LETTSONIAN LECTURES

ON
THE PATHOLOGY AND TREATMENT OF SOME
DISEASES OF THE LIVER.*Delivered before the Medical Society of London.*

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LECTURE II.—THE LIVER AND ITS VESSELS.

THE connection of the liver with the nervous system occupied our attention at the last lecture, and we will now pass to the consideration of some of those pathological conditions which are connected with the vascular supply of the gland. The hepatic veins, the vena portæ, and the hepatic artery, constitute three blood-channels to or from the gland; they unite at the minute lobules, but the import of their pathological changes is very different, and the symptoms which indicate those changes are also diverse in their character and in their results. I would premise a few words in reference to the arrangement of these vessels.

[After describing the circulation of the liver, the lecturer proceeded.]

The acinus of the liver receives branches from three different sources: the hepatic capillary veins occupy the centre, the portal capillaries are at the circumference, and the latter are joined by the capillaries of the hepatic artery. According to Virchow, these three portions have different pathological relations: the circumference of the lobule presents us with fatty changes—the portal part—that which is in closest connection with the absorbent mucous membrane of the intestine (and the blood of this vein has been shown to be of a milky character after digestion); the central part first shows indication of increased pigmental deposit—the part that is nearer to the heart, and more indicative of hæmatomic change; and, thirdly, lardaceous disease is most marked at the centre of the lobule.

The pigmental deposit may certainly be seen in the centre of the lobule, and in partial fatty degeneration globules are more abundant at the circumference; but in many instances of lardaceous disease that I have examined, it was quite impossible to establish any such definite limit of disease, probably from the change having assumed a more general character. The capillary vessels surround the cells of the liver, which are contained in the meshes between them; they are extremely delicate in their character, and with difficulty defined. The injections of the liver are not satisfactory; but, by careful washing and gentle manipulation, the hepatic cells may be washed away, and the capillary vessels are left, as in the accompanying drawing, made eighteen years ago, from the liver of a child who died from traumatic tetanus, and in the acini of the liver a cellular arrangement could then be made out, as seen under a magnifying power of $\frac{1}{2}$ inch. As to the cells of the liver, they vary in size, and sometimes undergo atrophy, independently of the deposit of oleaginous particles or albuminoid substance in them. It is important to bear in mind that, as the function of the liver is a double one, having a relation to the blood as well as to the bile, so also is the circulation double in character; there is a constant current of blood towards the heart, whilst there is a constant secretion of bile and flow towards the bile-ducts.

The pathological changes in the *hepatic veins* are comparatively few, and are generally of a passive kind. In the distension from heart-disease and chronic bronchitis, etc., these veins first present signs of congestion; the centre of the lobule is deepened in colour; the capillaries are enlarged, and the mottled appearance (called nutmeg-liver) is produced in some cases. I have seen fibroid cells in the acini as if from *ante mortem* fibrillation of the blood. We rarely find contraction of the hepatic veins from inflammatory fibroid deposit, as with the portal vessels; but in some cases hepatic abscess, extending into the veins, leads to disease and thickening of the coats of the vessels.

In passive congestion of the hepatic veins, the gland gradually becomes enlarged, and after a time may be felt several inches below the ribs. It is found, on manipulation, to be harder than natural; the increase in size is uniform, the surface is smooth, and the thin edge may be recognised by sudden but gentle pressure. The secretion of bile is altered, and the countenance of the patient becomes sallow; the urine is deepened in colour, and often scanty in quantity; there is a sense of painful tension produced by the stretching of the fibrous envelope; the bowels are irregular, and sometimes the excreta are paler than natural. All these symptoms are more marked if the congestion extend to the

portal venous capillaries, and if there be distension of the vena portæ. Then there is evidence of congestion of the mucous membrane of the stomach, the digestion is impaired, catarrhal mucus is secreted in excess, food does not digest, but flatulent distension is produced, with increased dyspnoea and distress; the intestinal mucous membrane is congested, and colic and irregular action of the bowels may be produced; whilst similar congestion of the hæmorrhoidal veins induces piles. If the congestion of the liver and of its vessels increase, serous effusion into the peritoneum follows, with the attendant symptoms of ascites.

