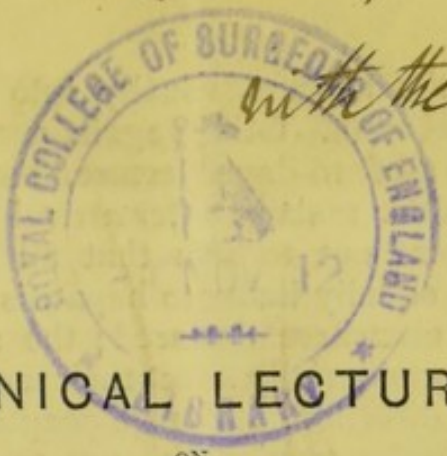
**Clinical lecture on tropical abscess of the liver / by Sir Dyce Duckworth.**



# CLINICAL LECTURE

ON

# Tropical Abscess of the Liver.

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Gentlemen,—The subject of hepatic abscess is one of great interest to all English students of medicine. It cannot fail that some of you will have to practise your profession in some part of our tropical possessions, and in these days, when locomotion is so rapid and easy, some of you will also as certainly not fail to have experience of this disease at home. I think it is not unlikely that abscess of the liver is now less often met with in the tropics than formerly. I have no facts to support this view, but I conceive it to be likely, because Europeans nowadays take better care of their health, eat and drink less than formerly, come home at shorter intervals to recruit their powers, and are thus less exposed to, and saturated with, malarial influence.* For twenty-one years I have had a special experience of Anglo-Indians of all ranks in connection with the services of several of the great Indian railway companies, and I may at once state that my experience of hepatic abscess as a cause of invaliding in these persons has not been extraordinary. To be sure, the worst cases have died in India or on their way home: others have had favourable, and generally rapidly favourable, terminations

* Sir Joseph Fayrer, however, informs me that he thinks hepatic abscess is now as common as formerly.

by rupture of the abscess, usually into some part of the gastro-intestinal tract. Others again, such as the case I take for the subject of to-day's lecture, have developed the symptoms of their malady after arrival in this country. I wish first to point out to you that all Europeans in the tropics are not equally liable to hepatitis and abscess, as I have here and elsewhere declared; the special predispositions of persons materially affect their tendency to go wrong in any morbid direction. A large chapter in the history of medicine within the tropics has yet to be written, which shall relate to the influences of climate, and the habits thereby engendered, upon individuals of the several constitutional conformations or so-called diatheses. To such a chapter the late Dr. Norman Chevers contributed very notably and with great ability. He formed the opinion that persons with tubercular proclivity were more than others apt to suffer in India from dysentery, while strumous persons were similarly predisposed to hepatic abscess. In respect of idiopathic hepatic abscess, he did not consider that any of the ordinarily assigned causes, such as malaria, intemperance, dysentery, portal pyæmia, and tuberculosis, were essential, and he concluded that the influence of tropical heat was specially causative.*

You have been already taught in your systematic lectures that hepatic abscesses are mainly divided into two classes; first, the pyæmic or multiple form, and, secondly, the tropical or solitary form. This is an artificial division, and worthy of retention; however, you must expect to find in practice that there may be more than one abscess owning no direct connexion with either dysenteric ulceration or pyæmia. The etiology of liver-abscess has been a hotly debated question, especially in respect of the relation which dysentery bears towards it. At one time the abscess was thought to induce the dysentery; later, it was thought that dysentery led to the abscess. Again, it was believed that both were due to a common cause. The late Dr. George Budd, of King's College and the *Dreadnought* Hospitals, was one of the earliest advocates of the direct connexion between dysentery and hepatic abscess, teaching that the latter was due to direct absorbtion from the ulcerations in the colon of purulent or infecting matters. This theory,

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* Sir Joseph Fayrer and others with large Indian experience do not, however, agree to this.

