jected, and the pulse, which was about 180 at the end of the operation, came down to 120, and was much stronger. He appeared to be considerably relieved.

NOTES ON AFTER-TREATMENT.

January 23th, 1897. Patient looked much better. Temperature 98.6°, pulse 126, respirations 28. Wound dressed. January 29th, 1897. Wound was dressed and part of the gauze gently withdrawn. Flatus passed *per anum*. February 2nd, 1897. The gauze packing which had been shortened daily was wholly removed. Temperature fluctuating between 99° and 100°, pulse 100. Patient cheerful and looked well. There was no fullness of epigastrium and no tenderness. There was no marked anaemia. February 4th, 1897. Bowels moved.

February 5th, 1897. The temperature rose to 104° last night owing to a stitch abscess, although the remainder of the wound looked quite well. This was manifestly dependent on the operation having to be undertaken without time to prepare the skin properly. There was neither fullness nor tenderness of the epigastrium and no sign of intraperitoneal suppuration. The daily dressings were discontinued, and boracic fomentations ordered to be applied every four hours. February 7th, 1897. There was free suppuration around several of the sutures, which were all removed. February 11th, 1897. The wound was beginning to look better and there was less pus on the fomentations. February 14, 1897. Very little suppuration. February 16th, 1897. Wound doing well. Fomentations were discontinued and the wound dressed simply. February 25th, 1897. Wound almost completely healed.

March 4th, 1897. Wound healed. An abdominal belt was applied, and he was allowed to get up. March 6th, 1897. Patient was discharged to-day quite well.

REMARKS.

The nature of the injury is remarkable in that the perforating instrument, being somewhat blunt at the end, must have pushed aside the stomach and colon instead of perforating, though the fact of blood having been vomited shows that the stomach must have received the impact of the file, and have suffered injury to its internal coats. The mesenteric vein lying at the back of the abdomen should be one of the vessels least liable to injury, and probably could only be lacerated by a stab or gunshot wound. The extensive retroperitoneal hæmorrhage gave rise to the suspicion that the pancreas might have been injured, but on enlarging the aperture at the root of the mesentery made by the file, the freely-bleeding vessel was seen and easily ligatured.

Having arrested the hæmorrhage and cleansed the abdomen of blood and clot, the question arose in my mind as to the behaviour of the intestines under their new circumstances, for, in addition to the blood extravasated beneath the peritoneal coat, all the vessels must have been seriously congested owing to the return of blood along the superior mesenteric vein. Paralysis of bowel and peritonitis at least was to be feared—possibly gangrene—but, fortunately, neither complication interrupted recovery.

SALOL CALCULI.

By C. R. MARSHALL, M.B. VICT.,

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In connection with the recent communication of M. Brossard to the Académie de Médecine (March 30th),¹ the following account may be of interest.

Some time ago Professor Bradbury sent me an almond-shaped crystalline mass, possessing a yellowish colour and weighing about 1 gramme, for examination. It was said to have been vomited by a young lady who had been taking salol for some months. The drug had been ordered for intestinal flatulence, and had given marked relief. For nearly six months a cachet containing 10 grains was taken once or twice daily. Then attacks of severe colic, accompanied by vomiting, occurred, which nothing would relieve except the hypodermic administration of morphine. In one of these attacks the body in question was brought up, and at the time the statement was volunteered that similar masses had been frequently passed by the bowel. The substance had a salol-like odour, and gave the chemical tests of this compound. The melting point was almost correct, so that the substance was practically pure. The salol was stopped, and the patient improved. Dr. Bradbury tells me that no further attacks have occurred.

