present in one case at the age of 5 months and in another at the age of 8 months. The second cavity was the size of a tangerine orange. In neither of these cases was there any trace of disease in the abdominal cavity—not even of solitary tubercle in the spleen or liver. As is usual with such cavities in children, the tuberculous disease of the lung was obviously due to direct spread from caseous bronchial glands. Here a word may be said about primary affection of the bronchial glands. It seems to us that, as with the mesenteric glands, there is no doubt that the bronchial glands can be infected with tubercle bacilli without the presence of any lesion of the lungs or bronchi that can be discovered with the naked eye. Few pathologists perhaps deny this, yet it has been considered improbable by some. Tubercle bacilli undoubtedly at first not uncommonly set up a bronchitis. We have seen the sputum of a patient (who had been quite well a fortnight before) swarming with tubercle bacilli when but for the fact that the bronchitis from which he was suffering was associated with a rather suspicious temperature nothing more than a passing ailment would have been thought of. Not until three months later was there any discoverable physical sign of disease in the lung, and death did not take place until three years later. An almost precisely similar case occurred in an athletic fellow-student of one of us, who tested his own sputum during a microscope class, when he was suffering from what he only considered to be a passing catarrh. To his dismay he found tubercle bacilli present in abundance, and his death from phthisis took place a few years later.

One can understand how the setting up of bronchitis by the tubercle bacillus further complicates the difficulty of ascertaining from *post-mortem* evidence the primary source of infection with tubercle. The sputum containing thousands of bacilli when swallowed may readily produce more extensive lesions of the mesenteric glands than in the bronchial glands which are close to the seat of origin of the infected sputum. To return to the question of the evidence afforded by our statistics of the relative frequency of primary abdominal and thoracic affection. Curiously enough the proportion of definitely abdominal to thoracic cases appeared to be as great in the second as in the first twelve years of life. Thus there were 8 cases apparently primarily abdominal and 31 primarily thoracic; that is to say, the proportion was just over 1 to 4 in the second 12 years and under 1 to 4 in the first 12 years. It must be mentioned, however, that in the analysis of statistics for the years we have only placed 3 amongst the doubtful cases. It must be remembered also that fatal cases of disease in hospitals cannot be taken as illustrating the comparative frequency of those diseases or varieties of disease. A serious abdominal affection is far more likely to be taken into a hospital than a case of chronic pulmonary disease, especially if the latter is considered to be tuberculosis. The same remark is applicable when we consider the value of statistics for the third period of 12 years, that is, between the ages of 25 to 36, but here the proportion of primary abdominal cases is less marked.

The ratio of cases of primary abdominal to thoracic tuberculosis in this series of 12 years and during the periods of the same length before the age of 25 and after the age of 36 is shown in the following table:

0-12 years	...	1 to 4.70
13-24 years	...	1 to 3.87
25-36 years	...	1 to 9.66
37-48 years	...	1 to 9.25
49-60 years	...	No definitely primary abdominal case
Above 60 years	...	1 abdominal case and 7 thoracic—ratio, 1 to 7

The above cases do not include all cases of disease either of the abdomen or lungs. In a few cases disease of internal organs was with little doubt a sequel to long-standing joint trouble; in 3 the kidney appeared to be first affected, and in 2 the epididymis. It may be interesting to note that in 2 of the fatal cases of Addison's disease tuberculosis occurred in no other organ of the body except the suprarenal capsules; and it may be added that these cases presented, what is sometimes seen in Addison's disease, marked hypertrophy of Peyer's patches and of the lymphoid follicles of the intestines.

Many other details which attract attention when reading through reports might be mentioned. A somewhat curious point is, perhaps, the rarity with which a case of general adhesion of the peritoneal cavity, due obviously to a previous attack of tuberculous peritonitis, is met with. One of us who has had experience of nearly 2,000 necropsies only remembers having seen one such case. Localized adhesions with some evidence of a tuberculous origin are, however, not so very

uncommon. In an apparently healthy boy, aged 16, who had been run over and killed, adhesions were present over the liver, and a calcareous mesenteric gland with several fibrous glands were present. Another case of similar character was met with in a girl aged 17, dying of heart disease. There were "firm adhesions" enclosing several calcareous nodules over the liver and some over the spleen. The mesenteric glands presented nothing abnormal. A caseous nodule was present at the apex of each lung, but no active tuberculous mischief existed.

