urine contained no sugar or albumin, and only the merest trace of acetone and diacetic acid. The smell in the breath grew stronger, and at the same time both dyspnoea and dropsy increased; the pulse became irregular, and a polygraph tracing showed this to be "total," associated with disappearance of the auriculo-systolic wave from the jugular curve. Mental obscurata became pronounced, and the urine, which did not total more than 16 oz. in the twenty-four hours, had to be withdrawn by catheter. Some excitement began to manifest itself, and this subsequently developed into an active delirium, the patient getting out of bed and demanding his clothes, and so on; the pupils were small. There was never any vomiting, but from the fourth day after admission an incessant foul diarrhoea began to develop, reaching its maximum on November 22nd, when no less than twenty-three actions were recorded. This may have been started by the alkaline treatment, which was begun on the day before the diarrhoea first appeared; but when alkalis were stopped the diarrhoea continued, and was checked partially and for a time only by the administration of bismuth, opium, and catechu. Treatment with full doses of alkali (3ss each of the citrate and bicarbonate of soda every four hours) had no definite effect, and digitalis (mxv of the tincture every four hours) did not alter the pulse, except to produce an irregular bigeminy of the usual digitalis type. He complained of great thirst and took plenty of milk. The temperature curve fell steadily from normal on November 19th to 96° F. on the 26th, rising again to normal abruptly on the day of death (the 27th). On November 26th the urine contained no acetone or diacetic acid, but lactic acid was present—at least, the Uffelmann test was positive. The ammonia coefficient, kindly estimated for me by Dr. W. J. H. Pinniger, was 8.4 per cent., within the normal limit.

Post-mortem examination discovered a heart weighing 18 oz.; the muscle showed some subendocardial striation but was not flabby, and the only valvular change was a slight pearly thickening of the margins of the mitral cusps. The kidneys were congested but the capsules stripped. The liver was "nutmeg" and the pancreas firm.

Microscopic examination disclosed the following changes:—Kidney: Most of the convoluted tubules were lined by ragged cells with indistinct nuclei and cloudy cytoplasm. Apart from their share in the general hyperaemia, which was intense, the glomeruli were normal. The connective tissue of the kidney was not increased. The arteries showed hypertrophy. Liver: There were marked "nutmeg" changes but nothing else, no cirrhosis or diffuse necrosis. Pancreas: The main artery showed great hypertrophy of the media. In one lobule there was extensive fibrosis, the rest being normal apart from passive hyperaemia. The heart will be examined later, as the patient exhibited auricular fibrillation.

The first case, then, was one of cardio-sclerosis, if the clinical data may be relied upon. The symptoms which suggest the existence of an acidosis are the powerful smell of acetone in the breath—so strong that it was remarked upon by several persons independently; the incessant thirst; the type of dyspnoea, which in its sighing, rhythmic character and its disproportionate intensity recalled the air-hunger of diabetic acidosis; and the nervous symptoms, of which delirium and pupillary contraction were the chief.

In the second case a similar train of symptoms marked the last phase. There was the same strong smell of acetone, the same kind of breathlessness, and a similar set of nervous phenomena. To this may be added the extraordinarily severe diarrhoea; that this was toxic is suggested by its intensity, its intractability, and its independence of colic or other abdominal symptoms. It may have been the outcome of an effort on the part of the body to rid itself of poisonous substances.

To these toxic phenomena the term "acidosis" has been applied, because of their resemblance in essentials to diabetic and other acid intoxications. The sweetish breath, the air-hunger, and the cerebral aberrations all seem to set down the phenomena seen in this pair of cases among the acidoses. Two important divergences remain to be noted—the almost complete absence of acetone and the β -hydroxy acids from the urine, and the failure of alkalization as a therapeutic measure.

None of the commonly recognized causes of intoxication were operative in these two cases; in neither man was there any trace of glycosuria or any hint at a pre-existing diabetes. Both patients were feeding adequately, neither was there any vomiting nor other cause of starvation, apart from the diarrhoea which supervened in the second case after the symptoms of acidosis had been observed. No drug had been administered in either case, such as might have given rise to acid intoxication. It seems, therefore, that these two cases represent an acidosis differing in origin and nature from those types which have been hitherto recognized.

