England, its recognition having been due to the labours of French pathologists, especially to Hanot, Letulle, and Brault. It is certainly rare, and I have not seen a case, though the liver of one of my patients was described by the pathologist as tough. It is sometimes but not always accompanied by bronzing of the skin. The liver is almost invariably large, its surface is covered by small granulations, and its consistence is tough. On section the increase of fibrous tissue is very apparent. According to some authorities the new formation of fibrous tissue commences around the hepatic radicles, but in some cases at least the growth commences equally in the portal canals. It is frequently, but not constantly, associated with an abundant deposit of pigment both in the liver cells and in the newly formed fibrous tissue. It does not appear to give rise to marked venous obstruction, and has not been observed to be accompanied in any case by ascites.

The disease is rare, curious, and therefore interesting, but its clinical relations, except its association with bronzing of the skin in certain instances, have still to be elucidated.

This concludes the brief outline of the varieties of hepatic cirrhosis which I have ventured to bring before you. I have not adopted any anatomical classification such as hypertrophic and atrophic cirrhosis, because these are accidents, the most typical atrophic form—the alcoholic—being sometimes large, and the most typical hypertrophic—the biliary—being sometimes small. Nor have I been able to follow Charcot in classifying these varieties according to the details of their microscopical structure as multilobular, monolobular, and monocellular, because these divisions are purely pathological, and have no clinical significance whatever. I am strongly of opinion that in the present state of medical science etiology is the best basis for classification, as that can be appreciated at the bedside.

In conclusion, gentlemen, I would ask for your opinions and experience first on the clinical symptoms and pathological characters of the various forms; and, secondly, as to their prognosis and treatment.

ŒSOPHAGEAL VARICES AS A CAUSE OF HÆMATEMESIS IN CIRRHOSIS OF THE LIVER.

By T. STACEY WILSON, M.B., C.M., M.R.C.P.,
Assistant Physician to the General Hospital, Birmingham.

WITH ILLUSTRATIVE CASES BY

J. R. RATCLIFFE, M.B., C.M.,

Late Assistant to the Professor of Pathology, University of Edinburgh,
Pathologist and Registrar to the General Hospital, Birmingham.

I DESIRE in this paper to draw attention to a morbid condition of the veins of the œsophagus in cirrhosis of the liver which is not often described, but which, on account of the anatomical relationship of these veins, must often occur, and which has, moreover, a most important bearing upon the etiology of hæmatemesis in this disease.

I refer to the occurrence of a dilated and varicose condition of the veins at the lower end of the œsophagus. This dilatation is due to the development in this situation of a collateral circulation whereby blood from the portal vein can reach the heart without passing through the liver, thus relieving the portal engorgement which tends to occur in cirrhosis of the liver.

This collateral circulation may develop to such an extent as to prevent the occurrence of ascites in these conditions or to permit of its disappearance when it has once occurred, but the dilatation so occasioned renders these œsophageal veins liable to rupture and so to become the cause of hæmatemesis.

The nature of the circulation around the lower end of the œsophagus is such as to admit of an intercommunication between the blood in the portal vein on the one hand and that of the general systemic veins on the other, for we have here the veins of the cardiac end of the stomach belonging to the portal system while, those of the œsophagus belong to the azygos and inferior vena caval system of veins. Thus the capillaries in the mucous membrane at the cardiac orifice of the stomach are continuous with both systems. This can be proved by means of injections, either from the coronary vein of the stomach or from the œsophageal veins, and is well known. The particular channels whereby the anastomosis can take place may not be so well known, and I shall therefore describe them a little more in detail.

The blood from the cardiac end of the stomach is normally carried to the liver by means of the coronary vein, which runs from right to left along the lesser curvature and then turns back behind the stomach to join the portal vein just above its formation by the union of the splenic and superior mesenteric veins. A certain part of the blood is also taken by a smaller vein, which runs from left to right along the lesser curvature and may be described as the right or the small coronary vein, and which opens into the vena portæ near to the gastro-duodenal vein.

A third channel is that furnished by the vasa brevia of the splenic vein. The branches of these three veins intercommunicate to a greater or less extent, and those of the first and last named form a plexus which surrounds the lower end of the œsophagus and which forms the starting point of the anastomotic channel that opens up in cases of portal obstruction.

When the passage of portal blood through the liver is hindered, the blood finds an escape through the œsophageal veins, and there is therefore, to a greater or less extent a reversal of the normal flow in the veins mentioned, but mainly in the coronary vein

Fig. 1.—Diagram showing the course of the blood in the cases described. P.V., Portal vein with left coronary sending a branch up to the diaphragmatic and periesophageal veins; L.D.V., large diaphragmatic vein opening directly into inferior vena cava; L.I.P., left inferior phrenic vein opening into left suprarenal; P.P., periesophageal plexus in connection with the submucous and from which the intercostals take blood.

(that is, the left or great coronary vein). In cases where this vein is blocked by a thrombus the vasa brevia of the splenic vein take the main part in carrying the portal blood to the œsophageal veins.

Let us now consider the branches of the vena cava and azygos veins, whereby the portal blood finds its way to the heart under these abnormal circumstances. The plexus of veins before mentioned, which surrounds the cardiac orifice of the stomach, is more or less continuous with (1) the left inferior phrenic vein, which opens into the left renal, and so into the inferior vena cava; (2) it is continuous with a number of small veins which run in the substance and on the surface of the diaphragm, and open directly into the vena cava as it passes through this organ. These veins play an important part in this connection, owing to the short distance that the portal blood has to travel before reaching the main current in the inferior vena cava. In these cases of cirrhosis, etc., the whole surface of the diaphragm may be seen covered with a dense network of vessels, which coalesce to form four or five trunks of from one-tenth to one-sixteenth of an inch in diameter, and so empty themselves into the vena cava.

