ABDOMINAL PAIN.*

BY

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As one of the individuals deputed to open this discussion on abdominal pain I warmly appreciate the honour, greedily claim the privileges, and reluctantly assume the responsibilities of the task. The honour is great in direct proportion to the names of those who precede and follow me, and in this instance, therefore, is one of high degree. The privileges entitle me not only to a first and last word, but to the choice of any aspect of the subject under discussion on which to base my opening remarks. The responsibilities lie in raising questions and making dogmatic statements such as will invite criticism and provoke attack.

Pain has always played an important part on the stage of human life. In the eyes of the moralist its chief function has been that of a corrective; to the physiologist its interest lies in its influence as a protective agent; to the man of medicine its share in promoting the healing of wounds and in the detection of disease makes its widest appeal.

In this assembly we may expect that more attention will be paid to pain as a noxious stimulus or an aid to diagnosis than as an instrument of punishment. On the other hand, the usefulness and uselessness of physical suffering constitute a theme so large and so full of difficulties and dangers that we may well leave its philosophic consideration out of to-day's programme.

The increment of knowledge which has accumulated during the last fifty years concerning the various functions of the nervous system, and particularly in regard to afferent and reflex mechanisms, has led us to understand much about the origin of pain in all parts of the body, and has encouraged us to look upon and recognize it as a valuable asset for purposes of diagnosis. We have learned that the threshold of pain, under normal conditions, is higher than that of other forms of sensation, and, when passed, leads not only to recognition by the higher centres, but to other phenomena which are more or less outside the control of the will. Urgent motor responses, such as the withdrawal of the leg when a noxious stimulus is applied to the sole of the foot, or the active lacrimal secretion associated with a painful injury to the surface of the eyeball, are familiar examples of such reflex phenomena within the sphere of action of the ordinary spinal and cranial afferent nerves.

On the other hand, the territory of the sympathetic nervous system presents problems which are fundamentally different. Here we are faced by an involuntary musculature which carries on its activities in health without intruding upon consciousness, and by viscera which can be maltreated in many ways without the production of pain. These viscera are protected from outside injury by one line of defence, the abdominal parietes, which reacts to painful stimulation as do other parts of the surface of the body. But many of these viscera are hollow, open to attack from within, and dependent on a special nervous mechanism of their own to maintain their integrity and to preserve their functions. The chief menace to the latter is provided by disease or deformity of the visceral walls, or by the presence of obnoxious and abnormal substances within their cavities.

The abdominal wall is designed to protect the viscera against external trauma or against morbid processes originating in its constituent tissues by means of an afferent and reflex mechanism of the ordinary cerebro-spinal type, with pain, aroused by various familiar stimuli, as its vigilant outpost. The daily experience of frequent attacks, or of threatened attacks, of greater or less seriousness, upon the abdomen, raises this mechanism to a level

high enough for discrimination and localization to be added to mere reflex response.

On the other hand, the hollow viscera are provided with a nervous mechanism of sympathetic origin which usually carries on its functions quietly, unobtrusively, and undisturbed by the outside world. Variations from the normal routine are rare events, generally insufficient in degree to produce or necessitate more than passing notice, easily met without any call for help on the organism as a whole, and too trivial to require any special discriminative or topographical recognition on the part of the higher cerebral centres.

But exceptional and untoward events may happen even in this tranquil territory. Obnoxious strangers are introduced or appear from unexpected quarters, and their ejection is not only imperative and urgent, but demands unaccustomed effort and perhaps the help of auxiliary forces. The alarm is signalled through the agency of unusual afferent impulses, the spasmodic efforts of the involuntary musculature of the visceral walls are reinforced by skeletal muscles summoned to its aid, and the clinical picture of colic is evolved. Such, broadly, is the physiological story of a bedside incident familiar to all who practise medicine and surgery.

Pain is the outstanding feature which overshadows all else in this picture, and it is to the origin of this pain that so much research has been directed of recent years. Its indefinite localization, its intermittent paroxysmal character, the effortful and doubled-up attitude it provokes—all serve to proclaim its distinction from the localized, continuous, and immobilizing pain associated with disease of, or injury to, the abdominal wall, including in the latter term the parietal peritoneum.