Any comments on its possible origin and nature must of

necessity be speculative to a large extent; but there are certain available facts which afford hints as to a possible solution of the problem. Lewis, working with Ryffel, Wolff, Cotton, and Barcroft, has recently published an account of extremely thorough researches into the cause of the paroxysmal dyspnoea which occurs in cardio-renal patients without adequate mechanical cause; their first paper¹ being written mainly from the laboratory, and the second² from the bedside, point of view. Eight of their patients were suffering from Cheyne-Stokes breathing, and were found by special methods to exhibit an acidosis of undetermined nature; three others exhibited chemical evidences of acidosis, but without the characteristic change of respiration. In a majority of these patients cerebral disturbances were noted, several complained of thirst, and in one case a sour odour was noted in the room. It seems possible, therefore, that the acidosis which these writers discovered to be the cause of paroxysmal dyspnoea in their cases of cardio-renal disease made itself manifest in other ways than by producing dyspnoea only; and that the train of similar symptoms described in relation to my two cases may represent, indeed, just what it appeared to represent when observed at the bedside—namely, an acid intoxication.

The nature of the acid is so far undetermined. In my two patients it is pretty certain that the β -hydroxy acids were not responsible; for, though acetone was perceptible in the breath it was scarcely so in the urine, and in neither patient were the kidneys in such a bad way that one could assume an acid retention so severe as to dam back β -hydroxy acids into the blood in lethal quantity while admitting almost none to the urine. Lewis and his collaborators did not find any evidence of excess of β -hydroxy acids. As for the α -hydroxy acids, taking lactic acid as a type, they conclude that this was not directly and solely responsible, since they were unable to find an adequate excess of this acid in the blood of their patients. They are inclined to blame the kidneys for the acidosis which they describe; though they say, on the other hand, that, apart from the extreme cases, the results of urea estimation did not indicate failure of excretory power. It seems possible that the hepatic lesions of chronic cardiac disease might constitute a factor in the causation of this intoxication.

Summary.

Two cases of chronic myocardial disease are described in which the terminal phase was marked by clinical evidences of acid intoxication (acetone smell in the breath, "air-hunger" dyspnoea, thirst, and cerebral symptoms).

This acidosis was apparently not of the type associated with diabetes and other well-known disorders, in that the β -hydroxy acids were not present in excess in the urine.

Neither alkalis nor oxygen inhalations had any effect on the course of this intoxication.

REFERENCES.

¹ Heart, 1913, vol. v, pt. 1, p. 45. ² BRITISH MEDICAL JOURNAL, 1913, vol. ii, p. 1417.

A SUPPURATING HYDATID OF THE CRURA OF THE DIAPHRAGM.

By JAMES OLIVER, M.D., F.R.S. EDIN.

A. C., a single woman aged 23, sent to me by Drs. Townley and Kimbell, of Brynrhös, Crickhowell, was admitted into the Hospital for Women on September 23rd last. Her sole complaint was that the abdomen had gradually been enlarging since about Christmas, 1912. She had experienced little or no discomfort, and she would not have consulted her doctors when she did had it not been for her mistress, who advised her to do so.

Physical Signs.—As the patient lay on her back the anterior abdominal wall was pushed markedly forward by a swelling which extended from the pubes to 2 inches above the umbilicus. The lower abdomen was slightly more prominent on the right than the left side. The tumour could be pushed slightly up and away from the pubes, and pressing the tumour then in the left iliac region caused pain; but in its natural position the tumour was not

painful to the touch. An indistinct feeling of fluctuation could be elicited. *Per vaginam*, the cervix uteri was near the right pelvic wall. The body of the uterus was felt in front on the right side; it was not enlarged. Neither the right nor the left ovary was felt, and no portion of the abdominal tumour could be felt in the pelvis.

Diagnosis.—From the clinical history and physical signs I came to the conclusion that the tumour was ovarian and was probably a dermoid.

Fig. 1.—A, Lesser sac; B, stomach; C, colon; D, duodenum; E, lappet of omentum.

On passing my hand into the pelvis I discovered that the uterus and ovaries were normal, and that the tumour had not originated in the pelvis. The lower part of the tumour was coursed from side to side by a free lappet of omentum about an inch and a half in width and this lappet partially covered the transverse colon. On raising the lappet I found the transverse colon was so incorporated with the tumour that the muscular coat of the colon was spread out digitately on the tumour. I incised the thinned omental covering of the tumour from below upwards—in a vertical central direction—and freed the tumour as far as possible by enucleation from its omental capsule. I then found that the stomach was so intimately incorporated with the hydatid cyst that the muscular fibres of that viscus were also spread out digitately on the tumour. The transverse colon with the lappet of omentum was stripped from the hydatid and thereafter the stomach was similarly dealt with. The cyst was then found to have a neck about $2\frac{1}{2}$ in. wide and 3 in. long springing from the region of the crura of the diaphragm. The neck had to be dissected out and the fibrous and muscular tissue incorporated with it was ligatured piecemeal and cut, and thus the neck was freed. It was the anterior wall of the stomach which was incorporated with the cyst, and when the two were separated the anterior surface of the stomach was almost completely deprived of its peritoneal covering and the muscular coat was ragged and bulged here and there somewhat irregularly. Fortunately I was able to reclothe the anterior surface of the stomach with a peritoneal flap of attenuated omentum which had been stripped from the right half of the tumour. In like manner I was able to reclothe partially the bared transverse colon by a flap of peritoneum from the left half of the

Operation.

