CASE I.—*Pancreatic Cyst.*

A labourer, aged 47, was admitted into Addenbrooke's Hospital on December 24th, 1913, with severe haemorrhage from the stomach and bowels; he was very collapsed, and was unable to keep anything down. On examining his abdomen there was no general distension, but a swelling was present in the epigastrium of the shape and size of a dilated stomach. The swelling was tender, and waves of peristalsis could be seen passing over it from left to right; after vomiting, the swelling diminished considerably in size. The pulse was very feeble and the temperature was subnormal. The urine was acid, and contained a distinct trace of albumin, but did not reduce Fehling's solution.

The history of the case was that in the previous September, four months before admission to the hospital, he had received a severe kick in the abdomen from a horse; he vomited soon after, and was obliged to keep his bed for a week. After this he suffered from indigestion and got much thinner, but was able to do light work. Three weeks before he came to the hospital he had suffered from constant sickness, the vomit containing much blood.

From the history and clinical symptoms the supposition was that he had received a kick on a full stomach, and that subsequent to the injury ulceration of the stomach or duodenum had taken place. He was placed on saline injections in the hope that he might recover sufficiently to undergo an exploratory laparotomy. The haematemesis and melaena, however, continued; he could retain nothing in the stomach except a little whey, and he had constant diarrhoea of thin, greenish fluid. It was noted that, after vomiting, the whole of the swelling in the epigastrium did not disappear, but a tender soft mass, dull to percussion, was left.

The diagnosis was revised, and in view of the possibility of a pancreatic lesion a twenty-four hours' specimen of the urine was sent to Mr. S. W. Cole at the physiological laboratory, to be examined for diastase. It was found to give a high diastase index of 50 units; also a trace of sugar was found by Benedict's method. Loewi's reaction was also tried, and marked dilatation of the pupil was produced in a short time by two drops of 1 in 1,000 adrenalin solution. Taking into account the clinical conditions associated with these tests, the diagnosis of a pancreatic lesion was rendered highly probable. The patient, unfortunately, never rallied sufficiently for an operation to be performed, and he died soon afterwards.

The post-mortem examination showed, on opening the abdomen, a distended stomach, and immediately behind and closely adherent to it was another sac with thick walls containing fluid. There were abundant submucous haemorrhages at the cardiac end of the stomach, and a few patches of fat necrosis in the mesentery. The sac, which was a pancreatic cyst, was closely adherent to the posterior wall of the stomach, and of about the same size; it contained over a quart of thin fluid rich in ferment, which gave a diastase index of 6,600 units. On the posterior wall of the sac, at either end, were the remains of a thin layer of pancreatic tissue. The other organs were healthy.

The pancreatic duct towards the duodenum was compressed between the stomach and the distended cyst, but the greater part of it was incorporated in the cyst wall and could not be identified.

CASE II.—*Probable Pancreatic Infection.*

A woman, aged 55, was admitted into Addenbrooke's Hospital on September 20th, 1913. The history was not very reliable, but she had had jaundice on several occasions during the last two or three years; she had had no attacks of pain, but some indigestion and loss of flesh. Her teeth and gums were foul, and there was some pyorrhoea. The skin was yellowish, and there was a tendency to pigmentation. The liver dullness was as high as the fourth rib in the nipple line, but could not be distinctly felt below the costal margin and the gall bladder was not palpable. There was no ascites. The urine contained some bile, a trace of albumin, no sugar; the stools were normal in colour.

During the next two months she lost considerably in weight, the jaundice was intermittent in intensity, the stools sometimes light in colour, at other times normal, and the urine nearly free from bile; she had no attacks of pain. There was an intermittent pyrexia of moderate degree. Early in November the urine began to contain small quantities (about 1 to 2 per cent.) of glucose, no diacetic acid or acetone; for the next few weeks there was a condition of intermittent jaundice and intermittent glycosuria, the urine on a few occasions being free from either. On two occasions a twenty-four hours' specimen of the urine examined for diastase gave an index of 40 to 45. Loewi's adrenalin test was positive in less than half an hour. The stools contained much fat, and a Cammidge reaction was negative on one occasion.

The differential diagnosis lay between malignant disease of the pancreas and chronic pancreatitis due either to a gall stone or to infection of the ducts from oral sepsis, the latter being the more probable. The patient declined any operation, and was treated with urotropin and pancreatin. The amount of sugar in the urine steadily increased up to 6 and 7 per cent. in spite of a diabetic diet. However she gained 6 lb. in weight, the temperature became normal and steady, and she went home feeling considerably better.

