Original Communications.

THE ETIOLOGICAL RELATIONS OF HEPATIC ABSCESS.

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IN a recent issue of the *Indian Medical Gazette* a paper entitled "The Pathological and Etiological Relations of Tropical Suppurative Hepatitis," appeared from the pen of Surgeon-Captain P. Hehir, I. M. S., in which he states that "by far the most common are cases (of abscess) occurring consecutive to dysentery, and arising from a secondary infective process affecting the liver through the portal circulation."

As the etiology of hepatic abscess is a subject of much practical importance from a pathological, and even more so from a therapeutic, point of view, it appears desirable that some of the arguments which disprove Surgeon-Captain Hehir's statement, and which show that we must look beyond dysentery for the true causation of tropical abscesses of the liver, should be briefly stated.

But first it is necessary to have a clear idea as to what is meant by the phrase "dysenteric abscesses of the liver." They are abscesses which are caused by the action of septic matter, or, as septic matter owes its virulence to the micro-organisms which it contains, to the action of micrococci, which are absorbed from the rectum and colon, and conveyed to the liver by the vena porta. When it occurs, therefore, the disease is essentially a local pyæmia which involves the portal system of veins and the liver. The intestinal ulceration bears the same relation to the morbid condition of these parts that a foul and septic wound bears to pyæmia. We are, therefore, justified in concluding that in *genuine* dysenteric abscesses, the condition of the mesenteric and portal veins will correspond with the condition of the veins which lead away from the septic wound in cases of pyæmia.

Now, in a case of pyæmia due to traumatism, the wound is sloughy and offensive; the veins leading from it contain extensive thromi which are undergoing puriform softening; the vein-walls are thickened, softened, and infiltrated with leucocytes, and present numerous minute hæmorrhages and small collections of pus. Micrococci are found everywhere—adhering to the living membrane of the veins, infiltrating their walls, plugging the terminal branches of the arteries, and crowding even the minutest abscesses. When the liver is involved in pyæmia the abscesses are small, varying from the size of a pin to that of a hazelnut, though occasionally one is seen as large as a walnut. The abscesses are numerous, occupy the surface of the liver, and are wedge-shaped with their bases directed outwards.

How different are these morbid appearances from those which are seen in the great majority of hepatic abscesses which occur in India! In the latter cases, the portal system of veins is healthy in every respect, whilst the abscesses are large, are usually single, and are almost always situated in the centre of the liver. In a small number of cases two, or even three, large abscesses are present, but to find numerous minute abscesses studding the surface of the liver is quite exceptional.

A little consideration will convince us that it is scarcely less than a physical impossibility that large single abscesses can be caused by the absorption of micrococci from the intestines. Granting that such absorption does take place, the organisms would be so intimately mixed with the blood current by the time it reached the liver that they would be more or less equally distributed to every subdivision of the vena porta, and would lodge at numerous points throughout the organ. Granting, further, that these organisms are capable of causing suppuration in any one situation, they will be equally capable of exciting it at every point where they lodge. This being so it is inconceivable that micrococci, which are absorbed from septic ulcers in the intestines can lodge in such a way as to excite a single large abscess of the liver. In this connection it is important to note that it is only in those cases in which the liver is found the seat of minute and multiple abscesses that the vena porta and its tributaries present signs of septic inflammation.

If all, or even a majority, of the hepatic abscesses seen in India were genuinely dysenteric (that is, pyæmic) in origin it would be a matter of not uncommon occurrence to find liver abscesses attended by symptoms of general pyæmia; for it is certain that the subdivisions of the vena porta do not possess the power of preventing the onward passage of micro-organisms. This is abundantly proved by what we know of the pathology of puerperal fever—a disease in which the liver and the system generally become rapidly affected by micro-organisms which gain entrance through the tributaries of the vena porta. In abscesses of the liver, however, constitutional symptoms are often conspicuous by their absence, and general blood-poisoning is of rare occurrence.

Again, if dysentery is a frequent cause of hepatic abscess, it is strange indeed that the mortality from the former disease has fallen to its present low figure (about 2 per cent.) amongst European troops; and it is even stranger that recovery from the latter should be a matter of frequent occurrence, for were the majority of liver abscesses septic in origin, such a termination would be impossible.