Another point of interest that has frequently struck us in the *post-mortem* room is the presence of calcareous nodules in the lower lobe of a lung, and not very uncommonly limited to such a lobe. It appears that the tubercle bacillus can more commonly than is generally thought acquire a temporary foothold, so to speak, in some part or parts of a lower lobe, but that it almost invariably ceases to be able to materially advance, and consequently basal phthisis as we know it is extremely rare. Yet we have seen on three or four occasions cavities of considerable size in the lower lobe, while tuberculous disease of less advanced nature was in the upper lobe. In connexion with the reference to isolated calcareous nodules, it may be interesting to remark that in some cases there appeared to have been, earlier in life, recovery from widespread tuberculosis of both lungs. For example, in a middle-aged man "brought in dead," having died of sudden cardiac failure, both lungs were found to be studded from apex to base with calcareous nodules the size of peas. There were from forty to fifty in each lung, and they were as abundant in the lower as in the upper lobes. Precisely similar nodules were found scattered through both lungs of a man aged 25, who died of tuberculous meningitis, but recent tuberculous pulmonary lesions were absent.

To sum up, if we take local foci of disease as evidence of primary disease in the neighbourhood of the foci, our statistics tend to show that during the first twelve years of life tuberculous infection by the air passages is four times as common as by the alimentary canal. Our statistics also seem to show that the comparative frequency of infection through the alimentary canal during the second twelve years is equally common. Deaths in hospitals above the age of 12 years can hardly, however, be taken to represent the average causation of death from varieties of tuberculosis for the whole population above that age, because cases of tuberculosis of the lungs are more chronic than at an earlier period of life, and are not usually admitted to ordinary hospitals.

In spite of the exclusion of chronic pulmonary troubles from hospitals, the proportion of abdominal to thoracic tuberculosis in the institutions from which the reports were taken was very small after the age of 24.

TWO CASES OF PRIMARY MALIGNANT DISEASE OF THE PANCREAS.

By JOSEPH STEWART, M.B., M.Ch. R.U.I.,
Leeds.

THE two following cases occurred in my practice last year within a period of four months, and as there is reason to believe that primary malignant disease of the pancreas is somewhat rare, it seems desirable to publish them, especially as one of the cases presented an unusual congenital condition.

CASE I.

Mrs. S., married, age 58, came to see me in December, 1902, suffering from intense jaundice and most pronounced muscular weakness. She had never had an illness before. Had been a cook for a couple of years in a restaurant and had indulged moderately in stimulants. She had been losing flesh gradually for six or eight months, although able to do her work.

State on Examination.—The jaundice affected skin and conjunctivae and urine, the latter being deeply coloured. There was no constipation, but instead several motions daily (as many as six), quite white in colour, formed, and considerable in quantity: not lumpy, nor dry, passed in pieces about two inches long, and extremely smooth on the surface. There was intense irritation and itching which prevented sleep. The liver was very large, the right lobe extending into right iliac fossa, the edge and notch in the margin being easily felt. The left lobe was likewise enlarged and could be distinctly felt. The gall bladder was very much enlarged and tense, so tense that it could be grasped in the hand without making any impression on it. In the epigastric region there was a large mass rising as it were from the smooth surface of the liver and continuous with it. This was painful on pressure and was the only tender spot detected on palpation.

Progress and Result.—The patient continued to lose flesh and strength,

and died on April 7th, 1903. At no time did she complain of pain, except the "pain of weakness," and this was distressing. Twenty-four hours before death the urine was of the colour of black ink and scanty.