The passive enlargement and congestion of the liver to which we have referred is greatly relieved by those remedial measures which unload the capillary vessels of the intestinal tract and of the abdominal glands—free mercurial purgatives, with or without squill; saline purgatives, as the aperient salts of soda and magnesia; saline mineral waters, etc. But it is a great mistake to regard this condition of the liver as the primary disease; it is only a secondary one, though, unfortunately, often regarded as the most important malady.

In the *portal vessels* the morbid processes are more numerous, and are often of a very distinctive character. The congestion to which we have already referred is secondary to that of the hepatic veins, and is dependent on obstructive disease affecting the pulmonary circulation. The congestion reaches the interlobular plexus; the nutmeg mottling is more intense; and the gland sometimes assumes a coarse and granular appearance, as if it were undergoing fibroid degeneration, cirrhosis, etc.; the lobule is said to be atrophied first in its central portion. The congestion of the portal system is, however, sometimes of an active character, and quite independent of pulmonary and cardiac disease; it is the interlobular venous plexus which then alone becomes congested; and it is this change which we find in the hyperæmia after intemperance, after malaria, and other allied states.

The vena portæ and its branches are subject to obstruction from causes affecting either the interior or the exterior of the vessels. As to the former, the vessel is obstructed by the coagulation of blood, thrombosis, or by the entrance into the vein of cancerous growth, or the perforation of an abscess or cyst; in the latter case, external disease, inflammation of the coats of the vein, produces fibrinous effusion and contraction, even to the complete obstruction of the vein; œdema and fibrinous effusion into Glisson's capsule, acute inflammatory disease and suppuration, chronic inflammation of the peripheral portions of Glisson's capsule—as in ordinary cirrhosis—senile fibroid degeneration, each produce contraction of the vena portæ. The inflammatory disease set up by the passage of gall-stones, the pressure from abnormal growths, from hydatid cysts, from enlarged glands, from chronic peritonitis, may also affect the vena portæ in a partial or general manner.

To revert, however, to those obstructions that are of an internal character, we will mention, first, the coagulation of blood in the portal vein. This coagulation is more frequent than is usually supposed, and it is the cause of some of the symptoms which are observed in great exhaustion: the obstruction may be in the trunk of the vein or in its branches. In the coronary veins of the stomach, it is the cause of the effusion of blood and the coffee-ground vomit which are sometimes observed towards the termination of disease, and in which no ulceration of the stomach can be found. In the mesenteric veins, this venous coagulation leads to appearances closely resembling internal strangulation. Another form of internal obstruction, however, is the extension of disease of a cancerous character into the canal of the vena portæ. Several instances of this kind have come under my own notice. In one, under the care of my late colleague, Dr. Addison, the portal vein became suddenly obstructed by the entrance of cancerous products, and the liver was injected with the abnormal growth. Severe hæmorrhage from the stomach and intestinal mucous membrane supervened, and in a few hours the patient died.

This form of portal injection and obstruction must be distinguished from another kind of cancerous disease, in which the heterogeneous product passes along the exterior of the portal vessels to its minutest branches, and may be seen distending Glisson's capsule throughout the liver. The accompanying drawing was taken from such an instance of disease. There was carcinomatous disease of the cervical and axillary glands, as well as similar disease of the spleen, liver, and mesenteric glands.

The vena portæ sometimes communicates with abscess in the liver, with hydatid cysts, and leads to hæmorrhagic effusions. Our limits will not permit us to enter upon many interesting cases of that kind. The vena portæ may, however, be obstructed by disease external to it, or from inflammation of the coats of the vein itself. The result of this is sometimes seen in the entire wasting of considerable portions of the gland, as we have seen of the whole of the left lobe; and if the obstruction have occurred during foetal or early infantile life, the appearance is still more remarkable. The walls of the vein sometimes be-

come thick and fibroid, and the vein completely obstructed, as in the following instance, which was under my care in Guy's Hospital during the year 1870. A large blood-cyst had been formed at the base of the liver.