The plexus above mentioned is more or less continuous by

branches through the opening in the diaphragm with another plexus of veins which surround the œsophagus above that organ. The blood circulating in these latter veins is discharged into the general venous system by various vessels: (1) by the above mentioned short diaphragmatic veins; (2) by the œsophageal branches of the azygos minor; (3) by the œsophageal branches of the intercostal veins; (4) in the upper part of the œsophagus by branches of the bronchial and even the inferior thyroid veins.

This plexus may be called the periesophageal plexus on account of its situation external to the muscular coat of the œsophagus. Supplementing this channel which we have just been describing is another in the mucosa and submucosa of the cardiac orifice and œsophagus. This is of more importance from a clinical point of view than the periesophageal one.

The capillaries of this situation communicate, as before pointed out, with both systems of veins, and in cases of portal obstruction they become dilated to form an extensive venous plexus in the submucous tissue which reaches from the stomach for a considerable distance up the œsophagus, and empties itself by means of occasional larger branches which pierce the muscular coat into the periesophageal plexus, and so into the systemic veins. This plexus, owing to its situation, has been called the submucous plexus.

In this submucous plexus we have a communication between the two systems of veins which is normally of little importance, but which may become under pathological conditions of great importance. The tissues here are soft, and we find in cirrhosis of the liver and similar conditions the veins of the mucosa and submucosa opening up under the pressure of the engorged portal system until they become very much enlarged, and we can sometimes find two or more veins of one-quarter to one-third of an inch in diameter coursing in the mucous membrane of the œsophagus, and evidently taking a most important part in carrying on the portal circulation.

In this connection I should like to direct attention to the important part played by the sphincter muscle of the cardiac orifice of the stomach in regulating the amount of communication that takes place between the two venous systems at this spot.

In health, the action of this muscle is such, probably, as to prevent any considerable reflux of blood through this submucous plexus from the coronary into the œsophageal veins; but when the pressure in the portal system undergoes a pathological increase, this restraining influence is overcome, and the portal blood is enabled to find an escape by this route.

I judge that the sphincter has a restraining action of this kind from the appearance presented by the veins of this part when the portal circulation through the normal channels is obstructed. In such cases, so far as I have observed, the dilatation of the veins when present is always greatest below and above the sphincter, where, as within the area of the muscle itself, their place is largely taken by smaller but very numerous straight veins.

As soon as the possible existence of large and thin-walled varicose veins in the mucous membrane of the œsophagus is recognised, the possibility of their rupture, leading to extensive hæmorrhage into the œsophagus or stomach, becomes apparent, and the following cases show that this is actually the case, and that the rupture of such varices is in many instances the cause of hæmorrhage.

The occurrence of these varices used to be treated as a mere pathological curiosity, and they are scarcely ever mentioned in English textbooks, so far as I have been able to find; but I am firmly convinced that as soon as their real importance and the frequency of their occurrence is more generally known, a careful search will reveal them in almost every case of hæmatemesis from portal obstruction, and we shall hear much less than we have done in the past of a "general capillary oozing" as the cause of hæmatemesis in cirrhosis of the liver and allied conditions.

Let us now turn to the records of cases where hæmatemesis has been traced to the rupture of the œsophageal or gastric varices.

The first case observed, so far as I can find, was one by Fauvel in 1838, and from that date till 1870 there were nine cases published, including one by Dr. Bristowe,¹ which is the only case published in Britain, with the exception of one of the present cases, that I have been able to find. In this case no cause for portal obstruction was found, but as no mention is made of the portal vein there may have been portal thrombosis.

I do not, however, mean to imply that this condition was or is

unknown among British scientists, for it is dealt with fully in Ziemssen's *Cyclopædia*, and Professor Greenfield gave it as a cause of hæmatemesis in his lectures on pathology at Edinburgh University in 1882; and reference to it will doubtless occur in many of our textbooks, but these references will, I fancy, speak of it as a pathological curiosity rather than as a fact with important clinical bearings.

In the next decade, from 1870 to 1880, eleven cases were published, and several exhaustive papers were written on the subject, among which may be mentioned the article by Zenker in Ziemssen's *Cyclopædia*, which contains a good bibliography. A very good bibliography will also be found in a paper by Longuet in the *Union Médicale* of Paris, 1886, page 769.

During the last ten years there have been about twenty cases published where hæmatemesis (in cases of portal obstruction) was shown to be due to the rupture of varicose veins, but in several of these the obstruction was due to cancer pressing on the portal vein.

On looking at these statistics more closely, it is instructive to find that ten of the above cases were published by two observers. This means that those who once know how to look for the condition will find it very frequently; and in support of this contention I may state that in my own experience, which is limited to the observation of but four cases of hæmatemesis from cirrhosis of the liver, these varices have been demonstrable in each instance.

Of these four cases, the first observed was that published in the *BRITISH MEDICAL JOURNAL* for June 26th, 1886, by Dr. Saundby; and the last three are those shortly to be demonstrated to the Section by Dr. Ratcliffe, the pathologist to the General Hospital.

If, then, this condition be so common, why is it that it has been so frequently overlooked? There are two main reasons, I think, for this, namely:

First, the œsophagus is an organ that is very often not examined as a matter of routine, and I can quote no less an authority than Professor Bamberger, of Vienna, in support of this, for, in the discussion after a paper on this subject by Kundrat, in 1886, he gives this as the probable cause of his never having traced hæmatemesis to œsophageal varices.

The second reason is that when the thin-walled veins have collapsed there is extremely little superficial evidence of their presence, and even the most careful observer might be unable to detect them by inspection alone. Unless one of the veins be blocked by a thrombus, the only evidence of œsophageal varices may be one or two longitudinal folds of the mucous membrane, and a little dull violet staining something similar to that seen round varicose veins elsewhere. In some cases the only way to diagnose their presence with certainty is, after opening the œsophagus along its anterior aspect, to carefully search the cut edges for the cut end of a vein, and then inflate it with a blowpipe. On doing this, what previously appeared to be a normal mucous membrane may be found to be literally covered with dilated veins, some of them even a quarter of an inch in diameter.