It has been the custom to attribute all abscesses of the liver to dysentery, provided there is a history of the patient having suffered from the latter disease, no matter at how remote a period, and the presence of cicatrices in the large intestine is taken as proof positive that large abscesses of the liver are due to dysentery even though these cicatrices have existed for years. The length to which this theory has been carried may by judged of by the fact that one writer referred a large abscess of the liver to "a minute cicatrix of the bowel, so small that it might easily be overlooked." Our present knowledge of bacteriology, however, must convince us that such a method of reasoning is entirely delusive; for it is impossible to believe that pathogenic organisms can lie dormant in the liver for an indefinite period. Even if we take this supposition for granted, however, there still remains a large majority of cases which have no connection with dysentery. For instance, in 300 cases of hepatic abscess published by Dr. Waring 73 per cent. occurred in patients who had never suffered from dysentery; whilst in Dr. Parkes' cases the percentage was even higher than this, namely, 78·26.

It is possible, however, that an abscess of the liver may co-exist with an attack of dysentery, and yet the diseases may have no etiological connection with one another. Or, if any such connection does exist between them, it may be the reverse of that usually taken for granted, for Dr. Morehead has shown that dysentery may be caused by the presence of pus in the liver, and that few patients die of abscess of the liver without manifesting symptoms of dysentery, although they have never previously suffered from it.

The differences of opinion which at present exist on this subject are due in a great measure to the fact that two diseases, which differ entirely both in their etiology and pathology, are included under the head of abscess of the liver. Thus, dysenteric abscesses are small and numerous, and their pathology is the pathology of pyæmia abscesses in whatever situation or structure they may occur. On the other hand, the *genuine* tropical abscesses of the liver are large, are usually single, and correspond in pathology with laudable abscesses in other situations.

The question naturally arises.—How are we to explain the occurrence of tropical abscesses of the liver? When stationed in Northern India, the writer undertook a careful inquiry as to the manner in which great atmospheric heat affected the functions of the liver, and the conclusions arrived at appeared in the *Medical Press and Circular*, vol ii, 1889. These may be briefly stated as follows:—

During the first year an European spends in India the amount of work done by the liver is greatly increased. So great is the amount of bile passed that the stools are often black, almost resembling tar in appearance. During this time the bowels may be moved three or four times a day, the motions being copious and semi-fluid. In full-blooded persons attacks of biliousness and of bilious diarrhœa are frequent. These attacks are often followed by a marked sense of relief and a feeling of increased vigour. Provided sufficient exercise is taken, and moderation in food and drink observed, this increased secretion continues throughout the first year's residence in India. While the action of the liver remains unimpaired, the health rarely suffers from exposure to the climate, and there is little susceptibility to the action of malaria. During the second and third year's residence, or sooner in intemperate persons and in those who are constitutionally predisposed to hepatic derangements, a marked change takes place. The liver acts in an irregular, spasmodic way. After a period of profuse secretion, the hepatic cells appear to become exhausted. When this occurs the secretion is greatly diminished, in some cases almost suppressed.

The condition of an exhausted liver may be aptly compared to that of an eye which has been overworked in looking at small objects. Under such circumstances the eye becomes irritable and unfit to perform its work. If at this time it is allowed a few days' rest it is quickly restored to health. If, however, the work is persevered in, acute congestion, followed probably by deep-seated mischief, or perhaps by loss of vision, becomes established. Unfortunately in the case of an exhausted liver, we cannot insure rest as we could do in the case of an overworked eye. On this account hepatic exhaustion is usually followed by congestion. The two conditions, however, are quite distinct, although often associated.

In a well-marked case of hepatic exhaustion the symptoms are unmistakable. The patient becomes pale and sallow, and has a haggard appearance. The appetite is almost lost. There is often a marked feeling of hunger, accompanied by an indescribable sense of sinking at the epigastrium, but a few mouthfuls of food produce a sense of distention. Flatulence, accompanied by a cold crampy sensation at the pit of the stomach, is often complained of, especially in the early morning. There is a feeling of uneasiness and distention

over the right hypochondrium. The tongue is usually pale and flabby, and indented by the teeth. Shortly after meals there is a feeling of nausea, and this may be followed by vomiting. Morning sickness may also occur. The bowels are usually somewhat relaxed, and the motions are putty-coloured and offensive. There is a constant feeling of languor, which is increased after meals. There may be frequent attacks of vertigo and dimness of vision, and the sleep is heavy and unrefreshing. There are occasional chills, followed by a slight rise of temperature, varying from 1° to 3° F. The head and hands feel hot and dry, and headache is usually present. The urine is hazy and has a strong unpleasant odour. It often deposits lithates on standing, and may contain a considerable amount of emulcified fat.