Cordelia W., aged 34, was admitted in November. She had been a spirit drinker. Eight months before she first presented symptoms of ascites, and was tapped in two months. Whilst under the care of my colleague, Dr. Rees, she left the hospital, but returned in an almost dying state in five months, and was shortly afterwards tapped again. The fluid soon re-collected, and on November 2nd, paracentesis abdominis was performed a third time, and thirty-seven pints were removed. On the 5th, the peritoneum was filling rapidly, but the patient sank from exhaustion. A dull, heavy pain had preceded the swelling of the abdomen, and five months afterwards was followed by anasarca of the legs. The skin was dry and sallow; the body was wasted; there was no jaundice; the urine was healthy and free, and menstruation regular. The *post mortem* examination was made by Dr. Moxon. The pleura presented acute recent inflammation at the lower part of the right lung. The lungs were collapsed—partially carnified. The bronchi were healthy; the heart small and healthy. The abdominal cavity contained turbid serum. The peritoneum showed subacute peritonitis, long shreds of lymph passing between the coils of the intestine. The intestines were gathered together in front of the spine; the omentum was thickened, and was drawn up; the stomach, colon, and liver, were adherent; and the gall-bladder was adherent to the duodenum. The liver was fatty and softish. Many lobules were wasted; some were small, others were large and pale from fatty degeneration. At the back and under surface of the liver was a large apoplectic cyst, distending the hinder and middle part of the organ, so as to deflect the cava. The cyst was placed beneath the hepatic capsule; it was of the size of an orange, and contained laminated clots. The neck of the cyst was like a flattened tube towards the fissure of the liver, and it extended to the region of the head of the pancreas: at that part an old dense portion was found to be surrounded by thick tissue, and was connected with the portal vein. The portal vein itself was occluded throughout the liver; and the fusiform aneurism of the vein had given way, forming a blood-cyst. The walls of the aneurism were of stony hardness from calcareous patches. The stomach was covered with thick mucus. The spleen was very large, and contained embolic patches. The kidneys were pale; the genitalia were normal.

In this remarkable case there was no true cirrhosis, but the liver was fatty and degenerated.

Fibrous bands sometimes extend throughout the liver; or, if local obstruction have taken place, considerable depression and pits are observed upon the surface, indicating fibroid contraction and atrophy.

A more frequent cause of portal obstruction is inflammatory change in Glisson's capsule, the chronic form of which is called *cirrhosis*. We have already adverted to the contraction of the liver which follows venous congestion; this kind, however, is due to *inflammation* of Glisson's capsule and the tissues connected with it, and must be distinguished from a *third* form of cirrhosis—namely, *senile fibroid degeneration*. Before speaking of cirrhosis, it may be well to refer to the *hepatic artery* and to some pathological considerations connected with it, for the hepatic artery is essentially connected with the early stages of inflammatory cirrhosis, although the portal vein is secondarily affected, and causes many of the prominent symptoms of the disease. The hepatic artery is probably concerned in all acute inflammatory diseases of the liver; it receives a large supply of nerves, and the pressure of blood is greatly affected by the division of the nerves in the neck, according to the observations of Cyon. The capillary branches of the hepatic artery are largely distributed in the capsule throughout the liver; and whilst it is probable that direct absorption takes place by means of the branches of the portal veins in the stomach and intestine, the glandular structure of the liver is thereby irritated by acrid ingesta. Still, very many of the symptoms of ordinary hepatic disturbance are due to the vaso-motor nerves upon the vessels; if the nerves become paralysed, there is undue congestion; and we believe that we are warranted in this opinion from the phenomena observed in other parts when the sympathetic is divided. Congestion of the hepatic capillaries is followed by the effusion of serum, by oedematous swelling, by the effusion of fibrin, and by alteration in the secreting power of the hepatic cells. These are the stages of cirrhosis; the congestion of the vessels and effusion of serum causes swelling and enlargement of the whole gland; and it is probable that in inflammatory cirrhosis increase in size *always* takes place at an early stage. This inflammatory product extends as far as Glisson's capsule to the lobules themselves, and in many instances the fibroid capsule of the liver also takes part in the morbid process, and perihepatitis is the result. The tissue forming Glisson's capsule does not in man so completely separate the lobules