Necropsy.—The peritoneum was full of very dark-coloured fluid, and a nodule of growth about the size of a walnut found in the great omentum. The liver was studded with whitish deposits varying on the surface from the size of a pin's head to that of a crown-piece. The medium sized deposits were umbilicated and resembled the structure of the liver in the arrangement of the vessels. The pancreas was felt to be enlarged and hard from head to tail; it was extremely friable, and on pulling it up to the surface it broke across like a piece of carrot. It was quite white in appearance. The gall bladder was full of bile. There were no stones in either bladder or ducts. No albumen or sugar was present in the urine at any time during the illness.

CASE II.

Mr. H., aged 73, had been attended by me many times for giddiness and faintness, the result of cardiac degeneration. Four weeks ago he noticed a little dark coloration of the urine, and a week later some jaundice was observed, coming on painlessly. Up to ten years ago he had very severe attacks of "spasms," which could at times be averted by an aperient; but since that time he had been quite free from such attacks.

State on Examination.—He was very deeply jaundiced, the skin of the face, cheeks, and body bronze in appearance, and conjunctivae golden yellow. On scratching the skin with the finger-nail the track of the nail assumed a light heliotrope colour. The skin was itchy, loose, and flabby, and he looked as if he had lost much weight. The urine was very high-coloured, and dark like coffee. Part of the right lobe of the liver could be felt filling the space bounded by the right costal cartilages down to the tenth, and a line extending from this cartilage across to the middle line of the body. There was no pain on pressure over this mass, but if the fingers were inserted under its free edge and the mass lifted up pain was complained of at a spot near the middle line of the body. At a point $1\frac{1}{2}$ in. above the umbilicus and 1 in. to the left of the middle line, which could be covered by a shilling, there was a very tender spot on deep pressure. This painful spot could always be detected up to the last. No gall bladder could be felt. He complained of great weakness and loss of strength, but slept well. He had no appetite, and frequently vomited, and stated that everything he took tasted and smelt like his urine, which was as offensive as if it had undergone decomposition. No bladder or prostatic trouble was present.

Progress and Result.—The motions were invariably white, never constipated nor lumpy, and quite smooth on the surface. They appeared to have the consistency of soft putty, and resembled pieces of this material squeezed through a sausage machine. The quantity was extraordinary considering the intake, and so marked was this that it attracted the patient's and the friends' attention. On May 20th, 1903, vomiting increased and hiccough was very distressing. As much as six pints of gelatinous, tenacious, dark-coloured material was vomited during the night, although not more than $1\frac{1}{2}$ pint of fluid food had been administered during the day. All food by the mouth was stopped for eight days, and during this time the vomiting and hiccough ceased. On May 28th he had a prolonged rigor lasting two hours. When I saw him at 9 p.m. his pulse was hardly perceptible, the feet and legs cold up to the knees, and he was evidently dying. On the following morning the patient was still alive. Warmth had returned to his extremities, and for the first time during the whole illness the skin was acting. The tongue was cleaning at the edges and tip: large, dry, brown crusts were peeling off and leaving a moist surface. It was the best attempt at a recovery from death I have ever seen, as the patient declared he had been dead during the night, and I have no doubt that probably he was so. Food was now taken by the mouth with no return of vomiting or hiccough. On the 30th, however, he sank, conscious to the last. At no time during the illness was sugar or albumen present in the urine.

Necropsy.—There was a thick layer of fat in the skin, with peculiarly greasy soapy feeling. No free fluid was found in the peritoneum. The liver was slightly enlarged and of normal appearance except for whitish nodules about the size of a sixpence dotted here and there on its surface, and umbilicated in the centre. A prolonged search of over half an hour was made for the gall bladder, but none was present. There were two separate ducts leading from the liver, and as far as I could follow them down to the duodenum they remained separate. No gall stones were present in the ducts. The pancreas was white in appearance and slightly enlarged and harder than normal, but neither the enlargement nor the hardness was so marked as in Case I. It tore away easily.

REMARKS.