It is sometimes extremely difficult to find a vein even with the blowpipe, and it may be necessary to make one or two small transverse incisions into the mucous membrane in the most likely looking places before an entrance can be obtained into the dilated but collapsed veins.

From the uniform way in which we have found this condition in every case of cirrhosis of the liver in which we have looked for it, we feel satisfied that if all such cases are examined with the care and precautions we have above indicated, this condition of the veins will be much more frequently found than has hitherto been the case, judging at least from the number of cases that have hitherto been recorded.

And now a few words as to the clinical symptoms. These are, I regret to say, very obscure; in fact there may be, and often are, none at all until a sudden and profuse hæmorrhage occurs, and this attack may be followed immediately by a period of several years' duration as free from symptoms as those preceding the attack. This is well illustrated by the history of one of the cases shortly to be demonstrated by Dr. Ratcliffe. The suddenness and copiousness of the hæmorrhage, together with frequently a freedom from admixture with gastric contents and an absence of any signs or symptoms of gastric ulcer, form a very characteristic group of symptoms, and one which may fairly arouse a suspicion of ruptured varicose veins. When in addition to the above there are signs or symptoms of cirrhosis, and more especially the

¹ *Pathological Transactions*, London, 1859.

disappearance of an ascites, the presence of oesophageal or gastric atresias may be diagnosed with considerable certainty.

The occurrence of vomiting is not invariable when the blood passes into the stomach, and then melæna is the only symptom. In this case the diagnosis of varices would not be so easy, although some authors have gone so far as to say that all cases of intestinal hæmorrhage in cirrhosis of the liver are due to the rupture of these varices. I have not known or read of such symptoms as pain or dysphagia being observed, but they might occur, and their presence might aid the diagnosis.

As to prognosis, persons suffering from this condition may live for many years, as in the case of a patient of Le Deberder's, who, after having twelve recurrences of the hæmorrhage in the course of fifteen years, finally succumbed to pneumonia. On the other hand, the patient may succumb in from a few hours to a few days from the onset of the hæmorrhage, and especially if it recur frequently. It is certainly a most serious condition, and the prognosis after any extensive hæmorrhage must be very grave on account of the liability to a fatal recurrence.

Treatment.—The correct diagnosis here is of great importance, and the main indication will be to keep the oesophagus as much at rest as possible, and to prevent as much as possible every act of swallowing, and so favour coagulation in the veins. I think that even the swallowing of ice is likely to do more harm than good, and I can remember one case of slight oesophageal hæmorrhage in a girl with some symptoms and signs of cirrhosis, probably due to scarlatina, where the act of swallowing ice was most evidently the cause of several slight recurrences of the hæmorrhage.

Another most important point in the treatment is that the routine treatment of arterial hæmorrhage by the administration of ergot should not be adopted. It cannot, so far as I can see, fail to do harm by driving the blood from arterial into the venous system.

It is, I think, a question whether we should not in these cases adopt exactly the opposite line of treatment, and by means of nitrite of amyl rapidly draw a large amount of blood into the capillaries, and I believe that the fall of pressure so obtained would more than counterbalance the increased permeability of the capillaries leading to the affected area.

In conclusion, while apologising for the want of clearness and fullness of detail caused by the narrow limits into which this paper has to be compressed, I hope I may have said enough to enable others to take up the line of investigation here indicated, and so lead to an important increase in the extent and usefulness of our knowledge of this subject.

CLINICAL CASES OF HÆMATEMESIS FROM OESOPHAGEAL VARICES.

CASE I.—J. R., aged 36. Admitted to hospital under the care of Dr. Saundby, September 20th, 1885, complaining of vomiting

blood. He had been addicted to alcohol for fourteen or fifteen years, and had had syphilis. Had always been fairly healthy till ten weeks previous to admission, when he vomited a pint of blood. The vomiting was preceded by a sensation of nausea for an hour or so. He felt relieved after vomiting, and was all right next day. The attack for which he was admitted commenced eight or ten hours previously with nausea and general discomfort, which culminated in hæmatemesis and melæna, for which he was brought to hospital. After admission he vomited 20 ounces of blood, which coagulated in black stringy clots. There was no ascites and no history of any. The liver was most markedly diminished, and there was scarcely any dulness to be detected on percussion. There was no return of the hæmatemesis, and he was discharged

on October 10th. He was re-admitted in six days' time with a recurrence of the hæmorrhage, possibly brought on by his intemperance on returning home. He vomited a pint or more of blood while under observation, and gradually sank from exhaustion six days after this, on October 23rd.

Post-mortem.—Body pale and bloodless; no jaundice.

The liver had shrunk away from the ribs, and its place was taken by the hepatic flexure of the colon. The surface was seamed and puckered by scars. No great increase in the interstitial tissue. There was well-marked amyloid reaction. An attempt was made to estimate the rate of outflow from the hepatic veins when water was injected into the portal vein, but, owing to the liver having been incised, this was not found possible. It was apparent, however, that a large quantity of portal blood had passed away from the liver by the subcapsular veins. These entered the lateral ligaments and were greatly dilated. The vein in the round ligament allowed of the passage of a No. 6 catheter. The stomach was greatly dilated and filled with dark brown altered blood. The cardiac end was rosy, and the rest was covered with thick blood-stained mucus. The intestines also contained altered blood. The sub-mucous oesophageal veins in the lower half of the oesophagus were enormously dilated and varicose, and, when injected, they were nearly the size of the little finger, but at the cardiac orifice they stopped abruptly, and their place was taken by smaller veins, as shown in the photograph (Fig.