one from another as in some of the lower animals; but we find it permeating between groups of acini, and probably in a lesser degree between every lobule. The inflammatory product thus diffused throughout the gland becomes more fully organised; it contracts and becomes firmer, and most important secondary changes ensue. As to the changes in the lobules themselves: the cells waste, at first towards the periphery; fibroid tissue may be seen even between the cells, or rather between small groups of them; clusters of acini are separated and give the gland a granular appearance; and, as larger groups stand out on the surface from the contraction of intervening fibroid tissue, the more distinct irregularities are produced, and the hob-nail liver is the result. Similar effusion and contraction takes place in the larger portal canals, and the obstruction of the portal veins becomes extreme; the liver is wasted, its colour is changed, and it is paler; its edge is rounded, its surface roughened, and the gland has a rounded appearance. The wasting in the gland is not uniform. Sometimes it is more marked in the left lobe, sometimes the margin of the right lobe; but the wasted appearance is greatly altered if fatty degeneration be associated with the fibroid disease, and if the tissues are stained from jaundice. In many cases of cirrhosis the capsule of the liver, continuous as it is with Glisson's capsule, becomes inflamed, fibrin is effused, the capsule is thickened and opaque, and frequently a thick covering is spread out upon the serous surface; this fibrin may be peeled off; it forms adhesions with adjoining tissues; it becomes vascular in character, and sometimes presents a hard and nodular appearance. The local obstruction of the portal circulation in cirrhosis produces enlargement of the spleen, which is often covered with a thick fibrinous investment. The branches of the vena portæ are greatly distended—sometimes to actual rupturing of capillary vessels—and hæmorrhage takes place from the stomach, or from the hæmorrhoidal veins of the rectum. As the obstruction increases, serous effusion into the peritoneal cavity and ascites supervene; and it must always be borne in mind that the capillary vessels of the peritoneum and mucous membrane are in a state of intense congestion. This congestion is important, for it interferes with the absorption of medicines, and diuretics, etc., are powerless; and, again, if paracentesis be performed, there is great danger of peritonitis. Another result of this long continued venous obstruction is hypertrophy of the coats of the vena portæ, to which I have previously referred.

It is a great mistake to regard cirrhosis as a merely *local* disease; it must be considered in its general relations, as well as in reference to those local changes which arise from the contraction of the liver-structure. It is essentially a wasting disease; changes take place in other structures; there is atrophy of the cerebro-spinal system, the lungs often become affected, and we observe fibroid disease of the lung and true phthisical disease (for it is a great mistake to suppose that excess prevents phthisis). Again, the kidneys are often involved, and renal disease constitutes a most serious complication in cirrhosis of the liver. A patient with sallow countenance, bloated, but pale, with enlargement of the liver, with loss of appetite and impaired digestion, nervous exhaustion, and depression, may have organic disease at an early stage, and we may be hopeful if the patient will manfully stay his evil course; but, if with these symptoms we find the urine albuminous, and there be attacks of momentary loss of consciousness or vertigo, the condition is infinitely more serious. In senile cirrhosis, a contracted state of the kidney is frequently observed; but reference is made to the acute inflammatory congestion from great excess. It is quite true that the wasting of cirrhosis is greatly increased by the imperfect absorption of nourishment, and by the defective digestion; but this will not explain the whole of the symptoms.