When fully developed, these symptoms indicate a grave case of hepatic exhaustion. In such cases the condition of the liver quickly reacts on the whole digestive system. The portal circulation becomes obstructed, and this obstruction gives rise to congestion and catarrh of the stomach and intestines. We know that all tissues in a state of exhaustion have more or less completely lost their powers of resisting any injury or strain to which they may be exposed, and the liver does not form an exception to this rule. This diminished power of resistance is a point of great importance, and will be again referred to.

The question now arises: What are the causes of this excessive action of the liver in tropical climates? Sir Ronald Martin, Dr. Parkes, and most of the older writers believed that it was due entirely to diminished activity on the part of the lungs. Dr. Parkes pointed out that the amount of oxygen in the air varied inversely with the temperature. He found that at 80°F., there was nine per cent. less oxygen in the air than at 32°F. In 1872 Surgeon Rattray, R. N., proved by careful observations that the number of respirations per minute were decreased 18 per cent. in the tropics. Allowing for the diminished amount of oxygen and the lowered rate of respiration, Professor De Chaumont calculated that the exhalation of carbonic acid by the lungs was diminished 25 per cent. We know that next to the lungs the liver is the chief organ concerned in the excretion of carbohydrates; and we are, therefore, justified in concluding that this diminished action of the lungs throws more work in the liver.

The writer's observations, however, have convinced him that by far the most important cause of the increased action of the liver is the blood deterioration, which occurs as the result of prolonged exposure to great atmospheric heat. Dr. Lauder Brunton has proved experimentally that exposure to great heat accelerates tissue metabolism, and greatly increases the excretion of urea. More recently Dr. Metschinkoff has shown that even a short exposure to great heat causes a marked diminution in the vitality of the red-blood corpuscles. The truth of Metschinkoff's observations must be obvious to every physician who has observed the effects on Europeans of residence in a tropical climate. In the words of Sir William Moore, "blood-degeneration is initiated in the system of Europeans from the very commencement of tropical residence."

For at least six months of the year Anglo-Indians are exposed to a temperature, which varies from 90° to 120°F. in the shade. The continued exposure to these high temperatures causes at first an increased tissue metabolism, and this is soon followed by diminished vitality, and then by greatly increased destruction of the red-blood corpuscles. The increased destruction of blood corpuscles takes place independently of, but is much increased by, attacks of malarial fever. Thus, Professor Kelsch estimated the destruction of blood corpuscles during attacks of ague as amounting to more than one million per cubic centimetre in twenty-four hours. Whilst studying the effects of attacks of ague on the blood, Marchiafava and Celli found that the terminal branches of the hepatic artery became blocked by the dead corpuscles and their escaped pigment.

We now know that the liver is chiefly, or perhaps wholly, concerned in the removal from the system of effete blood corpuscles. It converts their albuminous elements into urea, and their colouring matter into the pigments of the bile and urine. Thus, in the tropics the liver is called upon to excrete a large amount of carbohydrates, which would in temperate climates pass off by the lungs; whilst at the same time it has to rid the system of the products of excessive blood destruction. It is this combined strain which causes hepatic exhaustion.

The influence which excessive blood destruction exercises on the liver, even in temperate climates, has been shown by the experiments of Dr. W. Hunter—*Lancet*, vol. ii, 1888. By injecting pyrogallic acid, toluylendiamin and other substances into the blood he produced rapid destruction of the red corpuscles.

Amongst many other interesting results he found that this was quickly followed by derangement of the hepatic functions, and by an augmented flow of bile, which rapidly became increased in consistence, very viscid, and stagnated in the bile ducts. At the same time the liver and spleen became enlarged. In fact, Dr. Hunter had produced experimentally that condition of the liver, which the writer had previously described as always preceding the advent of hepatic exhaustion.—*Indian Medical Gazette* 1887.