In both these cases the urinary diastase index was distinctly high, though not so high as in some recorded

cases of pancreatic disease, but taken in conjunction with the positive Loewi's test and the clinical history, pointed strongly to the diagnosis of a pancreatic lesion. There may be more causes than one of an increase in the diastase ferment in the urine, but it is probably due to some degree of obstruction in the course of the pancreatic ducts which causes reabsorption into the blood stream. This may take place in pancreatitis, cysts, or growths of the pancreas; but each case must be judged by due consideration of all the surrounding circumstances, and with our present knowledge it would not be safe to place too much reliance on a single test.

REFERENCE.

¹ *Quarterly Journal of Medicine*, April, 1913, p. 351.

ACIDOSIS TERMINATING CHRONIC
MYOCARDIAL DISEASE.

BY CAREY COOMBS, M.D., M.R.C.P. LOND.,
ASSISTANT PHYSICIAN, GENERAL HOSPITAL, BRISTOL.

THE two cases recorded here displayed certain features in common which can scarcely be explained away as an example of mere coincidence. Both patients were elderly men with chronic myocardial degeneration, and in both the last phase was characterized by symptoms suggestive of acid intoxication.

CASE I.

A dairyman, aged 62, was admitted to the Bristol General Hospital on August 4th, 1913, to Dr. Parker's beds, when I was in temporary charge. He had been ill five weeks with dyspnoea and swelling of the legs, the onset of which is attributed to getting wet through at work. Four weeks before admission the dyspnoea became so bad that he had to give up work, and at the same time the other symptoms became troublesome. He had felt a little pain over the left hypochondrium. The dyspnoea was very severe at the time of admission, and came on in paroxysms, during which he sweated profusely. In his previous history there was nothing of importance.

On admission he was found to be cyanosed and very dyspnoeic. The heart was dilated to the left and feeble in action, and a gallop rhythm was heard at the apex. The chest was emphysematous in shape, and some expiratory rhonchi were heard. The belly contained free fluid, and the legs were very oedematous. The blood pressure was 120 mm. Hg (systolic); the pulse beat at 80-100 per minute regularly, and very feebly. The pupils were equal and contracted; the knee-jerks were not obtainable. The breathing was definitely of the Cheyne-Stokes type, most violent respiratory excursions alternating with periods of complete apnoea.

Three days after admission the characteristic ethereal smell of acetone was noted in his breath, and he complained of thirst, asking continually for hot water. The urine contained neither sugar nor albumin; but two days later it gave a faint positive reaction with the ferric chloride test for β -hydroxy acids, and a day after this a trace of acetone was found also. At times he was delirious; his tongue became dry and furred, and the sweet smell persisted. The conjunctivae became yellow, the legs became more oedematous, and the general condition passed from bad to worse, till he died on August 15th. The bowels were opened daily without medicine while he was in hospital, and the average daily output of urine was over 30 oz. The percentage of urea was not ascertained. Of the various drugs given none made any difference; large doses of alkalis by the mouth had no effect.

CASE II.

A warehouseman, aged 60, came to my out-patient department on November 13th, 1913. He was so ill that he was admitted at once to the wards. His replies to questions were vague, and he could not even remember the word "warehouseman" when asked about his occupation. His history, gathered mainly from relatives, showed that for three or four weeks he had been short of breath and oedematous. A cough had been noticed during the same period. Cyanosis had been present for a week. He had been in hospital for lead poisoning over a dozen years previously, and also for cardiac symptoms in 1907. There was no history of rheumatism or syphilis, but his friends said he had been a heavy drinker.

He was cyanosed about the nose and ears and fingers; he gasped for breath, but was not orthopnoeic, and his respiratory movements showed a rhythmicity or grouping qualifying them for the title of Cheyne-Stokes breathing, except that the apnoeic pauses were rarely complete. The pulse was 110 and regular, except for periods of alternating extra-systoles; the systolic pressure was 175 mm. Hg. The ventricular impulse was feeble and diffused, the only visible cardiac pulsation being in the epigastrium. There was occasionally a *bruit de galop*, and the first sound, which was feeble, was followed by a systolic murmur at all areas. At the bases of the lungs crepitant râles were heard. The legs and abdomen were anasarctous, and the peritoneum contained some fluid. Two days after admission a very distinct acetone smell was noticed in the breath; the

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 obscurity 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