A patient affected with cirrhosis often presents a wasted appearance: the skin is dry and harsh; the capillary vessels on the cheek are enlarged; the eyes are sunken; the nervous power and mental courage are lessened; the gait is less vigorous; the digestion is impaired; the appetite is dainty; the tongue is injected at the tip, irregularly furred, and, at later stages, becomes red and irritable, or aphthous; there is flatulent distension of the abdomen, sometimes heartburn and acid eructations. Vomiting and irritability of the stomach are often present at one or other stage; the bowels are irregular, confined or loose, and often with symptoms of catarrhal irritation of the colon; hæmorrhoids and loss of blood from them are not unfrequent. The urine is scanty, high coloured, having a deposit of lithates, or of uric acid, or of brighter coloured purpurates; and with nitric acid the urine assumes a very deep tint. On the surface of the skin spots of purpura are often observed; and if there be any ascites and anasarca, the veins on the surface of the abdomen are enlarged, the superficial epigastric joining the mammary. The countenance is often sallow, but jaundice is not a constant symptom of cirrhosis; pressure on the bile-ducts or catarrhal inflammation of these ducts may, however, induce it. The blood in

cirrhosis becomes changed in character; there is diminished power of coagulation, and if epistaxis occur, it is with difficulty checked; and in former medical treatment, when leeches and the cupping-glass were more frequently used, the persistent oozing of blood sometimes became a most serious question. Ascites then takes place, and afterwards anasarca of the lower extremities; but the legs in cirrhosis are often found to be small and withered, without any dropsical swelling. If there be inflammatory disease of the surface of the liver, pain in the side and pain in the shoulder are often present. Cirrhosis occurs at a very early period in life, when the wretched parent has allowed alcohol to poison the child; one occurred at Guy's even at 7, others at 8 and 10 years of age; but it is very difficult to state the period to which it may last. Some persons of intemperate habits at an advanced age have cirrhosis; but it is impossible to decide when the disease commenced. So also as to the prognosis: at an early stage, if the patient will submit to treatment, the disease may certainly be checked; and, even when contraction has taken place, our means of producing alleviation may be effective; but when the disease has advanced to a chronic stage, and when exhaustion is indicated by attacks of bronchitis and of pneumonia, the prognosis is very unfavourable. It is also a very unfavourable sign in cirrhosis, when the appetite having completely failed, the bowels become irritable, and the tongue has a morbidly and clean red appearance. The association of renal disease, as we often find in senile cirrhosis, is a very serious complication; for it not only shows that another most important excretory organ is affected, but that degeneration arises from general senile change; in these cases the patient is very likely to become comatose. The diagnosis is, however, at any early stage obscure, and when the disease is unattended with inflammatory symptoms on the surface of the gland, the onset of the malady is very insidious; it is often by a process of exclusion that we are led to suspect cirrhosis. Wasting with ascites, without pulmonary or cardiac disease, when no cancerous disease can be detected, when the liver is diminished in size, and there is no albumen in the urine, indicate cirrhosis; and, if with these symptoms we find enlargement of the abdominal veins and disordered digestion, we may strongly suspect chronic inflammatory disease of the liver; but pressure on the vena portæ from enlarged glands, etc., may cause ascites; it is sometimes very difficult to distinguish the ascites from small cancerous tubera upon the peritoneum from cirrhosis, and so, also, chronic disease about the uterus and ovaries. A case of that kind occurred under my care in the clinical ward at Guy's three years ago.

An old woman was admitted in a prostrate condition with ascites. She had pain in the right side and in the region of the sigmoid flexure, but there was no indication of thoracic disease; the pulse was regular; there was no cough, no bruit, and no abnormal physical signs; the mind was clear; the urine was healthy; there was no uterine discharge, and no complaint of "bearing down," etc. The distension of the abdomen, evidently from fluid, with pain in the right side and in the region of the sigmoid flexure, were the only signs of disease beside the general weakness. Cirrhosis was regarded as the probable cause of the ascites, especially as we had evidence of some perihepatitis in a friction-sound audible over the region of the liver. She was allowed to sit up, and soon afterwards became faint, not, however, having left her bedside; increased exhaustion supervened, and she died on the second day. There was some thickening of the peritoneal covering of the liver, and so far the diagnosis was correct, but the disease was uterine; there was cancerous disease of the body of the uterus, with but little enlargement; the os uteri was blocked up so that there was no vaginal discharge; local inflammatory disease and suppuration had supervened in the walls of the uterus, and the walls gave way, causing fatal peritonitis. The ascites was atrophic in character. Another form of disease which is apt to be mistaken for cirrhosis is the ascites arising from carcinomatous tubera upon the peritoneum. The disease is gradual; it occurs after middle life, inflammatory disease of the surface of the viscera is set up, and it is only by close attention to the general symptoms that diagnosis may be correctly arrived at.