Fig. 2.—Case I. Lower portion of the oesophagus laid open so as to show the varicose sub-mucous veins. One of them, in the upper part of the specimen, is thrombosed. In the lower part the veins are seen to diminish in size at the level of the sphincter muscle of the cardia.

2). One of these large oesophageal veins was thrombosed, and it stood out as thick as a lead pencil. No injection mass passed into it, as it was completely filled with clot for about two inches. On examining the mucous membrane, no distinct ulcers could be seen, but over the thrombosed vein there was an erosion, and it seemed very probable that there had been a small ulcer here which had become filled with clot.

CASE II.—F. M., aged 32, admitted to the General Hospital, under the care of Sir W. Foster, on May 14th, 1886, suffering from ascites and rather acute gastric catarrh, accompanied by slight jaundice and a spongy and bleeding condition of the gums. The liver was enlarged and there was a history of syphilis—patient having had a primary sore eighteen months previously.

After three weeks' treatment the ascites disappeared, and he was well enough to be made out-patient. A prolonged drinking bout, however, entirely neutralised the benefit he had received, and he was readmitted after ten days in as bad a condition as ever, and with a certain amount of anasarca added to his other symptoms. Treatment was of little avail, the jaundice persisted, the ascitic fluid quickly accumulated after removal, and he was rapidly weakened by recurrent acute attacks of gastric catarrh. He died six weeks after his second admission from a sudden attack of hæmatemesis.

Post-mortem.—Well built man, distinctly jaundiced with a purpuric eruption on the trunk and limbs. Abdomen much distended with a large amount of bile-stained serum. Epigastric and other superficial veins in the abdominal wall much distended. The liver was "hob-nailed," no perihepatitis. The veins running in the suspensory ligament were prominently distended. The stomach contained 3 to 4 pints of dark blood. The veins on the upper and lower surface of the diaphragm were much distended, tortuous, and varicose. They formed an anastomotic network around the cardiac end of the stomach and œsophagus. The vein along the lesser curvature of the stomach (that is, the great coronary vein) was much dilated, and the area from which it came deeply congested. On injecting it the injection fluid passed into a series of large veins at the cardiac orifice of the stomach, which opened into greatly dilated veins in the lower part of the œsophagus. In one of these dilated veins in the mucous membrane an opening was found that would admit a duck quill. The injection mass passed directly into these œsophageal veins, and showed their dilated and varicose condition. In addition to the above mentioned anastomoses the venules and capillaries of the peritoneum were markedly dilated wherever vessels of the portal system came into relation with those of the general venous system, as for example, along the line of insertion of the mesentery, etc. In this case we see that the collateral circulation through the œsophageal veins, the veins of Sappey and numerous smaller channels was just sufficient to carry on the portal circulation under favourable circumstances, as was shown by the fact that all trace of ascites had gone when he was first discharged from hospital. It was not, however, sufficient for a sudden emergency, and the disturbance of the circulation in the liver brought about by three or four days of drunkenness was enough to destroy the equilibrium and cause a rapid return of the ascites.

CASE III.—R. P., aged 46, coal dealer, admitted under Dr. Saundby, June 11th, 1890, died June 19th. Patient was admitted suffering from well-marked ascites and cerebral symptoms. He was a heavy drinker. There was no history of syphilis or any previous serious illness. The present illness commenced six months ago, previous to which time the patient had been quite well. The first symptom was an attack of hæmatemesis, the patient vomiting a pint and a half of blood, but since that time

there was no return of the hæmatemesis. From the time of the occurrence of this hæmatemesis till his death there were marked delusions and mental symptoms. For the last three months there was slight jaundice, and dropsy developed during the fortnight preceding admission.

On admission there was marked œdema of the legs and ascites. There was varicosity of the superficial veins. The liver dulness was decreased; the splenic dulness was not increased. On the 12th the abdomen was tapped and 252 ounces of fluid removed. The delusions were very well marked. There was no hæmatemesis, but the stools, which on admission had been light, became very dark for four or five days before death. After death blood came from the mouth and rectum during the washing.

Post-mortem.—The abdominal and both pleural cavities were full of fluid, the abdominal containing eight or nine pints, and the pleural three or four each. The liver weighed 3 lbs. 3 ozs., markedly cirrhotic, shrunken, and hob-nailed. Sappey's vein in the ligament was markedly dilated. No perihepatitis. The stomach contained half a pint of dark clotted blood. The mucous membrane was stained, somewhat thickened, but showed no ulceration. On looking at the mucous membrane of the stomach and œsophagus nothing unusual was noticed with regard to the veins. On further examination, however, a small punched-out ulcer, about the size of a pin's head, was seen on the posterior wall of the œsophagus, one inch above the upper border of the cardiac sphincter. On blowing into this with a blowpipe, keeping the nozzle fully a quarter of an inch above the mucous membrane, there was an almost immediate injection of the submucous veins with air. These veins were seen to be much dilated and varicose. These were afterwards injected with Prussian blue mass from the cut edge of the mucous membrane, and the right coronary vein was also injected. The submucous veins were markedly varicose, one especially, and into this the ulceration had taken place. The branches of the left coronary were also dilated and varicose, and from these branches near the cardiac orifice a large number of dilated, fairly straight, vessels were seen passing up over the cardiac sphincter to anastomose with

Fig. 3.—Case IV. Lower two-thirds of œsophagus with cardiac end of stomach showing great varicosity of submucous veins.

the varicose submucous œsophageal veins. The diaphragmatic veins were also much dilated and very varicose, while the azygos and intercostals were also slightly dilated.

CASE IV.—T. M., aged 63, admitted under the care of Mr. Chavasse on May 28th, suffering from an epithelioma of the forehead. Otherwise he complained of nothing, and seemed in such perfect health that the tumour was removed on the 30th. He got on perfectly well until June 12th, when he suddenly, and without warning, had a profuse attack of hæmatemesis. He gave a short cough, and then the blood rushed out of his mouth "as from a hose pipe," and he brought up 84 ounces of almost pure blood. The next day (13th) he again had a sudden attack of hæmatemesis, bringing up 48 ounces, and on the day following (14th) he again brought up 40 ounces of blood.