Brossard's case² was a neurasthenic lady, aged 45, suffering from gastric dilatation, with paroxysmal gastralgia, gastrosuccorrhœa, and hyperchlorhydria. Vomiting was frequent, and was sometimes accompanied by hæmatemesis. The patient refused lavage, and the treatment consisted in the administration of large doses of alkalies and a milk diet. The attempt at a milk diet produced febrile symptoms, and salol and calomel were

therefore given. The salol was administered in 0.5-gramme ($\frac{7\frac{1}{2}}$ grain) doses, and 4 to 5 grammes (62 to 77 grains) were given daily. After ten days (that is, after the administration of 40 grammes, rather more than $\frac{1}{4}$ ounce), the patient, who was habitually constipated, presented severe symptoms of intestinal obstruction. Purgatives, large enemata, the continuous current, were repeatedly tried, but without effect, and the symptoms continued for thirty-six hours. Finally, a motion was passed, and thinking that a biliary calculus might have produced the pain it was carefully washed. About ten crystals, weighing altogether 4 grammes (62 grains), were found, the largest of which weighed 1.8 gramme. Similar small calculi occurred in the two following motions. They all consisted of pure salol.

An interesting feature in the case I have mentioned is the occurrence of a calculus in the stomach. As the vomit was not bilious it is probable that this was the seat of formation. Girode,³ in a case of cholera which had been given salol two days before death, found two masses of salol, weighing 3 grammes, in the stomach *post mortem*, and as far as I am aware this is the only similar case on record.

The question of the formation of these calculi is an important one. Brossard consulted Pappel, a chemist, and they concluded, probably rightly, that it was inadmissible that salol could be decomposed into its constituents, and that these should unite again. Experiments were then made to determine if, in an acid or alkaline medium, in the presence of pepsin or pancreatin, salol could be dissolved without decomposition, and, on changing the medium, if it could be deposited anew in the crystalline form. But only negative results were obtained. It was then thought that on account of the constipation the drug would only be eliminated slowly, and that a new dose would come into contact with the decomposition products of the old ones, that is, with salicylic acid and phenol. The effect of these upon the physical condition of salol was then determined. Salicylic acid was without effect. A solution of phenol, however, caused the powdered salol to agglomerate, and after some time to become liquid. It did not mix with the carbolic solution, but assumed the form of a transparent ball. If now the phenol solution was replaced by water, and a crystal of salol added, the whole of the salol was converted into a crystalline mass. This displacement of the phenol may be regarded as analogous to its absorption in the body.

The theory seems to me weak in many points. The phenomenon described by Brossard cannot be obtained at ordinary temperatures in this country. It is quite true that salol will melt at a lower temperature in an aqueous phenol solution than in ordinary water, and this may account for the results obtained by Brossard and Pappel, whose experiments were carried out at Cairo. (In connection with the phenomenon it should be mentioned that phenol is capable of dissolving salol.) But even if we grant the results of those observers to hold at all temperatures it is difficult to accept the view put forward by M. Brossard. The most essential factor—the presence of sufficient carbolic acid to produce any influence—is improbable. Salol is only gradually decomposed in the intestine, and it is very probable that most of the carbolic acid formed is quickly absorbed. The explanation seems to me a simpler one. It is due to the fact that the melting point of salol (42° C.) is a little above the normal temperature of the body. Very often, especially after meals, the temperature of the stomach is above this point, and if salol is present melting of the crystals must occur. The fluid thus produced does not readily mix with the food, and when the gastric temperature sinks to, or better, as after partaking of cold water, below the normal, recrystallisation occurs. The mass thus produced is not composed of minute individual crystals as in the first instance, but is in the form of single solid lumps made up of the crystals in close union. That this may occur can easily be demonstrated by a few test-tube experiments. The solidification does not necessarily occur in the stomach. It may happen in the intestine, even in the presence of a 1 per cent. alkaline (sodium carbonate) solution. When pure salol is administered in cachet form the best possible condition for the formation of a calculus is present. The crystals, when softened or melted, readily cohere, and may subsequently recrystallise. Even in a well-made emulsion, when heated to 40° to 42° C., the salol tends to aggregate, though I have not seen perfect conglomeration. Probably the reasons that salol calculi have not been described oftener are (1) they have not been recognised; and (2) that some gastro-intestinal disease is necessary as a predisposing cause. In Professor Bradbury's case