you see, is especially an embolic or pyæmic one. Hepatic abscess, as most commonly seen in Europe, is of the pyæmic or multiple variety, and is naturally regarded as a most fatal malady. No one now doubts the connexion I have just described, cases being frequent in which purulent absorbtion takes place from breaches of surface in any part of the alimentary canal drained by branches of the portal vein. Thus, gastric ulcer and ulceration of the gall-bladder and bile-ducts, and operations on the rectum, may induce embolic abscess in the liver. A noteworthy point in the matter is this, that dysentery is far more common *uncomplicated* with hepatic abscess. There must, therefore, be some other factor than mere ulceration to account for infecting absorption and resulting hepatic abscess, and this factor is without doubt *a special infectivity attaching to the detritus of the bowel ulcerations.* It is very noteworthy that no such accident occurs in connexion with the ulceration of enteric fever or tuberculosis. With suppurative pylephlebitis we are not now concerned ; the mischief here extends along the veins involved. Next, observe that hepatic abscess in the tropics is common enough when no history of dysentery is to be had in the case, no pyæmia is present, and no very obvious cause can be adduced. The pyæmic theory fails to fit such cases ; and in the majority of instances in the tropics the abscesses are either solitary or limited to two or three, whereas in Europe we commonly find innumerable points of suppuration in the liver, and readily discover their origin in some distempered breach of surface in the line of the portal circulation, or in some other part of the body. Hence, both forms of abscess may occur in the tropics, but the pyæmic or multiple form only arises in Europe. A large solitary abscess may occur in connexion with dysentery in the tropics, or several small dysenteric abscesses may coalesce gradually into a large one ; but this is not often seen in this country. Multiple pyæmic abscesses are the rule here. The late Dr. Moxon thought the differences between tropical and pyæmic abscesses depended on their different ages or duration, and that ulcerations or cicatrices of them would be usually found if carefully sought for. But this view will certainly not apply to the history of many cases. The embolic theory has captivated the attention of pathologists to the exclusion of that which explains the production of abscess as the outcome of a specific hepatitis. The latter explanation appeals rather to the

bedside than the deadhouse physician. Many differences of opinion in medicine are, in my thinking, to be thus explained. Clinical explanations may be seemingly fanciful sometimes, but they cannot be dispensed with till post-mortem research has modified or confuted them. It would appear that with improved methods of treating tropical abscess the opportunities for examining the intestines for cicatrices of ulcers will grow fewer and fewer.

What, then, are the probable causes of the solitary or tropical abscess? Without doubt they depend on peculiarities of life and climate. It is certain that tropical climates induce in many Europeans congestion and enlargement of the liver. Unusually hot weather in Europe produces this much, unless great care in diet and habits of life be practised. Indulgence on the part of tropical residents in European habits as to eating and drinking, with undue exposure to the sun, are to blame to a large extent for this. But beyond these causes, there is something specific in the effects of prolonged heat upon the functions of the liver; all constitutions not being equally susceptible, however. Again, it seems certain that malarial poisoning has largely to do with this congestion. The spleen suffers with the liver under this exposure, and both organs fail of their due functions. It is noteworthy that hepatic abscess is less common by far in the West than in the East Indies and China. Malaria is perhaps equally pernicious in both. It is not remarkable that the bowels should suffer sometimes together with the other viscera mentioned, and hence, with exposure to chill, the risks of impure water and of improper food, all the conditions for the onset of dysentery are provided. In Indian nosologies you will often meet with the term "hepatitis": acute and chronic varieties are mentioned. No very clear distinction has been drawn between congestion and hepatitis. If the inflamatory process reaches a certain point, we naturally have to deal with abscess as the outcome of it. Chronic hepatitis is applied to a long-standing condition of hyperæmia and engorgement, which, if neglected, usually passes into a form of cirrhosis. As indicating in some measure the etiology of the troubles we are now considering, note that women are more rarely the subject of such hepatitis in India as leads to abscess, and that in children the disease is practically unknown, although dysentery is very common amongst them. Hepatic abscess is rare under the age of twenty, and more frequent after

twenty-five years. These facts, which I give you on the authority of able observers—Dr. John Macpherson and Sir Joseph Fayrer—would indicate that alcoholic intemperance, with greater exposure to solar influence and intenser grades of malaria, are not a little causative, and this is the opinion I have been led to form.

With these remarks, with which I have thought it well to begin my lecture to-day, I now pass to the case illustrating it. It is that of M. N.——, aged twenty-eight, single, a medical officer practising in the Bombay presidency. He went out to India in 1883. He is of excellent constitution, and his family history is satisfactory. He has been stationed in several districts, and been exposed to malaria. After being out for a few months he had a slight attack of dysentery, being laid up for ten days. He made a good recovery, and had no return of his trouble. He was always temperate. Within the first year of his sojourn he had an attack of catarrhal jaundice, and he also suffered six or seven times, but not severely, from malarial fever. As he expressed it, "he knew he had a liver." In June, 1886, he left India on leave of absence for three months and came home, landing on July 19th. He believed himself to be in perfect health. In August he suffered from chill after being overheated, and was seen by Mr. Coalbank of Twickenham, who found him confined to bed, suffering much pain from pleurisy in the left side (postero-lateral region). His liver was then enlarged and tender. His temperature reached 103° and 105°. (Dr. Norman Moore saw him later in my absence, and confirmed Mr. Coalbank's opinion as to pleurisy, with malarial fever). He lost flesh somewhat rapidly, and looked ill. On Sept. 16th he was recovering, but was still weak, had a coated tongue, and wavy, uneven pulse. Dr. Sedgwick Saunders, who saw him at that time, refused to allow him to return to India. On Oct. 28th I saw him for the first time. He was still thin and cachectic-looking. I could find no physical signs anywhere in the chest beyond a trifling amount of dulness, with imperfect respiratory note, in the left postero-lateral region. There was no jaundice, and the urine was natural. I sanctioned his return to India in November. On the night of Nov. 1st, after a busy day in London, he awoke with very severe pain at the epigastrium radiating to the left side. The pain was of a stichy, "catching" character, and there was agonising cough. In no way could he account for this attack. He had a slight