In the treatment of cirrhosis, the co-operation of the patient is essential for success; for if after partial improvement there is a return to injudicious habits and to excess, the disease soon makes rapid progress. At an early stage, the cautious use of mercurial medicines lessens the engorgement of the portal system, and promotes the absorption of fibrin; but it is most important to bear in mind that, in proportion as general degenerative changes take place mercury is injurious, and when the powers of digestion are thoroughly impaired, mercury increases the weakness of digestion. The long continued use of iodide of potassium is often of service, but generally it is discontinued as soon as partial relief is afforded, instead of continuing the remedy for several months. The hydrochlorate of ammonia has been recommended, and so, also, the bromide of potassium. If, however, there be great exhaustion with ascites, these remedies are ineffectual, and in no disease is there greater

disappointment in the action of diuretics; they are valueless, and are given, the one after the other, without effect. The reason is patent: the medicine is not absorbed; the disease produces distension of the vena portæ, and the remedies do not enter the blood nor reach the kidney. In this state, I have often found more benefit from quinine with mineral acids, and from those means which are likely to improve the general health of the patient, than from measures more directly calculated to promote the elimination of effused fibrin in the glandular texture.

An important question arises in cirrhosis as to the propriety of withdrawing the fluid by tapping; and I believe that the operation is often deferred too late. It is quite true that at an early stage we are anxious to employ other measures less alarming to the patient; but in several instances in which it has been performed early, patients have soon recovered without the re-collection of the fluid, whereas in the later stages paracentesis is always attended with danger, for the peritoneal vessels are intensely congested, and peritonitis is easily induced. Many patients with chronic cirrhosis die a few days after tapping. Peritonitis does not in these cases produce pain, but rather typhoid exhaustion. The continued use of saline aperient waters is often attended with the greatest benefit in this form of chronic disease of the liver.

In cirrhosis, the disease is diffused; but sometimes a hard fibroid mass is situated in one or other part of the liver, or on its surface, local in character and closely resembling syphiloma. In these cases there has been a local source of irritation or injury at the part. In reference to tapping at an early stage of cirrhosis, I may advert to an instance which was lately under the care of my colleague, Dr. Rees, and myself.

Mary C., aged 29, was admitted into the Clinical Ward on October 27th, 1871. She had been a servant at eating-houses for ten years, and had been in the habit of taking very freely of beer, and sometimes of spirits. Two months before admission, her appetite failed; there was nausea and occasional vomiting. Two weeks later, the abdomen and then the legs began to swell; the bowels were constipated; aperient medicines produced vomiting, and she brought up half a pint of dark blood. She had suffered from hæmorrhoids. The patient was well-nourished. There was considerable ascites and oedema of the parietes, as well as of the lower extremities. There was no enlargement of the veins; there was tenderness over the region of the liver, and the dullness was increased. She had not menstruated for two months. There was a blue line along the margin of the gums; the urine was scanty and high-coloured; the specific gravity 1021; it was free from albumen. The bowels were constipated. Purgatives were used freely; but, as the quantity of fluid increased, on November 9th seventeen pints of fluid were drawn off, with great relief to the patient. Some fluid re-collected; but, under the use of the perchloride of mercury, and afterwards of iodide of potassium, it disappeared. The whole of the oedema ceased. Steel was afterwards given, and she left the hospital, nearly well, at the beginning of the present year.

It may be said that mercurials and iodide of potassium alone would have cured this affection—and we certainly could not state that the disease would not have been so cured—but the paracentesis at an early stage certainly promoted recovery; it saved much time, and spared the patient a long continuance of medical treatment. Whilst adventing to this instance of disease, I may mention that, whilst cirrhosis is especially an inflammatory disease of Glisson's capsule, there is a condition in which the glandular tissue of the liver is also involved; the liver becomes considerably enlarged—often permanently so; its tissue is indurated; and this form of disease is, at an early stage, with great difficulty distinguished from early cirrhosis. In this state there is greater sallowness than in cirrhosis, or even jaundice: it has been called inflammatory induration.