Two days after this last, he brought up between 2 and 3 ounces of blood, and he died on the 20th.

The previous history was that three years before, while apparently enjoying perfect health, he suddenly vomited half a pint of blood. He did not feel ill, however, and he did not stop away from work. Although there was a marked history of drink, he said he had never had indigestion nor had he ever suffered from an ache or pain. There was no particular tenderness over the stomach, nor any history of ulcer of the stomach.

Post-mortem.—Body of well-nourished man. No cedema or ascites. Liver weight 3 lbs. 6 ounces; markedly cirrhotic and hobnailed. The gall bladder was distended with bile. There was thrombosis of the portal vein, which extended from just above the opening of the splenic vein up into the smaller branches of the portal vein in the liver. This thrombus was of recent formation; it did not entirely occlude the vein, there was a channel left on the upper side of the clot between it and the vein wall. Sappey's vein was dilated, and the diaphragmatic veins were markedly so. The azygos veins were dilated. The stomach and intestines contained altered blood. The mucous membrane of the oesophagus and cardiac end of the stomach showed a brown discoloration, and it showed marked ridges. On the posterior wall of the stomach, immediately below the cardiac sphincter, there were two punched-out ulcers close together, each about one-eighth inch in diameter (see Fig. 3.) They extended through the mucous membrane, and had opened into one of the large varicose veins and on injecting the veins of the oesophagus and the left coronary vein, the injection mass simply poured out of these ulcers. After injection the veins were much better seen, and some not before seen came into view. The branches of the left coronary were much dilated and very varicose. Those passing up to the cardiac orifice to anastomose with the submucous oesophageal were exceedingly varicose, and into one of the largest the ulcers had opened. For about 8 inches up the oesophagus the submucous veins were exceedingly dilated and varicose, some standing out as thick as a lead pencil. Over the cardiac sphincter they were not so large, but gave place to a large number of dilated parallel veins. There was no ulcer of the stomach or intestines, and the mucous membrane was normal.

CASE V.—T. B., aged 45, admitted under the care of Dr. Rickards on May 19th, 1890, complaining of ascites, cedema of the legs, and epigastric pain. There was slight jaundice, the liver dulness was slightly decreased, and there was a history of heavy drinking. There was bleeding from the mouth and gums. There was no history of hæmatemesis, although when he vomited in the morning the vomit was streaked with blood from the mouth and nares.

During the whole time he was in hospital there was no mælena. The motions were yellow, and were mixed with a varying amount of red blood and clot from internal hæmorrhoids. There was no hæmatemesis whatever. The ascites became very marked, for which he was tapped. He died of exhaustion on July 5th.

Post-mortem.—The liver was markedly cirrhotic (alcoholic). Sappey's vein was dilated, so much so as to allow of the portal being injected from it. The left coronary vein to the cardiac end of the stomach was injected *in situ*. On removal and dissection of the organs, a large dilated and varicose branch of the left coronary vein was seen to pass upwards to the cardiac end of the stomach and into the substance of the diaphragm. Several branches of this vein passed through and anastomosed with the periesophageal plexus of veins above the diaphragm. In the substance of the diaphragm this branch was seen to anastomose directly with the dilated diaphragmatic veins, and was in direct communication with a vein as large as a crow-quill, which opened directly into the inferior vena cava as it passed through the diaphragm. The periesophageal plexus was very varicose for about three or four inches above the diaphragm, and resembled a bag of worms. On opening the stomach and oesophagus the mucous membrane was quite normal. The branches of the left coronary were dilated and varicose, and dilated branches passed from them to anastomose with the submucous oesophageal veins. These latter were much dilated, and three of them formed ridges as thick as a lead pencil, running upwards for about four inches and then tailing off. The most careful examination of the mucous membrane of the stomach and oesophagus failed to show any ulceration, and there was no escape of the injection mass, as there had been in all the other cases

where ulcers had been found before injection. The stomach and intestines contained no blood.

REMARKS.—In connection with these cases, we wish to draw attention, first of all, to the importance of the collateral circulation described. From the amount and dilatation of the veins, as seen on injection, it is probable that it is the most important means of relieving the congested portal system, and so preventing or diminishing ascites. The large amount of dilatation may be accounted for for the following reasons—(1) The veins are in soft structures; (2) it is the shortest route from the vena portæ to the cava; (3) it is nearest the source of resistance, that is, the liver.

That these dilated oesophageal veins are the most frequent cause of hæmatemesis in cirrhosis has been proved in these cases. For in the three cases of hæmatemesis and one of mælena there were three that showed distinct ulcers into the veins before injecting, and in the fourth the fact of there being an erosion over the thrombosed vein seemed to indicate that this had also been ulcerated into. In the last case (v) there was no hæmatemesis, with no mælena, and the most careful examination failed to show any ulceration.

With regard to ascites, it was absent in those cases which showed the most marked varicosity (Cases i and iv) and which had died of hæmatemesis. It was most marked in the cases which had no hæmatemesis (namely, Cases iii and v), and in which the varicosity and dilatation was not so marked. Thus this collateral circulation seems to play an important part in the prevention of ascites. It would also appear that when this collateral circulation is sufficiently dilated to wholly or partly prevent the ascites, then there is great risk of rupture of the oesophageal varices occurring.

This rupture is even more likely to occur in such cases if some increased resistance to the portal flow be superadded, as in Cases i and iv, in the first of which it was an excessive bout of drinking, and in the second it was portal thrombosis. The increased dilatation of the oesophageal and other veins, rendered necessary, brought about the rupture, and this would be very likely to occur during the strain of vomiting.

