SOME MISCELLANEOUS CLINICAL EXPERIENCES.

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ALCOHOLIC CIRRHOSIS OF THE LIVER.

CASE I.—T. J., a widow, aged thirty-eight, of intemperate habits, was admitted to the Whitworth Hospital in October, 1903, where she remained under my care till her death in November, 1904. She complained on admission of swelling of her feet and abdomen, and of pain in her right hypochondrium. She had previously been subject to bronchitis. She had had four children, one still-born, the other three died in early infancy.

Patient was well nourished, but rather pale. Conjunctivæ of a yellowish brown hue. The abdomen was distinctly enlarged, the lateral superficial veins evident; it was very easily palpated, as the muscles were weak and the recti were far apart. The liver was found to be uniformly enlarged, extending down to the umbilicus, abnormally tough in consistence, the anterior edge feeling very sharp. The spleen felt double the normal size at least, its rounded border contrasting markedly with the sharp edge of the liver, with which it was in contact. The intestines were flatulent. There was no ascites present. The urine was normal, and contained no bile pigments. A blood examination showed moderate anæmia, but no other abnormality. The lungs were slightly emphysematous. The apex of the heart was displaced a little outwards. Over all the precordial region a systolic murmur could be heard, its point of maximum intensity obviously being the second left intercostal space, half an inch from the edge of the sternum. This murmur was not affected by respiration; it became slightly more distinct on exertion; it could be traced only a short distance from the apex into the axilla; and it retained these characters till death. The feet were slightly œdematous. The diagnoses entertained were:—

- (a) Passive congestion of liver and spleen, the result of failing right ventricle. This was excluded by the absence of any

considerable enlargement of the heart, a normal pulse, and absence of other signs of passive venous congestion.

- (b) Spleno-medullary leukæmia or Hodgkin's disease, excluded by the blood examination and the absence of enlarged glands.
- (c) Biliary cirrhosis, excluded by the absence of jaundice or of its history.
- (d) Malignant disease, excluded by the regularity of the enlarged liver and spleen, and the want of any evidence of a primary form. The diagnosis finally arrived at was "hypertrophic stage of alcoholic cirrhosis."

During the patient's stay in hospital, on three or four occasions subcutaneous and sub-conjunctival hæmorrhages occurred, apparently spontaneously. Thrombosis supervened in the left femoral vein. Attacks of vomiting were occasionally troublesome. The liver became, as time went on, slightly smaller in size and less smooth on the surface. Severe pain in the right shoulders was a frequent complaint. Three weeks before death I had carefully examined the patient's abdomen one morning, and had felt the large, firm spleen as usual. Within an hour afterwards violent hæmatemesis occurred. On then examining the abdomen the spleen could hardly be palpated, so much reduced in size and softened had it become. The liver, I am inclined to think, was also reduced in size, but this point was doubtful at the time. During the next few days the spleen gradually regained its usual large size. This diminution in the size of the spleen after hæmatemesis can readily be explained on the supposition that the bleeding occurred from some of the veins along the greater curvature of the stomach which are tributaries of the splenic vein. Hæmatemesis recurred several times, and was the immediate cause of death. Only in the last two weeks of life as a terminal condition did ascites develop.

Post-mortem, made six hours after death:—All parts very anæmic. Abdomen contained a lot of clear fluid. Liver weighed 1,030 grammes, typically hob-nailed; adherent to diaphragm. Spleen enlarged, 390 grammes; congested, but rather soft. A single red infarct in its anterior border. A small accessory spleen. The edge of the great omentum was adherent to brim and contents of pelvis, the possible cause of thrombosis of left femoral vein. The stomach contained blood, but no point in stomach or œsophagus could be located as its source, though death resulted

from hæmorrhage. The kidneys showed signs of early chronic interstitial nephritis.

The surface of the heart showed numerous "milk spots." The musculature was in a state of fatty degeneration, the muscoli papillares of the left ventricle showing fatty striation. The left ventricle was somewhat hypertrophied and dilated. The cusps of the mitral valve were thickened and shrunken at their free edges, and their chordæ tendineæ were shortened; mitral orifice was dilated, admitting three fingers with ease. The right side of the heart was normal in size, as were its valves. The aortic valves were normal; lungs, emphysematous and œdematous.

The points of interest in the case to me were:—

The spontaneous hæmorrhages, as in hypertrophic cirrhosis.

The reduction in the size of the spleen after hæmatemesis, proving the congestive origin of its enlargement.

The very late appearance of ascites.

The opportunity of verifying *post mortem* that mitral regurgitation can cause a murmur, with its point of maximum intensity at the tip of the left auricle—*i.e.*, in second left intercostal space, half an inch from sternal edge.

ALCOHOLIC CIRRHOSIS OF THE LIVER.