cough for a few days previously. He came up from Twickenham and saw his friend, Dr. Goodhart, who examined him, but found no signs of pleurisy, but observed some fulness about the left hypochondrium. His temperature was raised to 102° and 103° at this time. Cough troublesome, with glairy, tenacious sputa, not rusty. He kept to bed for three weeks. The pain was very severe, and nothing relieved it. There was a dull dragging pain in the left costal arch, and much epigastric tenderness. No rigors. His weight was then 10st. 7½lb., his usual weight being about 12st. On Nov. 30th I found the same physical signs as before in the left pleura. On Dec. 11th I saw him again. Dr. Andrew had also examined him, and found nothing to explain his severe pain, which was attributed to pleural adhesions. The pulse was 84, and regular. He was still looking pale and ill. I had a suspicion of hepatic mischief as the cause of all his trouble, but there were no physical signs to justify any certainty of this at this time. I recommended an extension of leave for three months, careful regimen, and good diet. On January 19th, 1887, Dr. Andrew again examined him, and found a tumour projecting under the left costal arch, indistinctly fluctuant. No improvement had occurred in the general health. There was a slight icteric tint of the conjunctivæ. On the 21st Dr. Andrew and I saw him together. There had been no rigors, no sweatings, no shoulder-pain. The tumour was evidently an abscess in the left lobe of the liver. Slight pulsation was communicated to it from the heart. At the beginning of January severe pain had returned about the ensiform cartilage, but there had never been freedom from pain in the left side since October. Vomiting had occurred about once daily, generally about an hour after taking food. There was much flatulence. He was admitted to John Ward on Jan. 22nd. The temperature was 98·2° on the first night. The tongue was coated all over. Bowels open; stools bilious. Urine: specific gravity 1025; faintly alkaline; depositing phosphates; void of albumen, blood, glucose, and bile. Heart's apex in fifth space, in normal situation. Sounds natural. Pulse 72, of fair volume and power, and regular. In the chest nothing abnormal. The liver dulness began at the sixth rib. To the left of the epigastrium a rounded projecting tumour, everting the cartilages; deep-seated fluctuation made out; the skin was not red over it. Spleen not felt. Abdomen

otherwise natural. Lower edge of right lobe of liver not felt or apparently depressed. On Jan. 23rd the temperature reached the highest point recorded in the hospital—100·2°. On the 24th, after a restless night, the temperature was 99·2°. Chloroform having been administered, Mr. Langton aspirated the tumour, and twelve ounces of chocolate-like grumous matter were withdrawn, containing pus and débris of blood. Temperature last night 98°; to-day 97·8°. Sick after chloroform. Dragging pain relieved, but pricking pain in region of abscess. Pulse 64, of good volume and strength. Had better night. On the 26th, the temperature was 96·4°; pulse 72, feebler and irregular. Much nausea, and vomited once. Urine loaded with urates. On the 29th he was going on very badly; no action of the bowels. It was decided now to have the abscess freely opened. Under chloroform and with full antiseptic precautions, my colleague, Mr. Langton, cut down through the left rectus abdominis and came upon thickened adhesions between the sheath of muscle and peritoneum. He passed in an exploratory syringe, then a director, and then his finger. Reddish pus (six ounces) flowed freely away, and an indiarubber tube was inserted and stitched into the lips of the wound. Iodoform and antiseptic dressings were used. The nausea continued for two days and gradually passed off. Nourishment was badly taken, and a quinine and nitro-hydrochloric acid mixture could not be borne. Nutrient enemata were given in default of sufficient by the mouth, and about two ounces of brandy were given each day. The wound did well and discharged freely. It was dressed daily antiseptically, and remained quite sweet. The temperature remained subnormal. The appetite soon became large, and flesh was rapidly gained. On Feb. 12th the patient was sitting up daily and making progress in all respects.