Splanchnic pain is something very real, at any rate to anyone who has experienced or witnessed the tortures of colic, and yet we know that the abdominal viscera are quite insensitive to ordinary painful stimulation, such as section by knife or piercing by needle. For this reason we may assume an absence of true pain receptors in the visceral walls and attribute the production of pain to stimulation of nerve fibres. This is the more likely in view of its high threshold, its radiative and its non-discriminative characters—features characteristic of the pain resulting from the irritation of spinal nerve fibres in other parts of the body. There is a good deal of evidence to show that stretching the visceral tissues can provoke pain, and there can be little doubt that most examples of splanchnic pain are associated with unusual motor activity or muscular spasm. More speculative, however, must be any hypothesis attempting to describe exactly the form of stimulus which provokes pain and the precise nervous structures on which it acts.

Another question, too, awaits a clear answer, and that is the order of events which give rise to pain. Take, for instance, the case of a small calculus impacted in a ureter and attacks of colicky pain at long intervals. What initiates at any particular moment the paroxysms of pain referred at first to the penis and spreading all over the lower part of the abdomen? Is the afferent impulse which arouses the muscular spasm in the wall of the ureter a painful impulse, or is the pain only evoked by the raising of tension consequent on the muscular spasm?

Finally, we would welcome a clearer insight into the variations which, it seems, must occur in the threshold of painful sensibility under conditions of health and disease. Damage or disease of superficial tissues in the territory of spinal nerves will undoubtedly lower their threshold of pain, and it may be justifiably inferred that ulceration within a hollow viscus may bring about a similar result. Of particular interest from a clinical point of view is the length of time during which a lowering of the pain threshold may persist after a structural abnormality has been removed or all evidence of its presence has disappeared. An answer to this question might throw much light on the problem of certain chronic abdominal cases which are alike the despair and the livelihood of surgeons. We are inclined to attribute the hypersensibility of such patients to some alteration of mental attitude, to hide our ignorance behind a diagnosis of psychalgia or functional pain, always casting suspicion on the higher levels of the nervous system. This may be an

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unfair interpretation of symptoms which are difficult to understand, and we might consider the possibility of deranged function at a lower level—a sympathetic level—being responsible for a threshold of pain so depressed as to make many stimuli, innocuous under healthy conditions, assume noxious habits and qualities.

There is some support for such a view to be obtained on the clinical side. The patient who develops chronic abdominal pain after an abdominal operation, and who is prepared to undergo a relay of further operations for its relief, is often not of the neurotic type in the first instance. She does not respond to, or rather does not recover under, psychotherapy. Time, uninterrupted by much medical or any surgical treatment, may bring about complete recovery, unless fear and impatience combine to produce a psychoneurosis. After all, we are familiar with localized areas of hyperaesthesia in the distribution of the peripheral nerves after injury or sepsis, areas which may be exquisitely sensitive to all stimulation for months and years, and which may yet gradually approach a more normal condition if left well alone.

On the other hand, painful intestinal spasm of a paroxysmal type is a somewhat rare but very definite symptom in anxiety states, and may give rise to much difficulty in diagnosis, as well as to needless laparotomy. Such instances as I have met with have occurred in profoundly neuropathic patients, and have only yielded to psychotherapeutic measures. They are interesting from a physiological standpoint because they are examples of severe colic without any discernible local lesion. The intense pain and resulting tenderness, sometimes accompanied by a palpable knuckle of contracted gut resembling what can be felt in a cramp of a skeletal muscle, strongly suggest that splanchnic pain is chiefly due to muscular spasm, and may be independent of any morbid process either in the intestinal wall or in the bowel contents. Whether the pain is the result of pressure exerted on the intrinsic nervous structures, or due to interference with their blood supply, is a question which has yet to be answered. The early intermittent colicky pains associated with mesenteric thrombosis and embolism bring the circulatory condition into prominence as a causative factor.

There is perhaps no more important and difficult problem confronting a surgeon than the interpretation of pain in acute abdominal conditions; but, having referred to some of the features which characterize the splanchnic and peritoneal types, I shall leave their further elaboration to those who are much better qualified to deal with them than I am. In particular, we may hope to hear Mr. Morley giving us the result of his carefully considered research into the nature and meaning of the pain associated with appendicitis. Meanwhile, may I recall to your memory some of the heteronomous types of abdominal pain which are liable to entrap surgeon and physician alike?

