TRANSACTIONS

OF THE

PHILADELPHIA ACADEMY OF SURGERY

Stated Meeting Held October 4, 1926

The President, DR. CHARLES F. MITCHELL, in the Chair

GASTRIC TETANY

DR. W. HAROLD STORM, by invitation, reported from the service of Dr. A. P. C. Ashhurst, at the Episcopal Hospital, the case of a man, forty-one years of age, who was admitted to the hospital August 23, 1926, with the history of a recent attack of acute appendicitis recurrens.

On admission there was well-marked rigidity over the right lower quadrant, and though no mass could be felt and peristalsis was audible, and the patient did not appear ill, immediate operation was decided upon.

At operation, August 23, 1926, an adherent gangrenous appendix was enucleated from a small abscess cavity and removed. Four hours later, before complete recovery from anæsthetic, he had a very peculiar spell. There was fixity of position of extremities and his eyes had a wild stare. This condition lasted about an hour. No epileptic history. On the day following operation he complained of very severe gas pains and was given an asafœtida enema and rectal tube and was relieved. Passed gas and voided.

The following day he again complained of distention, which was only partially relieved by enema. Fairly comfortable during night. At the end of forty-eight hours, on August 26, the distention became worse during the day and was not relieved by an enema. Eserin sulphate, gr. 1/40, given hypodermically and repeated in an hour, gave some, but only temporary relief. Enema brought no relief.

On August 27, the patient presented the clinical picture of threatening acute intestinal obstruction. Peristalsis could only be heard faintly at long intervals. Rested more easily towards noon, but at 2 P.M. complained a great deal of the distention, which was extreme. There was no pain. At 6.30 P.M. patient could be heard groaning from a distance. He had his arms extended, wringing his fingers and crying out with pain. Had a very anxious expression on his face and was vomiting foul-smelling material. Enemata gave no relief, but morphine and gastric lavage seemed to afford some relief. Continuous enteroclysis and morphine ordered and nothing by mouth. Patient rested fairly well after 9 P.M. and had no more pain.

After two days of much relief, he developed, August 30, a violent diarrhœa. Towards evening he had spasmodic convulsive seizures in which he threw his arms out from his sides and shrieked with pain and said he could not breathe because of a feeling of oppression beneath the sternum. Upper abdomen ballooned up (stomach) and then went down with eructations of gas by mouth. Temperature, 102°. Convulsive seizures were quite frequent, but occurring at irregular intervals. These seizures continued during the next twenty-four hours.

The blood was tested for chlorides, calcium, and CO_2 content; and the urine for chlorides. Bl. calcium, 10.01 mgms. per 100 c.c. blood (normal is 10–12 mgms. per 100 c.c.). Bl. CO_2 combining power, 70.8 volumes

per cent. (normal is 60–83 volumes per cent.). Bl. chlorides, 200 mgms. per 100 c.c. blood (normal is 450–500 mgms. per 100 c.c.). Urinary chlorides as NaCl, 0.5 gm. for 1000 c.c. urine. An intravenous injection of normal salt solution (750 c.c.) was given at 11 A.M. At about 1 P.M., 250 c.c. of 15 per cent. solution of sodium chloride was given intravenously. During the administration of the hypertonic sodium chloride solution patient had a very severe convulsion, resulting in the displacement of the needles in the vein and spilling some of the solution into the subcutaneous tissues. This convulsion was by far the most severe of all. Eyes diverged, face looked as if it were being torn to pieces, both upper extremities went into a convulsive seizure. During none of the convulsions were the lower extremities noticeably involved. A duodenal tube was passed at 3 P.M. The man had no more convulsions after the intravenous 15 per cent. sodium chloride. The distended stomach went down after the passage of duodenal tube. Gastric analysis of one specimen from stomach (taken just after lavage) showed no free hydrochloric acid and only 25 total hydrochloric acid. During night he had severe diarrhœa but rested fairly well. Was fed cocoa, milk, orange juice, lemon juice, etc., every four hours, by duodenal tube, getting 250 c.c. each time. There were no more convulsive seizures, and on September 1, his condition was much improved, abdomen soft, stomach not distended, no vomiting, and diarrhœa checked. Convalescence was further complicated by the development of a pulmonary inflammation which is suspected to be tuberculous. He is still in the hospital.

DR. A. P. C. ASHHURST remarked that the patient whose history had been presented by Doctor Storm is one of the complicated cases of appendicitis which is mentioned in his paper on "The Mortality in Appendicitis." This man survived. He was very sick at the time and Doctor Ashhurst believes that he was made very much worse by eserin. He had a gangrenous appendix, lying in a mass of adhesions. For tympanitis three days after operation he was given a dose of eserin, causing too active peristalsis in the presence of a subsiding peritonitis and this in turn caused an intestinal kink and partial obstruction.

The first time he had used eserin was in a case of typhoid perforation with successful suture. After three to six days the patient became very much distended and a dose of eserin was given. The distention went down but the patient died, and at autopsy a second perforation was revealed. Since then he has been afraid of eserin in peritonitis. We should always remember that the patient is not sick because he is distended, but is distended because he is sick; the treatment, therefore, is not to reduce the distention, but to treat the sickness.

In the case reported by Doctor Storm, by washing the stomach and giving morphine, the patient was tided over the period of distention. Because of the obstruction the hydrochloric acid was not getting out of his stomach to be absorbed in the small intestine and this probably caused alkalosis. The patient's state was so very serious that he decided to give him hypertonic salt solution intravenously, which was done, and the patient got well.