Let me briefly review the history and course of this case, and comment on the procedures of treatment. The patient while in India suffered from most of the ailments that befall Europeans, and, though never severly ill, it is clear that the climate and the circumstances of his life told upon him. Thus, he had dysentery, malarial fever, and catarrhal jaundice. I have already told you that he was a very temperate man, and lived carefully. In June of last year he left India, feeling in perfect health, intending to stay a few weeks only at home. In August he began to suffer after a chill, and had pleurisy and remittent fever. At this time the pleurisy

was at the back of the chest, on the left side. He failed to make a satisfactory recovery of his general health, losing flesh, looking ill, and having a not very explicable cough. It would be interesting to know the temperatures that occurred at that period. Very early in November a second attack of pleurisy unaccountably came on, this time near the cartilages adjacent to the ensiform cartilage, the pain being very severe and the temperature raised. No rigors and no sweatings. Six weeks later, although the second attack of pleurisy had passed off, there was no improvement in the general health, and a pinched and sallow appearance was manifest. Physical signs alone were insufficient to explain this condition. In three weeks' time there was a return of the severe pain near the ensiform cartilage, and in less than three weeks afterwards a tumour was detected in the left lobe of the liver. Gentlemen, it is easy to be wise after an event, but the fact remains that while the symptoms I have detailed to you fall in admirably with those that might be expected in such a case, there was a marked absence of physical signs to justify a confident diagnosis of all that was proceeding in the liver. Suspicion there was in the minds of several of those who saw the case, but nothing definite could be established, and certainly nothing could justifiably have been done more than was done.

The case, then, illustrates a point which has long been recognised in respect of the latency and insidious character of hepatic abscess. Large collections of matter may occur in the liver without any symptoms distinctly referable to that organ. In this instance it is not clear when the abscess began to form. It is quite possible that the disease was already in progress before the patient left India. The pleuritic attacks were plainly significant of the mischief that was going on below the diaphragm, and the severe pains near the ensiform cartilage betokened adhesive capsular hepatitis of the left liver lobe, the results of which ultimately proved so protective and beneficial. You will understand that according to the special seat of the abscess so will the symptoms vary in any case. Thus, if they come to press or point upwards, pain will be felt and irritation be excited in the diaphragm, pleura, or lung, with cough ; if downwards there will be interference with the functions of the stomach, and nausea and vomiting may be expected. In this instance irritation occurred on both aspects of the left lobe, the pleura and stomach both being implicated.

The liver substance appears to be little sensitive, but when the capsule is involved there is commonly severe pain; hence the pain in hepatic abscess is probably due to pressure on the capsule and to the happily protective perihepatitis that is often set up as the abscess pushes its way.

The leading symptoms, when present, of abscess of the liver, are fever, hepatic pain, severe gastric catarrh, with foul, coated tongue, nausea, vomiting, hiccough, and inability to lie on the left side. The abdominal wall is rigid, one or other rectus muscle contracting to protect tender parts below. Pain in one or other shoulder or scapular region is sometimes met with, induced either through the reflex mechanism of the phrenic and cervical nerves, or through the pneumogastric and spinal accessory branches. There is great languor and lassitude, rigors are common, and nightly rises of temperature. Slight jaundice is present, simulating ague. The urine is loaded with lithates, and may contain albumen. As suppuration proceeds profuse sweats occur. Bile is present in the stools. Tropical abscesses point usually superficially, and present in a low intercostal space or under the cartilages in the hypochondria. The spleen is not enlarged as a rule. Now in this case there were no rigors, no shoulder-pains, and no sweatings. The absence of these symptoms added somewhat to the difficulties of diagnosis.

Respecting the treatment. There is now only one opinion as to the necessity of early and free opening for tropical hepatic abscess. At one time, in India, it was thought best not to operate, and to wait for spontaneous bursting. Nature was found to be sometimes kind, and to allow the pus collection to empty itself into some evacuant channel. But, unhappily, rupture into the lung or pericardium, or into the peritoneum, is greatly to be feared. Amongst the triumphs of modern antiseptic surgery is the treatment of hepatic abscess. A more important question remains for consideration in each case, and it relates to the establishment of adhesions between the liver-capsule and the parietal peritoneum. There is always an apprehension of the abscess-cavity collapsing after being opened and discharging into the peritoneum. To prevent this accident, preliminary measures have been recommended to set up inflammatory adhesions. This question also comes to be discussed in operating on hydatid cysts in the liver. It has been proposed to set an issue with caustic potass and peas, and thus