In these cases, at least, the theory of "capillary oozing" as the most frequent cause of hæmatemesis in cirrhosis is disproved. This theory is usually brought forward when the pathologist has carefully refrained from examining the oesophagus, and when he can find nothing to account for the hæmatemesis in the stomach. But the fact that in one case 84 ounces of blood were suddenly belched up, and that in three out of four cases distinct ulcers were found, seems to throw great doubt on this theory.

It is to be hoped that pathologists will look for these varicose ulcers, using the methods described, and I feel assured that if this is done, oesophageal varices as a cause of hæmatemesis will be found to be much more common than is supposed, and the theory of capillary oozing will gradually ooze away.

[NOTE.—I am indebted to Dr. Short, R.M.O., for the clinical notes of Cases iii and v, and to Mr. Heaton, R.S.O., for the notes of Case iv.]

Dr. W. R. THOMAS (Bournemouth), said that for many years, ever since Dr. Austin Flint brought before the notice of the Section of Medicine the utility of frequent tapping in the treatment of ascites, he had been in the habit of tapping frequently and with very great advantage. The thanks of the Section were due to Drs. Wilson and Ratcliffe for directing attention to the varicose condition of veins around the cardiac extremity of the liver and the presence of ulcers in this position, as this knowledge would help in the treatment of such cases, and showed that the very act of swallowing tended to increase the hæmorrhage. Cirrhosis of the liver was universally recognised to be a very serious disease, and one which, in the majority of cases, proved fatal. One writer stated: "It is rare for a patient suffering from cirrhosis of the liver to live longer than a twelvemonth after the symptoms have become so pronounced as to allow a diagnosis to be made;" another wrote: "When once distinctive symptoms have shown themselves the patient's days are numbered. He may, however, even then survive for a year or two." It was desirable, therefore, to consider what the symptoms were which preceded these distinctive symptoms, which presented themselves too late generally for treatment to be of great use. Before residing at Bournemouth he had been for many years physician to the Sheffield Public Hospital, and there had had opportunities on a large scale of dealing with a variety of cirrhoses of liver, in which treatment was of great use, and which treatment often cured. At the furnaces a vast number of men worked who were exposed to very

great heat, and who always, therefore, when at work were in a constant state of perspiration. Although many of these men drank oatmeal water and other fluids while at work, still a number of them preferred beer, which they took in immense quantities—one, two, or three gallons a day, or even more. When they presented themselves at the hospital they had furred tongues, yellow at the base, a congested condition of pharynx and tonsils, and a poor appetite. They complained of sickness, perhaps, in the morning, a feeling of weight and fulness in the epigastric and hypochondriac regions, uncomfortable sensations in the abdomen, generally diarrhoea, sometimes piles, which often bled, and occasionally slight hæmatemesis after excessive vomiting. The eyeballs were slightly congested, and occasionally a yellowish tinge might be seen when the lower eyelid was drawn down. The liver was enlarged, sometimes to twice its natural size; it was smooth, but more firm than and not so doughy as the fatty liver. These patients were for a time strong, healthy, muscular men, stout, large of build, and often imported from the country—from Lincolnshire and other counties. Owing to their occupation they were exceedingly apt to catch cold, and were carried off by pneumonia or other acute complaints. When an opportunity occurred of making a *post-mortem* examination the liver was generally found to be very much enlarged—sometimes twice as large as it was in health, the surface was smooth and firm to the touch, the edges were rather rounded, and in colour the surface was paler than in health. The cut surface was smooth, and palish yellow patches were to be seen, representing the lobules, which were much larger in size than the patches seen in the ordinary cirrhotic liver, and a greyish fibroid material filled up the spaces between the lobules. The surface generally was greasy. On making a microscopical examination the cells of the lobules were seen to be distended with fat, and fibroid tissue was present in abundance. Other patients of the same class, possessing similar physique, accustomed to the same habits, and of similar occupation, escaped these accidental acute diseases and lived on. If they pursued the same habits, the appetite failed, and spirits were often taken in addition to beer, the vomiting became more severe, hæmæmesis or melæna occurred, and diarrhoea or piles were troublesome. The patient became thinner and weaker every day—a mere wreck of his former self—suffering from abdominal dropsy, from cirrhosis, a jaundiced hue of countenance, and at last he died of some of the consequences of the disease. This affection he had observed in all classes. Merchants, manufacturers, and commercial travellers suffered in this way. He had seen young men start in life quite well, had had to treat them in a few years afterwards for the symptoms above described, and had found the liver enlarged. Later on in life he had had to treat the same men for cirrhosis of liver with abdominal dropsy. In such cases he had not had opportunities of making a necropsy, but had no doubt about the condition of the organ from examination before death. This disease was brought on by the patient drinking a quantity of alcohol largely diluted with a fluid containing sugar, gum, and other constituents. The alcohol taken in this form did not, in passing through the portal veins in the liver, irritate the tissues around them or stimulate them to increased formation of tissue to the same extent as alcohol diluted with water. The condition was not like the fatty liver, inasmuch as there was an increase of fibroid tissue between the lobules, which tissue ultimately contracted. It was not like hypertrophic cirrhosis ordinarily so called, inasmuch as the fibroid tissue did not permeate the lobules, separate its cells, and interfere with the secretion of bile very much. He believed that it was a liver caused by drinking beer, stout, or similar fluids, and one that would ultimately contract and cause the death of the patient. Hypertrophic contracting cirrhosis of the liver would be a suitable name for it.—Dr. C. R. DRYDALE (London) said that there were four causes to which cirrhosis was attributed: alcoholism, infinitely the most frequent; syphilis, rather infrequent as a cause; malaria; and heart disease—"cardiac cirrhosis." By far the most common cause of cirrhosis of the liver was the abuse of spirituous liquors, especially of brandy, whisky, rum, or gin; although wines and beer, if partaken of immoderately, often produced it. This was well shown by the perusal of the eighty-eight cases of hepatic cirrhosis published in 1890 by Dr. Eugene Williams, in the history of which cases wine played a prominent part. In a large number of cases of hepatic cirrhosis the exciting cause was not evident. Syphilitic cirrhosis had been recognised by early writers on syphilis—for example, Fallopius and Villa. Of late, Gubler (1852), S. Wilks, Virchow, and many others, had published cases of syphilitic