the cause of the colic would not have been diagnosed but for vomiting of the calculus. Retention in some part of the alimentary tract is perhaps necessary, or is at least a favourable condition; in both the cases described constipation was a prominent symptom. As the mass of the largest calculus in Brossard's case was much greater than any individual dose given, some accumulation must have occurred, and probably the same thing happened in Dr. Bradbury's case. Unfortunately, the mass was not weighed at once, and it was afterwards lost in my absence during the cleaning of the laboratory. The necessity of retention at some point is thus explained, as it is improbable that very small calculi (which must ordinarily be found, if found at all) would produce symptoms of a serious nature.

The importance of the formation of salol calculi, apart from the serious symptoms to which they may give rise, lies in the fact that the activity of the drug is markedly diminished. Even in the intestine a mass of salol can only be acted on slowly by the intestinal juices and microbes, and probably a great part passes out unchanged. This slow action all along the intestinal tract may be an advantage in some conditions, but it is certainly not in all. The chief fault seems to be in the method of administration. Salol—and the same thing holds good for other insoluble bodies of similar melting point—ought to be rubbed up with some innocuous powder, or given in the form of an emulsion, as recommended by Sahli. In this way the activity of the drug is not diminished, and the possibility of calculous formation is reduced to a minimum.

I express my indebtedness to Professor Bradbury for his kindness in giving me the notes of the case.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, April 24th, p. 1062. ² *Bull. Gén. de Thérap.*, April 30th, p. 363. ³ *Les Nouveaux Remèdes*, 1893, p. 270.

A NOTE ON EXPLORATORY INCISION AND DRAINAGE VERSUS PARACENTESIS,

IN THE TREATMENT OF ASCITES; AND EXACT DIAGNOSIS BY PERITONEAL SPECULUM.

By JOHN WALLACE, M.D. EDIN.,

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IN the address, which as President I delivered to the North of England Obstetrical and Gynecological Society in 1890, I demonstrated the etiology and diagnosis of localised peritonitis—localised ascites in many instances—and its treatment by abdominal section; and further clearly pointed out that in every case there was a well-defined cause, only to be absolutely ascertained by exploration.

In connection with this group of diseases, the word "idiopathic" does not apply. Large experience as gynecological surgeon to the Royal Infirmary very soon drove me to the conclusion that, even so far as general ascites was concerned, the same word "idiopathic"—which is only a term to cover our ignorance—had no longer any *locus standi*. For, by abdominal section the causes of ascites began to be discovered and differentiated, the condition of the peritoneum seen, and the organ at fault brought under the direct sense of touch, and if necessary of vision, thus indicating the treatment with exactitude. Paracentesis abdominis enabled one to detect the existence of a tumour or of some enlarged viscus, as liver or kidney; but the true conditions, even in the hands of the most able and experienced, were at best matters of surmise, frequently contradicted by subsequent *post-mortem* examination. Moreover, I never knew tapping cure ascites, although I have been told of successful cases and the relief given was only temporary at best. Surgically I believe it to be more dangerous than abdominal section, even where the incision is $1\frac{1}{2}$ to 2 inches in length. The latter is carried out carefully on surgical principles, the former is not—it is done in the dark, and no doubt in many cases where collapse has followed its performance some internal viscus had been wounded.

It is hardly necessary for me to trace the evolution of the prolonged tentative course of treatment which led me to the

conclusion that every case of ascites had an originating cause, the immediate one being generally in the peritoneal cavity. If the exciting causes were cardiac, the condition of the liver would be the link in the chain. The kidney can be felt, and its peritoneal covering can be seen, and may show, as I have also observed in the case of the liver, intensified redness and effusion of lymph; or a peritonitis—of an asthenic character—only to the extent of slight suffusion, which fades away in more distant parts of the peritoneum. The ascites, in short, began as a local peritonitis, a perihepatitis, or a perinephritis. Any one of the abdominal viscera may be the *fons et origo mali*. We need not wonder when we consider the close relation of the peritoneum to the whole course of the *prima via*, a fact of special importance in the lower part of the intestine, which is practically a sewer with all the varied conditions of septicism more or less present—the cæcum to wit, as in perityphlitis.