As to the diagnosis of pancreatitis, in general there should be no difficulty—the painless jaundice, the intense weakness, great wasting, and the enormous evacuations are characteristic. I believe if I were shown the evacuations alone I could diagnose the disease. The only one I ever read of who has laid claim to that diagnostic skill besides myself was the celebrated Dr. Hornbrook, made famous by the poet Burns.

As to the differential diagnosis between simple and malignant pancreatitis, in Case I there was no difficulty, as there were present the deep painless jaundice and the enlarged gall bladder pointing to cancer of the head of the pancreas—Courvoisier's law. In Case II no gall bladder

could be felt, for a very good reason, so that the diagnosis was difficult. I accordingly sent a specimen of the urine to Mr. Cammidge, of Wakefield, who kindly sent me the following report with permission to publish it.

The specimen of urine is free from dextrose and albumen, but contains a considerable amount of bile pigments and bile acid. It also gives a well-marked pancreatic reaction, and I should from the character of the reaction expect to find a chronic inflammation of the pancreas.

I sent Mr. Cammidge also portions of the liver and pancreas. The report is as follows:

The liver shows patches of secondary malignant disease. The cells are of a glandular character, and suggest the pancreas as the probable origin. In the section of pancreas the cells are neither normal in appearance nor in arrangement, and I should say that this is part of the pancreas showing early malignant disease, it would probably be more advanced in the head of the organ.

It can be seen from the above that during life it was very difficult to come to a correct diagnosis.

There are two questions which seem to be of interest to physiologists: (1) The fact that there was no constipation, although not a drop of bile entered the intestine throughout the duration of my attendance; (2) considering the destructive changes in the pancreas, the fact that in spite of these there was no sugar present in the urine. One might fairly have expected some change in the urine in one or both of these cases.

SOME UNUSUAL CONDITIONS IN GALL-BLADDER SURGERY.*

By E. STANMORE BISHOP, F.R.C.S. ENG.,
Honorary Surgeon, Ancoats Hospital, Manchester.

It is perhaps scarcely true that, as Rutherford Morison says, gall-bladder surgery has only a history of ten to fifteen years, but there can be no doubt that it has greatly progressed during that period, and that, before asepsis was possible, it could hardly be said to exist as we know it now at all.

It may be possible at an early period to generalize our knowledge, and to formulate some rules with reference to this subject, but the time has scarcely yet arrived. At present it is of much more importance that anything unusual or peculiar should be duly noted and described, and it is with a view to this that I have brought forward to day certain cases of this kind.

The presence of biliary calculi in the gall bladder would appear to be a very common thing in persons over 40 years of age. Every *post-mortem* room is able to furnish examples; but large numbers of such cases pass through life without attention having been called to their existence. Kehr has estimated that only about 5 per cent. of the total number ever cause any symptoms. When this does happen, however, those symptoms produce usually intense distress and call imperatively for treatment.

And of all the symptoms thus evoked the most pressing are attacks of intermittent colicky pain, situated in or near the hypochondrium or in the epigastrium, radiating around the lower part of the right thorax, and often felt with equal severity in the dorsal region, near the apex of the right scapula. Such attacks are usually associated with vomiting, which often marks the acme of the attack, and appears to relieve it. It is noticeable that these attacks are often coincident with the time when the gastric contents begin to pass from the stomach into the duodenum; that is, two or three hours after a meal.

But these attacks do not always denote the presence of a calculus. They may occur in cases where, if an operation is performed, no stone may be found. Two other causes at least have been discovered which appear to be sufficient in themselves to explain the paroxysmal pain.

Normally, the gall bladder is attached to the under surface of the right lobe of the liver by some connective tissue, and by the peritoneal coat, which, covering the lower hepatic surface, is reflected over the bladder and maintains the two in contact. In most cases only the fundus is free, and occasionally, especially in shrunken, contracted gall bladders, even this portion is closely attached to the organ above it. But Krukenberg has described two cases in which the gall bladder, entirely free from the liver down to the cystic duct, has fallen downwards and away so as to kink that duct, and produce thereby the pain which accompanies an unduly distended

* Read at a meeting of the Lancashire and Cheshire Branch of the British Medical Association.