secure the end desired. I have seen this method practised twice, but do not expect to see it again. Simple tapping by aspiration is often performed in the hope of setting up adhesions in advance of the subsequent graver operation. This was done in the present case partly for completing the diagnosis, and with the prospect of aiding adhesions. Again, it is proposed to cut down to the surface of the tumour and stitch the edges of the wound to it, completing the operation a few days later. In by far the majority of cases these methods are unnecessary. In the progress of the abscess, as it pushes toward the capsule, perihepatitis is often induced, and firm adhesions are set up. This is not, perhaps, so often the case in suppurating hydatid cysts. In support of the general safety of immediate and bold operative measures, we have the unique experience of Sir Joseph Fayrer and of many of our sugical brethren in India, a body of men of whom this country is not, I think, sufficiently appreciative, who now never hesitate to evacuate these abscesses so soon as they diagnosticate them, and practically disregard the question of adhesions. Their results have been so satisfactory that we may fairly follow them in this practice. Any remaining considerations, such as insertion of drainage-tubes, relate so specially to surgical details that I cannot enter upon them. If decided improvement does not follow the evacuation of the abscess, we may suspect either imperfect drainage of it or the existence of an independent one. You remember that there may be two or three centres of suppuration in even tropical non-embolic cases. I would add a few words on the subject of washing out the abscess-cavity. I believe this to be unnecessary if free drainage is secured, and even if done antiseptically to be a possible source of mischief. In this case, as in cases of empyema, I believe it to be the better practice to abstain from washing out the cavities. You may ask if any surgical interference is ever called for in respect of dysenteric or multiple abscesses. Even in these cases, according to Sir Joseph Fayrer, puncture and evacuation may be useful.

Respecting the prognosis for the future in any case where the abscess has burst favourably or been opened successfully, we have no grounds for believing that any special immunity from a future attack exists. Such patients do best to leave the tropics and live at home, but not seldom they return and do well. Second attacks may occur, but it is generally believed that these may be due rather to undeveloped or

residual abscesses, which have later become active. A residual abscess may long remain encysted, and be symptomless, till by gradual thinning of its walls it suddenly gives rise to fatal symptoms. Dr. Maclean and Professor Aitken have related such a case to me from their experience. Hence, there must always be a somewhat guarded prognosis in any case, and for some long period. In my experience where abscesses have burst favourably, the patients seldom long remain unmindful of their trouble. Dragging pains, probably caused by adhesion, are sometimes suffered from. In the case of an officer of Horse Artillery under my care, he could never wear his sword comfortably through a field day. With care in diet, and in all other respects, some of these patients manage to complete their tropical career fairly well. The drugs which prove useful are quinine, cinchona, and mineral acids. When the tongue is coated and there is no appetite, alkalies, such as sodium bicarbonate with bark, given after effervescence with a little citric acid, do better than quinine and acid. Subsequently, the chloride of ammonium, with some bitter, or with the mineral acids, is very useful. Prolonged stay at the seaside in some mild or sheltered part is the most efficient climatic agent. Sea-bathing and cold baths must be forbidden, and warm sea-water baths or tepid sponging only be sanctioned.

I will draw out for you on the board the leading characters of the two varieties of hepatic absesses, so as to impress them on your memory. Remember, however, that all such categories are made on somewhat rigid lines, and that large bedside experience tends to impair one's estimate both of tabulation and statistics :—

HEPATIC ABSCESS (two chief varieties).

A. *Tropical* (specific suppurative hepatitis).—Solitary, or few in number. May be very large, form rapidly, or occupy some months before urgent symptoms arise. Course often latent and insidious. Connexion with dysentery not commonly traceable. Infrequent in women ; unknown in children. Rare before twenty ; commoner after twenty-five. May occur in temperate and careful-living persons. Tendency to burst ; jaundice slight ; spleen not enlarged. Probably due to solar, climatic, and dietetic influences. Some constitutions—*e.g.*, strumous—more apt than others to suffer. Commoner in East than in West Indies, and occurs on Mediterranean littoral.

B. *Pyæmic* or *Embolic* ("dysenteric abscess").—Multiple, may be very numerous and small. Course rapid. Follows on dysentery or septic inflammation of parts drained by portal vein. Part of general pyæmia, especially after head-injuries and damage to medulla of bone. Jaundice marked; spleen enlarged. Seldom bursts. Occurs independently of climate, but much connected with dysentery.

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