cirrhosis. It was almost always accompanied by some marks of tertiary syphilis in other parts of the body. Pain in the region of the right lobe was occasionally present, jaundice more rarely. Ascites was rarely seen. As to diagnosis, this disease was likely to be confounded with cancer of the liver in some cases, since nodules might be present in both these affections, but perhaps the prominences were softer than in cancer. The existence of other syphilitic symptoms might aid the diagnosis. It was successfully treated by large doses (gr. xx, t. d.) of iodide of potassium. The most curable of all the forms of cirrhosis of the liver was that caused by the irritation of alcoholic drinks; that form of it which was designated hypertrophic cirrhosis was, with its accompanying ascites, not infrequently cured on condition that the patient abandoned the use of alcohol. This was a new view of the disease, for cirrhosis of the liver used to be considered as certainly fatal. Ascites was curable even in the case of cirrhosis in its advanced stage; but in such cases the prognosis must be guarded, as relapses were frequent. The cases of cirrhosis which were curable were those of the hypertrophic kind. In this form there seemed to be less destruction of the liver cells than in the atrophic form. But even in atrophic cirrhosis, if not too far advanced, a seeming cure might be effected, if there was not too great a diminution in the urea excreted, and no great jaundice, fever, or weakness. The best treatment for all forms of ascites and cirrhosis was an entirely fluid diet, consisting of four or five quarts daily of milk, combined with frequent tapping (which should not entirely evacuate the cavity of the peritoneum), and the administration of iodide of potassium. Above all, alcohol in every form should be entirely proscribed from diet or from the remedies recommended, since the only hope for the patient was that a sufficient number of hepatic cells might remain healthy, and capable of carrying on the functions of the liver.—Dr. W. H. BROADBENT (London), admired the clear method in which Dr. Saundby, and the authors of the other papers, had laid the subject before the meeting. He was aware that varices had been discovered in cases of cirrhosis, but was not aware that they played so important a part as had been shown. He did not advocate the use of styptics in cirrhotic hæmorrhage, but the employment of lowering measures; to depress blood pressure, he suggested the use of amyl nitrite.—Dr. HANDFORD (Nottingham) hesitated to add to the already long list of varieties of hepatic cirrhosis yet one more. But if Dr. Saundby's classification were adopted, certainly enteric cirrhosis would have to be included. The changes were constant in all fatal cases, and occurred even in children of 10 years of age. This form of cirrhosis had been described and illustrated by the speaker in vol. xl of the *Transactions of the Pathological Society of London*. Changes in the liver were met with in certain percentages of cases of diabetes, but it was very questionable whether they should be called cirrhosis. Dr. Handford had seen a very well-marked specimen of this change in a diabetic liver, but considered the tissue to consist chiefly of the walls of capillary vessels. The question of primary chronic atrophy of the liver cells in the etiology of cirrhosis had not been adequately considered. The essential treatment of cirrhosis was suitable and limited dietary. Tapping was only the treatment of ascites, which might or might not be present.—Sir WILLIAM MOORE said that Dr. Saundby had stated that malarial cirrhosis occurred after repeated attacks of congestion of the liver arising from malaria. Sir William Moore, however, thought that malarial cirrhosis should not be added to the list, because it was doubtful whether there were any specific malaria, and if there were no malaria there could not be malarial cirrhosis. Secondly, he thought malarial cirrhosis should not be added because it was usually found that attacks of congestion of the liver arising in the tropics, not from malaria, but, as he contended, from chill, were generally followed by enlargement of the liver of the fatty or the lardaceous character, this being determined by the constitution of the individual. He would also like to ask Dr. Saundby if among the very early symptoms of cirrhosis he had noticed repeated headaches, inability to read or apply the mind, or numbness of the extremities of an intermittent character.—Dr. WARDROP GRIFFITH (Leeds) called attention to the other channels of collateral circulation in cases of alcoholic cirrhosis, and discussed especially the circulation through the parumbilical veins and the superficial abdominal veins. The establishment of collateral circulation, beneficial so far as the dropsy was concerned, was attended with the disadvantage, and even danger, of the products of digestion passing directly into the general venous system without being altered by the liver. Possibly some modification in the diet in such