With reference to cardiac ascites, the intimate relationship between heart and liver, and the induction of obstruction to the portal circulation, is the only explanation possible. The normal secretion of the serous moisture of the peritoneum is increased by enormous exosmosis which is not compensated for by equally active endosmosis, plus kidney conditions, pressure, etc., which I need not enter into. Exploratory abdominal section, followed by drainage by means of a short glass drainage-tube, or rubber by preference, if inflammation is advanced, under antiseptic precautions, has these advantages. First, it gives relief from pressure just as well as tapping; secondly, the peritoneal inflammation is cured by drainage but not by tapping, the flow of ascitic fluid being often great, and continuing for a week or ten days, when it gradually ceases. Tympanites subsides, kidneys act, anasarca disappears, and intense relief is given, even should the patient subsequently die from incurable disease such as cirrhosis of the liver, cancer, etc. In hepatic enlargements due to alcoholism I have had permanent cures by laparotomy and drainage where tapping had failed. A two-inch incision through the peritoneum not only taps and drains the ascitic cavity but also the vascular system of the whole peritoneal sac, so that for days the discharge is sero-sanguineous. Be that the explanation or not, we know that opening the abdomen for lesions—pelvic chiefly—where it would be injudicious to proceed further with the operation, the mere manipulation, the flushing, the drainage, and subsequent sero-sanguineous discharge mostly result in cure even when least expected. Such, at any rate, was my experience in cases where I expected no benefit at all.

For bringing under direct vision those parts of the peritoneal cavity at a distance from the incision, I had made for me elongated Ferguson's tubular specula ($7\frac{1}{2}$ inches) of varying diameter. A little manipulation with these easily permits almost any part of the peritoneal cavity to be inspected through a 2-inch median incision.

Case showing use of abdominal speculum, reported by A. J. Wallace, M.D., Clinical Assistant.—A married woman, aged 40, was admitted to the Thornton Ward of the Liverpool Royal Infirmary on August 12th, 1896, complaining of swelling of the abdomen, and occasional swelling of eyelids, of seven months' duration. For about six weeks previous to admission there had been severe abdominal pain. She had borne nine children between 1880 and 1895. Menstruation was normal. The circulatory, pulmonary, and renal systems were normal.

The abdomen was more or less evenly distended, the skin being tense and shining, and having well-marked veins coursing under it. There were some irregularities in the flanks. On palpation and percussion signs of the presence of ascitic fluid were obtained. The pelvic viscera were normal, except that the uterus was bent slightly towards the left, and its mobility less than usual.

It was determined to make an exploratory incision, and this was carried out on August 25th, the peritoneal cavity being opened in the middle line midway between pubes and umbilicus for a distance of two inches. Nineteen pints of ascitic fluid escaped, and then, on exploration, the pelvic contents were found to be quite normal. The liver was considerably enlarged—about two inches in the nipple line—and on its inferior surface two rough areas were felt, from one of which a piece of fibrin was removed by the finger. On viewing these through the tubular speculum, they were seen to be the sites of lymph exudation, whilst the hepatic peritoneum surrounding them was thickened, smooth, and pale greyish-white in appearance. A similar condition existed on the superior surface of the liver, the latter in the intervals between the pale portions appearing congested, and deep reddish-brown in colour. The general peritoneum was much thickened, and its blood-vessels increased in number and congested; but there did not appear to be any more lymph exudation areas.

The cavity was well flushed with warm tap-water, a glass drainage tube placed in the lower angle of the wound, and the latter closed with stout silk sutures.