The inflammatory disease in Glisson's capsule, of which we have spoken, is fibroid in character; but we sometimes find that acute suppuration follows the course of this fibrous tissue or investment, and extends from the transverse fissure almost throughout the liver. The symptoms of this state are obscure; they are typhoid in character; and in every case that I have seen have been connected with abscess or with extension of disease from the lesser omentum, or with disease of the bile-ducts. This condition may be connected with an abnormal state of the lymphatic vessels and glands, for numerous lymphatics pass freely within Glisson's capsule. Perihepatitis is also productive, in some instances, of a local collection of pus on the upper surface of the liver.

Cirrhosis may be a partial as well as a general disease; and we sometimes find that fatty degeneration is present, or that some portions of a cirrhotic liver are involved in lardaceous degeneration. Acute yellow atrophy is sometimes associated with cirrhosis, or acute inflammatory jaundice.

An old man aged 67 was admitted into Guy's Hospital on February 1st, 1854, and died on March 5th. He was an emaciated spirit-drinker, with ascites and with jaundice. His strength had only failed for three months, the date of the ascites and jaundice. The skin was hot and dry; the liver could not be felt; the legs were oedematous; there was a cachectic lichenous rash on the body; the urine contained colouring matter of bile; and there were several spots of purpura on the hands. On inspection, there were atheromatous condition of the vessels of the brain, and subarachnoid effusion; there was degeneration of the coronary arteries and of the muscular fibre of the heart. The peritoneum contained two gallons of clear serum, with flakes of lymph. The liver weighed 4 lbs.; its surface was roughened from contraction; its section was irregular, congested, and lardaceous. The kidneys were granular; their arteries rigid; their weight 7½ oz. On examining the acini of the liver, fibroid tissue could be seen between the cells of the liver in the lobules. This condition was one of general degeneration, and the cirrhosis was a part of that general atrophy.

The full elucidation of these diseases would be impossible in the limits of one lecture. I have sought to give such practical illustrations as may aid in the diagnosis, and to encourage the scientific treatment of those maladies which are especially connected with the vascular supply of the gland.

NOTE ON THE CURE OF INVETERATE ECZEMA IN CHILDREN BY VACCINATION*

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[From the Br. Medical Journal]

We are so often confronted by cases in which it is alleged that eczematous eruptions have been produced by vaccination because they have followed it, that it is somewhat encouraging to have met with two cases where the eruptions existed in unvaccinated children, had resisted all the usual methods of treatment, and finally were cured by vaccination.

The first was the child of a commercial gentleman of great intelligence, who allowed me to try vaccination after everything else had been done that could be suggested. It was a most obstinate case of eczema over the whole body, the scalp being the seat of its worst display. The glands of the neck were chronically enlarged, and at one time suppurated so seriously as to endanger the child's life. Temporary benefit was derived from change of air, but drugs had no effect. Acting on the usual rule, I put off the vaccination of the child for three several periods of nine months. The dread of vaccinating a case like this depends on the probability of the formation of glandular abscesses—an apprehension that certainly is not unreasonable. I told the father of the child that, if we accepted the risk of this misfortune, I believed vaccination might cure the child by exercising some influence on its nutrition. He agreed to the experiment; and, to diminish risk as far as possible, I used lymph which had passed through one healthy child from the heifer. The result was most remarkable, for in a few days a marked improvement was visible in the child; and in little more than three weeks all traces of the eruption had disappeared; save a roughness of the skin, which still exists. The hair grew rapidly on the scalp, and the child now is in all respects as fine an infant as I have ever seen.