cases might lessen the danger. The distinction of hypertrophic and atrophic was good if used without necessarily employing a different causation. "Hypertrophic" should not be used as synonymous with "biliary." The "publican's liver" was often a large one. The ascites was probably not due to purely mechanical causes. The extent of liver dulness was not by any means a good guide as to the size of the liver. Although not denying that in those exceptional cases in which the inferior vena cava was surrounded by liver tissue, oedema of the lower extremities followed as a direct result, he was doubtful if cirrhosis could ever be regarded as a mechanical cause of oedema of the lower extremities.—Dr. HUTCHINSON (Scarborough) drew attention to the very lengthy list of causes of ascites, and was very much relieved to find on listening to the debate that most of them had no symptoms, and that after all the alcoholic form was the one which practical men had to face. He drew attention to the fact that cirrhosis had a tendency to generally lower the general tone of health, and that therefore small congestions here and there, in the lungs or kidneys, were very difficult to cure, hence the temptation to send the cases abroad for change. He mentioned a case which had been sent abroad on this account. Sea-sickness brought on hæmorrhage which ended in death, and he appealed to physicians to go a little more into cases before with a light heart sending them across the seas.—Dr. KATCLIFFE said, in reply to Dr. Griffith, that Dr. Wilson and he had purposely refrained from mentioning the collateral correlations other than the important one through the coronary, œsophageal, and pericœsophageal plexus, into the diaphragmatic veins, and so into the inferior vena cava. This was mentioned because it was capable of very great dilatation from the shortness and directness of its course, and because ulceration of the varices of the submucous œsophageal veins was probably the chief cause of hæmatemesis.—Dr. SAUNDY, in replying, thanked the Section for the kind way in which it had received his paper, and expressed his gratification at the nature of the discussion which had been elicited. He agreed with Dr. Thomas in thinking that the symptoms he described were very common in the early stages of alcoholic cirrhosis, but he believed these symptoms to be due to gastro-intestinal catarrh, and that no one of them was directly evidence of the existence of cirrhosis. While agreeing, too, that cirrhosis due to beer drinking was perhaps more commonly attended by hypertrophy than that due to spirit drinking, he could not accept the view that this was a rule, as he had in his mind two cases of enlarged cirrhotic livers due to spirit drinking. With reference to the criticisms as to the introduction of scarlatinal interstitial hepatitis as a form of cirrhosis, he agreed that it was wrongly so placed, as it was not certain that it did lead to the naked eye changes generally associated with the term cirrhosis. It was introduced into the paper because of the probability that this condition might be the cause of the cirrhotic livers of unexplained origin met with in young children. He agreed with Dr. Handford and Dr. Griffith in thinking that the ascites might have to some extent an inflammatory origin, and he had put forward this view in the article on the liver contributed last year to Fowler's *Dictionary of Medicine*.

ANTIVIVISECTIONISTS AT FOLKESTONE.—A meeting was held at Folkestone last week, which was addressed by Mr. Berdoe, M.R.C.S., and Mr. B. Bryan, secretary to the Victoria Street Society for the Suppression of Vivisection. The former speaker concluded a long speech by saying that "he could not put his hand on any one great discovery in medicine that could not have been come at by clinical experiments." Dr. Tyson answered many of the arguments of the former speakers, pointing especially to the advances gained from vivisection; he spoke of the triumphs of brain surgery, of the detection of poisons that had killed human beings by experiments upon lower animals (as in the Lamson case); he contended that vivisection had done more for humanity than any other kind of experiment, and concluded by remarking that he believed the result of Koch's experiments would be that thousands, if not millions, of humanity would be thereby saved. After other controversial remarks by Dr. T. Eastes and Dr. F. Eastes, the meeting passed votes of thanks to the lecturers. The best thanks of the medical profession, however, are due to our three *confrères* for this excellent endeavour to stem the tide of prejudice and of credulity respecting this subject; which experienced lecturers know only too well how to stimulate in the minds of a non-scientific audience.

THE BRADSHAW LECTURE ON MODERN ABDOMINAL SURGERY.

*Delivered at the Royal College of Surgeons of England,
December 18th, 1890.*

By SIR T. SPENCER WELLS, BART., F.R.C.S.,
Surgeon to the Queen's Household.

[Concluded from page 1416.]

BEFORE I pass on I cannot avoid contrasting Matthews Duncan's conclusion respecting ovariectomy in 1857, a year before my first case, with the present position of the operation. My own completed operations alone amount to 1,230, with 19 additional operations for the second time on the same patient, or 1,249 in all. Several other operators, at home and abroad, can count their cases by the hundred; and it is certain that the immediate results are fully as satisfactory as those of any serious surgical operation. The subsequent history of the patients for several years after operation has been more fully and accurately obtained and recorded than can be said of any equally important operative work. Let me ask, could anyone have imagined that, in such a small hospital as the Samaritan, in little more than thirty years, there would be performed, as there have been, 1,378 cases of ovariectomy, with a mortality of only 14.13 per cent., or that, in the last four years, in 259 cases, there have only been 12 fatal, a mortality of only 4.40, less than 5 in the 100? It is equally remarkable that in successive series of 100 cases, and in successive periods of five years, from the earliest cases until now, progressive improvement has been more steadily maintained in this hospital than in my own practice.

UTERINE TUMOURS.

When I lectured here in 1878, on uterine tumours, and gave the history of the whole of my practice up to that time of their removal, or attempted or partial removal, very few surgeons here or abroad had much experience of the operation. Now, it has become, if not as frequent as ovariectomy, still an operation in which not only some British and American, but German and French, surgeons can tabulate their cases by the hundred. And some important practical questions, still undecided, may be discussed with the help of accumulated facts. Perhaps the most remarkable conclusion of any is that of the most successful of all operators who has had a larger experience of these operations than I or any other surgeon has had—Dr. Keith. His results are magnificent; yet after trial of Apostoli's method of electrical treatment, he wrote:—"So strongly do I now feel on this subject that I would consider myself guilty of a criminal act were I to advise my patient to run the risk of her life—and such a risk—before having given a fair trial to this treatment, even though I were sure that the mortality would not be greater than that which hysterectomy has given me in my private cases—under 4 per cent."

This is a declaration which must be regarded as phenomenal, coming as it does from a man who is known to observe scrupulously, to think calmly, to reason logically, to decide deliberately, and to act conscientiously. It ought to be, one would imagine, sufficient to check the folly of reckless, indiscriminating laparotomists, and to make their imitators hesitate before risking human life, and it gives matter for grave reflection to all those who have to bear the responsibility of advising and cautioning in such cases.

Dr. Keith tells me that he has not since done more than three hysterectomies, and for fibrocystic tumours only, and one castration. This persistent abstention implies his own emphatic condemnation of his former practice, which, as he says, always "vexed him with anxious doubts and fears," and at the same time it indicates hopeful confidence in the alternative method of treatment he has adopted. Under the circumstances an attitude of watchful expectancy is what is most fitting; while everyone who has gone through the trial of dealing with the perplexities of giving counsel and acting in these cases must hope that further evidence will establish