer a few hints as to the directions in which investigation is likely to lead to successful results.