At the same time I had under my care the child of a clergyman, for which many prolonged and various courses of treatment had been adopted ineffectually for an eczematous eruption affecting the whole body, but mainly the face and flexures of the joints. It was nearly two years old, and had never been vaccinated. I told the father of the case I have just related, and he consented to the vaccination. He has sent me the following note of the history. "To the best of my recollection, the symptoms of skin-disease in baby were first manifested when she was about two months old. The disease appeared in a virulent form for the space of nine months, at the end of which time she was vaccinated. After vaccination, the child improved rapidly, and in a month not a trace of the malady was left."

Two is too small a number from which to make a generalisation; but the consequent and antecedent had in both of these cases too immediate a connexion for me ever to believe that the cures were mere coincidences. In the last case, I used lymph directly from the heifer, kindly supplied to me by my friend Dr. Greene, of Friday Bridge. I have no doubt that this is more efficacious and safer than lymph which has gone through numberless children. The rationale of the cure can only be theorised upon, and this I do not care to engage in. Suffice it for me if I may induce others to try the remedy.

* Read before the Pathological Section of the Birmingham and Midland Counties Branch.

A LECTURE ON DISEASE OF THE VALVES OF THE HEART.

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The General or Functional Symptoms of Valvular Disease.—Prognosis.—Treatment.

GENTLEMEN,—In my last lecture (BRITISH MEDICAL JOURNAL, Jan. 13th, 1872) I described the physical signs, and discussed the diagnosis, of disease affecting the different valves of the heart. We have now to consider the *general or functional symptoms* of valvular disease. And first I may remark that disease of one or more valves, as indicated by physical signs, may exist for years, yet, so long as the walls of the heart continue to be well nourished, there may be few or even no symptoms of functional disturbance. This fact is so well known, that it is scarcely necessary to give examples; but I may mention that it has happened to me twice, in examining candidates for life-insurance, to discover the unmistakable signs of aortic regurgitation in men of middle age who believed themselves to be in perfect health, and who declared that they had never been ill. Moreover, in one of these cases the medical attendant of the applicant certified that he was sound and in good health. In each of these cases I chanced to hear that death had occurred within a few months after the discovery of the disease and the consequent rejection of the candidate by the insurance-office. It is a matter of common observation that, when valvular disease has been caused by an attack of rheumatic fever, the physical signs are unmistakably present; but years may elapse before any symptoms referable to the heart are complained of.

Amongst the earliest and most constant symptoms of valvular disease is an excited or irregular action of the heart on exertion, such as going upstairs or running or walking fast. With this there is often hurried and difficult breathing, the result of an impeded circulation through the lungs. As the impediment increases, the lungs become congested and oedematous; moist sounds are heard over the lower lobes; and the patient becomes liable to attacks of catarrh and bronchitis.

In the advanced stages, *hæmoptysis* is not an unfrequent symptom, the result of passive engorgement and rupture of minute pulmonary or bronchial vessels.

When we were discussing the subject of *vesicular emphysema* of the lungs (see BRITISH MEDICAL JOURNAL, June 27th, 1868), I explained to you the manner in which valvular disease of the left side of the heart tends to bring about that condition of the lungs.

In most cases of valvular disease there is a sense of uneasiness, and in some there is actual pain in the region of the heart. The pain varies much in degree. In some cases of aortic regurgitation, with dilatation and weakness of the left ventricle, severe pain comes on in paroxysms, and extends down the left arm, constituting one form of what is called *angina pectoris*. These attacks sometimes result in sudden and fatal syncope.

Most of the symptoms of valvular disease are the direct result of the interrupted circulation through various tissues and organs; this interruption of the circulation involving, as we have seen, a defective supply of arterial blood and an impeded return of venous blood. I have already described the result of this interrupted circulation upon the nutrition of the heart's walls, and I have just now spoken of its effect upon the lungs. The abdominal organs suffer from the same cause. The interrupted circulation through the walls of the stomach results in a defective or an abnormal secretion of gastric juice. The consequence is, that the food, not being fully acted upon by its natural solvent, decomposes and ferments. The patient is dyspeptic and flatulent, and the flatulent distension of the stomach increases the dyspnoea and the cardiac distress. A similarly impeded circulation through the kidneys brings with it a scanty secretion of urine (which is sometimes albuminous), a consequent