Hepatic exhaustion having been excited other influences soon come into play. The obstruction to the portal circulation gives rise to congestion and catarrh of the stomach and intestines. and these in their turn produce derangement of digestion. Dr. Lauder Brunton has shown that during attacks of dyspepsia ptomaines are abundantly formed in the stomach and intestines, and that these, being absorbed, produce a form of toxæmia. Thus, as the result of hepatic exhaustion, we have conditions which eminently favour the development of inflammation of the liver. The vitality of the hepatic cells is lowered; its nutrient vessels are blocked by dead corpuscles; the blood within it is impure as it contains the unexcreted elements of the bile, together with ptomaines absorbed from the stomach and intestines; digestion is impaired, and the general health is below par.

Owing to their obstructed condition the nutrient vessels quickly loose their tone and become dilated. The resulting pressure and impairment of nutrition soon begin to affect the liver cells. The cells first become swollen and œdematous, and then commence to atrophy along their margins. As is shown by the analogy of Bright's disease, an impure condition of the blood, by exciting spasm of the smaller vessels, gives rise to obstruction to the circulation. When hepatic exhaustion is fully established the obstruction from this cause, originating in the intra lobular vessels, quickly reacts on the whole portal circulation, and causes not only congestion of the liver, but also that congestion and catarrh of the intestines which has been already referred to.

If a person whose liver is in this condition is exposed to a sudden chill, the usual determination of blood to the internal organs occurs. The liver, being the weakest point, gives way. The weakened vessels dilate still further, and exudation of leucocytes and liquor sanguinis takes place. Some of the leucocytes break up and liberate their fibrin-ferment, which unites with the fibrinogen of the liquor sanguinis. In this way the intra lobular spaces become infiltrated with leucocytes and filled with coagulated fibrin. This is the origin of the "drab-coloured patches" which Dr. F. N. Macnamara described as occurring in the livers of many persons who had been exposed to the influence of a tropical climate. These drab-coloured patches occur most frequently in the centre of the left lobe, and are usually single, although occasionally two or more may be found in the same liver. When they are once deposited these patches may persist indefinitely, and probably remain unabsorbed as long as the subjects of them are exposed to a tropical climate.

Chill is probably always the immediate exciting cause of idiopathic inflammation and abscess of the liver.* It has been stated that this cannot be so, else these diseases would be more common in Russia than in India. Such a statement, however, shows a want of appreciation of the effects of climate. In India chill frequently affects the liver owing to the fact that it is already the seat of grave structural changes, which have been produced by long-continued exposure to great heat. It may be noted that the incidence of chill is often a matter of pure accident. Thus, if a dozen persons are exposed to the same chilling influence the results are often totally different. In those whose organs are in every respect healthy no bad effects follow. Of the remainder, one may be attacked by conjunctivitis, another by nephritis, a third by bronchitis, and so on. The difference arises from the fact that in each case the affected organ was the seat either of some accidental irritation or of exhaustion due to excessive functional activity at the time of the exposure to cold. Being, therefore, in a weakened condition it was unable to bear the sudden strain thrown upon it and so became the seat of disease. The same rule applies to a person who is the subject of hepatic exhaustion. If, at the time of exposure to chill, he is already the subject of exudation (or drab-coloured) patches in his liver he will probably be attacked by hepatitis. If, on the other hand, his liver is free from exudation whilst his intestimes are in a catarrhal condition and his rectum overloaded, he will probably suffer from dysentery or dysenteric diarrhœa. In either case chill is the immediate exciting cause of the disease, but the mechanical irritation, either from the exudation patches or from the distended state of the rectum, is the condition which determined the diseased action in the affected organ.

In India, owing to the diurnal variations of temperature, people are exposed to slight, but daily recurring, chills. It is these slight, but constantly recurring, chills, I believe, which cause the exudation patches in the livers of those who are suffering from hepatic exhaustion. When they have once formed, the subsequent behaviour of these patches depends entirely on circumstances. If they occur in persons of sound constitution, who live carefully, they may become entirely absorbed on removal from the tropics. Occasionally, however, they break down after the subjects of them have arrived in temperate climates, in this way giving rise to those abscesses of the liver which sometimes occur years after Europeans have left the tropics. If they occur in persons who are

* The fall in temperature which can be borne without discomfort varies greatly in different persons. For instance, many persons will bear a sudden fall of 15° to 20°F. without discomfort; whilst in many others a fall of 4° or 5° will cause a feeling of general uneasiness, chilliness, and slight pains in the back and lower extremities.

otherwise healthy, but who live carelessly or do not guard against chills, they will sooner or later lead to attacks of hepatitis, which may end in recovery or may lead to rapid breaking down of the affected area with the formation of an abscess of the liver. Lastly, if the exudation take place in a person of poor constitution and strumous habit it may break down slowly and silently, and an abscess may form without the patient having shown any symptoms of hepatitis whatever.