CASE II.—J. D., aged forty, admitted to Whitworth Hospital on December 17, 1904, where she remained till her death, six weeks later. A month before admission she complained of vomiting, swelling of the abdomen, and cough. A fortnight later severe diarrhœa came on. On admission the abdomen was much enlarged from flatulent distention, a circumstance which prevented the accurate mapping out of the liver and spleen. The lungs showed evidence of slight chronic bronchitis. The bowels acted six to ten times per diem. The skin and conjunctivæ were somewhat discoloured. Urine contained a slight trace of albumen. In the course of a fortnight some ascites could be detected, and coincidentally the diarrhœa ceased. Three weeks later it became necessary to tap the abdomen, and 105 ounces were drawn off. The cells in the fluid were mostly desquamated endothelium. A week later tapping again became necessary;

then vomiting became severe, and death ensued from exhaustion and heart failure.

Post-mortem.—A small, typically hob-nailed liver was found and an enlarged congested spleen. The stomach and transverse colon were densely adherent to the gall-bladder, the walls of which were thickened.

The points of interest in this case were:—The marked flatulence, the late appearance of ascites, and the course of the temperature, which varied between sub-normal and 103° F. in a most irregular fashion. This temperature, the distended intestines which marked the real size of the liver and spleen, the violent diarrhœa, at first led to the erroneous diagnosis of some form of ulcerative enteritis, probably tubercular.

ALCOHOLIC CIRRHOSIS OF THE LIVER.

CASE III.—A country girl, aged thirteen, when admitted to hospital, was found to be suffering from ascites, enlarged superficial abdominal veins, uniformly enlarged liver and spleen. The diagnosis of alcoholic cirrhosis of the liver was arrived at, but on questioning the patient as to what stimulants she used she declared herself an absolute teetotaller. On further investigation the fact was ascertained that for two years before admission she had almost constantly been taking a medicine of herbs concocted for her by her mother. What the indications for this mixture were I could never precisely learn, but its composition was a decoction of dock leaves, with whisky generously added. This patient died in about a month from admission. An autopsy revealed a hob-nailed liver with its usual train of morbid anatomical findings.

BILIARY CIRRHOSIS OF THE LIVER.

CASE IV.—R. W., a farmer, aged twenty-one, previously healthy, and always temperate, in July, 1904, first noticed his urine becoming very dark in colour. At about the same time he noticed his eyes to be of a yellow colour, his skin yellow and itchy. After his skin got yellow he suffered from occasional attacks of vomiting, acid eructations, and a slight dull aching in the right hypochondrium. He was admitted to hospital four months later, in November, 1904. He was then found to be

well nourished and muscular. Moderate jaundice was present. The liver was thought to be enlarged on percussion, yet could not be palpated. The spleen was slightly enlarged, but a muscular abdominal wall prevented it from being felt distinctly. Pulse was 60; temperature subnormal. Urine dark and containing a quantity of bile pigments. The stools were acholic, abundant and clay-coloured. The bowels acted regularly. Blood was normal. By the end of December patient left hospital of his own request, between five and six months after the onset of jaundice. By that time the liver could be made out to be distinctly and uniformly enlarged by palpation. The spleen also could readily be felt enlarged. There was no ascites. The jaundice was as intense as on admission. The stools had, as a rule, remained acholic; occasionally they contained some bile colouring.

I made the diagnosis of biliary cirrhosis of the liver on the following data:—Jaundice persisting nearly six months, a gradual and uniform enlargement of the liver and spleen. Though such a condition of the stools as in this case more frequently indicates obstruction of the main bile duct than obstruction of a multitude of smaller bile ducts as from biliary cirrhosis, yet others have found acholic stools in association with hypertrophic cirrhosis. This diagnosis was greatly strengthened by the evident increase of the size of the spleen—a most important point.

ABDOMINAL ANEURYSM.

CASE V.—M. H., aged fifty, by occupation a butler, was admitted to the Whitworth Hospital complaining of attacks of violent pain in the left lumbar region extending into the left groin. He stated that, with the exception of constipation, he had always enjoyed good health till about three months previously. From that date he began to experience some pain in the left lumbar region, tending to radiate towards the middle line in front, but as it was not severe he did not seek medical aid. Four days before admission he got the first attack of violent pain which made him feel extremely ill, and incapacitated him from all work. The attacks repeated themselves several times before admission; on each occasion the pain radiated to the groin, and was most intense in that situation. On admission the patient was observed to be pallid, and wore an expression indicative of suffering. On examining the abdomen by palpation a tumour

was detected in the left iliac fossa. Tracing it inwards from this point it extended almost to the middle line, whilst in an upward direction it extended under the arch of the ribs, but receded from the middle line. The right border of the tumour was well defined, especially below, where it felt firm and almost nodular. The left border of the mass could not be limited by palpation. Doubtful fluctuation was detected, but no pulsation whatever. On percussion the tumour was absolutely dull, its area fusing with those of the left kidney and spleen. Dulness was also present at the base of the left lung behind, where the breath sounds were diminished. These signs gave rise to the suspicion that the tumour, whatever its nature might be, was pressing the left cupola of the diaphragm upwards. The left loin did not visibly bulge, though the skin there was œdematous. The patient was unaware of the presence of the tumour, so could offer no clue to the duration of its existence. No signs of aortic aneurysm, either thoracic or abdominal, were detected, despite careful examinations with that object in view. Blood films were made which negatived a suspicion of spleno-medullary leukæmia. The urine was normal.