With the idea of removing the tumour intact because of its probable dermoid nature I made a long central incision, but this eventually had to be extended almost to the ensiform cartilage. On opening the abdominal cavity the tumour was found to be concealed by stomach omentum and transverse colon.

Fig. 2.—A, Seat of attachment of stomach; B, seat of attachment of transverse colon; E, lappet of omentum.

tumour, and in this process the colon was drawn close up to the greater curvature of the stomach.

The tumour contained purulent fluid and daughter cysts, and the parent sac was so loose that it escaped with the fluid when the tumour was cut into. There was nothing about the clinical history to indicate when suppuration of the hydatid took place, and the patient's temperature, both when she was admitted into the hospital and when she was seen by Drs. Townley and Kimbell, was normal.

Sections of the cyst wall showed it to be composed of fibrous tissue arranged chiefly in regular layers with muscular tissue in places. The inner layer of the cyst was constituted of small round cells.

The patient suffered greatly from shock, but convalesced without an untoward symptom.

DYSpareunia AND ITS SURGICAL TREATMENT.

By JAMES PHILLIPS, F.R.C.S.E.,

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I had been in practice a number of years before it occurred to me that dyspareunia was other than a quite infrequent and unimportant symptom. Two facts help to explain how this opinion is likely to be held by a junior practitioner. The first is the insignificant space given to the subject in gynaecological textbooks. Less than one page out of the more than 1,700 in Kelly and Noble's *System* is devoted to it, and in some quite considerable textbooks "Dyspareunia" is not even mentioned in the index. The second fact is, that pain on coitus is not a symptom which the average woman finds it easy to confess to, as is evidenced by the fact that in quite a number of the cases which I propose to describe presently the symptoms first complained of have been other than dyspareunia even when that was practically the only condition which was making life miserable.

Experience has forced upon me the knowledge (1) that quite a number of women suffer more or less severe pain during coitus; (2) that this condition underlies many a case of otherwise unaccountable neurosis; and (3) that it nearly always depends on some pathological lesion which, if properly diagnosed and treated, can generally be cured.

The number of lesions capable of producing dyspareunia is legion; my intention is to describe a few typical cases, each of which illustrates some practical point either in diagnosis or in treatment.

(a) The cause may be at the vaginal entrance.

CASE I.—Unruptured Hymen.

A woman aged 28 years, who had been married three years, was brought to me by her mother. She was languid, slept badly, had a capricious appetite, was morose and irritable. These symptoms began to be noticed some months after her marriage. I carefully overhauled her, but found no evidence of organic disease. She had some slight menstrual irregularity, and on vaginal examination I found that the hymen with difficulty permitted the passage of one finger. I did what a greater experience has convinced me to be a mistake—I asked for an interview with the husband. I have found that in many cases the husband is kept in ignorance of the true facts, and even where (as in this case) the husband knows the facts, he commonly has a notion that it is impossible to candidly discuss his sexual relations. As Stevenson has it in his article on Whitman, the Philistines have been too strong to allow the sanctity of fatherhood to be set beside the sanctity of motherhood, and among men "one of the most serious and interesting parts of life" is commonly "looked upon as ridiculous or shameful." I was given to understand that there was no difficulty with coitus. Fortunately the patient herself, after a little time, took her courage in both hands, and let me know that proper coitus had never taken place. Under ether the vaginal orifice and the vagina itself were freely stretched. Three months later the patient was pregnant, and her nervous symptoms had almost entirely disappeared.

CASE II.—Hymenal Fissure.

A young woman was taken ill during her honeymoon abroad and was told that she had kidney disease. (I believe that she had a mild attack of that far from uncommon honeymoon disease, an infection of the urinary tract due to colon bacilli being rubbed into the urethra.) When she got home I was asked to see her, nominally because of this urinary trouble, but I found that her real complaint was an agonizing dyspareunia. On examination I found one or two tiny but very sensitive hymenal