THE EVOLUTION OF ANTISEPTIC SURGERY: A RETROSPECT.

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CENTURIES before the discovery and demonstration of micro-organisms and the realization of the part which they play in surgery, remedies were in use, which doubtless owed much of their value to antiseptic action. Thus we read that Hippocrates recommended the use of wine and oil. In later times various aromatic spirits and balsams were largely employed, such for instance as arquebuzade and friar's balsam. Such remedies must often have been most efficacious, especially when used as an outside dressing. The use of hot pitch as an application to stumps may be claimed as a primitive and empirical use of carbolic acid, if not of sterilization by heat. But probably most patients so treated would have found the application of dry lint a more comfortable procedure. The value of this simple method of treating fresh wounds was fully recognized by John Hunter.

So variable, however, were these agents in their action, that before the antiseptic era there was a wide divergence in the dressings ordinarily employed. Fomentations, poultices, wet dressings, dry dressings, unctuous and oily preparations competed with each other, and some surgeons adopted the open method of treating wounds. Now in many cases, excellent results were obtained. Union of wounds "by first intention" was of not unfrequent occurrence. Some surgeons were very careful and used quantities of water for flushing and cleansing wounds. Nevertheless, in spite of all care, a large number of wounds became foul and remained so for a longer or shorter time. And in those days when hospital construction and sanitation were less carefully planned and executed than now, and when surgeons were to a large extent ignorant of the real cause and nature of putridity in wounds, it is no wonder that hospital gangrene was prevalent and produced a very high mortality. Even trivial cases were often attacked and sometimes with fatal consequences. The treatment of large wounds was fraught with danger. Syme,* speaking of compound fractures and referring to the wound which constitutes their distinguishing character says: "There is apt to proceed from this source violent inflammation with fever, terminating in profuse suppuration or gangrene, or death without any remarkable local change."

Let us look at the results obtained in a totally different class of cases, the chronic abscesses. Referring to the dangers of opening these collections when large, Syme† writes: "The surface of the cavity * * * sometimes inflames and produces such violent constitutional disturbance as proves fatal in a few days: more frequently the bad consequence consists in a profuse and long continued discharge from the morbid surface, by which the patient's strength is gradually exhausted * * * at length fever is really induced, the patient shivers—his tongue becomes foul—he loses his appetite—and speedily sinks under the disease."

It was, however, at this time beginning to be believed that the *entrance of air* promoted putrefaction in chronic abscesses. And Abernethy took an important step in the right direction when he introduced the method of tapping with a trocar at intervals, allowing the wound to heal each time.

Now, what chemical agents were being applied to wounds in those days? We find that amongst other things borax, alum, the sulphates of copper and of zinc, nitrates of silver or mercury, oxide of mercury, acetate of lead and nitric acid were in use as astringents, stimulants or caustics. The value of deodorants and disinfectants was also recognized. Solutions of chloride of lime or soda and ointments containing turpentine and resin were freely employed for foul wounds. In 1859, it was suggested by M. M. Corué and Demeaux‡ that a powder made of coal tar and lime should be employed in surgery. Experiments were made with this in the hospitals of Paris and in the French army in Italy with gratifying results. Subsequently, Mr. Calvert, in England, and Parisel and Bouchardat in France, showed that carbolic acid is "the essential principle to which coal tar owes its antiseptic properties."

In 1860, Küchenmeister, of Dresden, reported most satisfactory results from the use of carbolic acid in surgical practice, and as a means of arresting putrefaction and preventing the development of fungi. In 1863, Lemaire, of Paris, published a work on carbolic acid in which he adduced cases and remarks to show its utility as an antiseptic in a great variety of surgical diseases and attributed this to its action on the low vital organisms which produce decomposition in wounds and ulcers. In England, Dr. Y. R. Wolfe published a paper in 1865, re-

* Principles of Surgery, 1863, p. 179.

† Principles of Surgery, p. 66.

‡ Braithwaite's Retrospect, vol. LVI, 1867, p. 166.