The diagnosis considered most probable was renal calculus leading to hydronephrosis of the left kidney, although the urine contained neither albumen, blood nor pus. The patient lived for seven days in hospital, each evening getting a violent paroxysm of pain relieved only by morphin. After each attack of pain it was noted that the swelling had extended and was tenser than before. Under these circumstances I advised the patient to submit to an exploratory operation. This he refused to do. He died a few hours later, just as the proposed operation would have been undertaken had he submitted.

Post-mortem.—An aneurysm about the size of a small fist, constricted in its middle by the aortic aperture in the diaphragm, was found. It had ruptured into the abdomen extra-peritoneally. An enormous amount of blood clot was found stripping the peritoneum from the post-abdominal wall down to the left iliac fossa, from the under surface of the diaphragm, and even from the left anterior abdominal wall. The left kidney was embedded in the clot, otherwise it was normal. The blood had not penetrated between the spleen and its peritoneal capsule. Nothing else abnormal was found of any note.

The interest of this case, apart from the interest always attaching to an autopsical correction of a clinical diagnosis, lies in the fact that a man could survive for eleven days with a ruptured aneurysm of the abdominal aorta, and for part of this period be able to get about.

A CASE OF PAROXYSMAL TACHYCARDIA.

CASE VI.—P. R., a labourer, aged sixty, was admitted to the Whitworth Hospital in December, 1904. He stated that four years previously he began to suffer from attacks of palpitation of his heart. They occurred once a month at first, lasting about two hours, but gradually became more frequent and of longer duration. These longer attacks were often interspersed with ones of momentary length. Patient stated that the attacks always began with a burning sensation in the neck, which rapidly travelled to his heart, when instantly his heart began to beat violently and rapidly. He was conscious of the great rapidity. The attacks were always accompanied by lightness in the head. Having had good results in another case of paroxysmal tachycardia by administering bromide of potassium, I at once put this man upon the drug. During his stay in hospital on this occasion the attacks were all short, and gradually grew less frequent, finally disappearing. How much of his relief was due to the bromides and how much to the rest and good hygiene it is difficult to estimate. He left hospital without giving me the opportunity of personally observing an attack. The resident staff on several occasions were present during attacks, and counted the heart beats at something over 200. As a rule, the attacks were so short that they were over before anyone could be summoned to his bedside.

The patient returned to hospital early in February of this year (1905), and stated that his heart was as bad as ever again, the attacks having again become longer and more frequent. Two days after admission, on visiting him one morning at 11 a.m., I made the following note:—Patient states that yesterday afternoon he had several short attacks of palpitation. At 8 p.m. the attacks became continuous, lasted all through the night, and only allowed one hour's sleep. He complains of pain below the left nipple and a burning sensation behind the sternum. He says

his head is light, but he is able to read a book. He looks calm, a little paler than usual, but appears in no distress. Extremities are warm and of good colour. On looking across his chest, uncountably rapid vibratory movements are seen of the precordial and epigastric areas. The veins of the neck are not distended, and do not fill from below. On counting the rapidity of the heart beats with a stethoscope they are 212 to the minute. Only the first sound can be heard at the apex, very short, yet distinct. At the base a very feeble second sound can be heard after the first sound. That the heart is really beating at this enormous rate is proved by the facts that the carotid pulse can be counted at 212, and precordial pulsation (more difficult to count) at something over 200. In the radial arteries only 86 *distinct* pulse beats can be counted per minute; they vary in force more than in rhythm. It is a matter of doubt whether feeble, rapid, indefinite beats may not be felt between the larger ones. The pulse in the brachial and femoral arteries is of the same character as that of the radials. Whilst percussing the heart to try to map out the area of deep cardiac dulness the patient said—"You will find my heart slower now." On immediately counting with the stethoscope the heart rate was found to be 96, and a minute or so later 84. The storm had suddenly passed, the rapid and violent heart's action subsiding in the usual manner by a sensation travelling from the nipple region to the neck. It is my belief that the heart was somewhat dilated during this attack. The attack lasted 15 hours. The patient was now given bromides again. For the next few days some short paroxysms occurred, but for the last nine days none have appeared. He is still in hospital.

Between the attacks the heart's rapidity is usually about 80. The heart cannot be demonstrated to be enlarged. The impulse cannot be felt. The sounds, though clear, are feeble, even allowing for a thick chest wall, and a prolonged rest in bed. The arteries are sclerotic, but not markedly so. The urine is normal.

In my opinion the paroxysmal tachycardia in this case is not purely of nervous origin, but is associated with some degenerative change in the myocardium.