First of all, there is the immobile painful abdomen met with in acute pulmonary disease—in pneumonia and infarct of the lower lobes, for instance. This is sufficiently common, and has been so long recognized that it deserves no more than a passing reference. Less common is the acute epigastric pain associated with vomiting and profound collapse observed in cases of coronary thrombosis, and generally found in persons suffering from arterial degeneration. It may simulate the picture of a perforated gastric ulcer so faithfully as to be a real diagnostic pitfall. Still more rare are the attacks of severe abdominal pain, colicky in type, which result from atheroma of the abdominal aorta and which have earned the title of "angina abdominalis." They resemble their cardiac namesakes in being paroxysmal, often at long intervals, and do not present associated physical signs such as should justify an exploratory laparotomy.

The gastric crises of tabes have an unpleasant notoriety for creating an atmosphere of diagnostic doubt, and the problem is generally dismissed in textbooks with the statement that the presence or absence of knee-jerks will suffice for its solution. As a matter of fact, the presence or absence of knee-jerks may guide us to the presence or absence of the tabetic process at the lumbar level of the cord, but has no bearing on the presence or absence of a lesion of the upper dorsal roots. On the other hand, the

pains of tabes, although pathogenetically obscure, are clinically characteristic, and those which infest the epigastrium present much the same features as those which inflict torture on the patients' limbs. They are lancinating, rapidly repeated, and chronologically paroxysmal. They are associated with intense superficial hyperaesthesia, but with no such muscular immobility or rigidity as is met with in peritonitis. The accompanying nausea and vomiting may be regarded as secondary and reflex in origin. In fact, careful attention to descriptive details and to the abdominal physical signs should serve to distinguish gastric crises from any morbid process originating in the abdominal organs without the necessity of worrying the patellar tendons.

Those who follow me will discuss the views held and supported by various authorities during the last forty or fifty years in regard to the origin and nature of abdominal pain. Since Ross advocated the distinction between splanchnic and somatic pain there have been many workers in this field of research. Mackenzie, Head, Hurst, Ryle, Payne and Poulton, Maylard and Bolton have all contributed much of value towards the solution of a very important and difficult problem from the clinical standpoint, and we may hope that its ventilation in a physiological atmosphere to-day may lead to its further elucidation.

Whether or not this hope is fulfilled, the discussion may be welcomed as evidence that the profession is taking an increasing interest in the meaning of pain, is more inclined to subject pain to careful analysis, and is more appreciative of the position of pain in the proper execution of its daily work. We may look forward with confidence to a time when the loose employment of such terms as rheumatic, neuritic, neuralgic, etc., for descriptive purposes is abandoned, and when due regard is paid, not only to the site and quantity, but also to the precise quality of pain as an essential factor in the diagnosis of disease.

As Hilton wrote sixty-five years ago: "Every pain has its distinct and pregnant signification, if we will but carefully search for it."

CONDITIONS SIMULATING PULMONARY TUBERCULOSIS;

WITH SPECIAL REFERENCE TO PNEUMONOCOCCUS
AND BRONCHIECTASIS.*

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THE substance of what I am about to say might be crystallized in one sentence: It is by bacteriological examinations of the sputum and faeces, and by these examinations almost alone, that we can establish a diagnosis of pulmonary tuberculosis or exclude it. I know that there are exceptions, such as the very early, sometimes the milinary and the latent case, and even occasionally, but very rarely, in other instances, but in the vast majority of cases the dogma will be found true. It is because of the neglect of this simple test that a great many cases of pulmonary tuberculosis escape recognition in the early stages. It is because of this neglect that many non-tuberculous conditions are diagnosed as tuberculous, and their true nature is unrecognized. If tubercle bacilli are not found after repeated examinations of the sputum, and a few examinations of the faeces, and there are definite signs and symptoms, it is certainly the time to begin to consider that the condition may be a non-tuberculous one.

Some may ask why I insist so strongly on the examination of the faeces, and an explanation still appears to be necessary. In many cases of active pulmonary tuberculosis either no expectoration is produced or what is produced is merely mucus or muco-pus from the nasopharynx. This is specially apt to occur in children and

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