DR. JOHN H. JOPSON recalled a case reported before the Academy about a year and a half ago from his service at the Medico-Chi Hospital, which

was one in which typical gastric tetany was observed following operation for a large inguinal hernia where the man vomited repeatedly after the operation. A Jutte tube was used to relieve him. The function of the Jutte tube is to relieve distention of the upper intestinal tract. Of the people that it keeps alive, a number are liable to develop alkalosis unless measures are taken to prevent it by hypodermoclysis of salt solution. Another case now convalescing is the third he has seen. This is a woman of thirty-nine who had incomplete intestinal obstruction. Operation revealed carcinoma of the ascending colon, for which resection of the terminal ileum, cæcum and ascending colon was performed. Acute dilatation developed on the second day and was relieved by the Jutte tube. She was all right for ten days when dilatation of the stomach again developed, due to adhesions around the pylorus. After treatment for several days with the Jutte tube, the patient became dehydrated and developed the typical symptoms of alkalosis. In view of her greatly dehydrated condition, glucose was administered intravenously in connection with hypodermoclysis. Four hundred c.c. of a 10 per cent. solution of glucose and continuous hypodermoclysis were given the first day with prompt relief of the tetany and the next day 500 c.c. of a 25 per cent. solution. Re-operation then relieved the adhesions around the pylorus. She made a good convalescence thereafter.

MORTALITY IN APPENDICITIS

DR. A. P. C. ASHHURST read a paper with the above title, for which see page 89.

DR. GEORGE P. MULLER said that Doctor Ashhurst's paper brings up the perennial discussion; and he does well to emphasize that the mortality is in the group of cases where abscess and gangrene and diffuse peritonitis are present. Two years ago Doctor Muller reported one year's work and yesterday he analyzed the past year's work. In neither one were there any deaths in acute appendicitis where drainage was not employed. Many of these cases were classed as peritonitis, *i.e.*, there was turbid fluid in the abdomen which when cultured showed microörganisms. Therefore, the problem is—as Doctor Ashhurst has said—in the drained cases.

In the past year he drained 36 cases and had 4 deaths, a mortality of 11.4 per cent. That is a high mortality. The total mortality for the whole group was 3.4 per cent. As compared with the year previous, when he showed 50 drained cases with 5 deaths (10 per cent.), there is an increase of 1.4 per cent., but actually the percentage of drained cases was less, because they now close more cases without drainage.

Guerry, of Columbia, S. C., published a paper in the ANNALS OF SURGERY for last August in which he gives his results for 25 years, showing a total mortality of 1.1 per cent.—688 cases with one death. The speaker had no deaths in two years in cases which were not drained. In the remaining cases he has a mortality of 2.1 per cent.—(664 cases) and in the group of cases—not diffuse peritonitis cases—the mortality was 1.7 per cent.

The difference in mortality as reported by various writers makes one wonder whether the 10 per cent. mortality is not too high. It leads one to question one's procedure. There is the question, how long shall one delay operations in these cases? A recent patient, fifty-five years of age, with a mottled abdomen, was placed on enteroclysis, Fowler position, etc., and seemed greatly improved; this was at 4 P.M., and he died the next morning. Autopsy showed the peritoneal cavity swimming with turbid fluid. He might have been better handled by decompression. The difficulty is to know what to do with persons who are terribly ill. He had had a number of operations on the third day, during which time the patients have had nothing by mouth. There is always the danger of throwing these patients into alkalosis. He used to think that the thing to do was to individualize the patient, and that it was better not to have a time schedule, as the delay in some patients might be six hours and in others two days. So far he had had nine deaths; five one year and four the next. Why does one have a mortality of 10 per cent., when Guerry only has 1½ per cent. to 2 per cent.? One does not have enough cases to solve this question in one year or even two.

BENIGN BONY ENLARGEMENT OF THE CONDYLOID PROCESS OF THE MANDIBLE

DR. ROBERT H. IVY read a paper with the above title, for which see page 27.

DR. GEORGE P. MULLER called attention to an error in technic which he made the year before last in connection with this incision. He was removing the loose cartilage from the joint; it was easy to expose and to remove, but at the time he encountered some hemorrhage in the lower angle of the wound in trying to reach the vein, and urged the assistant to retract harder, so that he could examine the bleeding and stop it. The day following the operation the patient showed all the signs of facial paralysis. This was distressing, as the patient happened to be an actress. In three months' time, however, this disappeared with the exception of an inability to wrinkle the forehead, which is still the case. She was able to close her eyes, etc., and by wearing her hair over the forehead she is able to conceal the fact that she is unable to wrinkle it. The dragging down to look at the vein was sufficient to give enough injury to cause the palsy.

CARCINOMA OF THE THYRO-GLOSSAL DUCT

DOCTORS HUBLEY R. OWEN and HELEN INGELBY reported the following case history: A woman, age forty-five years, was admitted to the Woman's College Hospital, Philadelphia, on September 21, 1925. Her chief complaint was swelling of her neck of three years' duration. No history of cancer in the family. Has been married for twenty-seven years. One child (deceased). She first noticed a swelling of her neck three years ago—very small in size, about the size of an almond. The tumor apparently began in the midline and extended to the left. This tumor grew slowly until one year ago when the growth became more rapid. Has never had any difficulty breathing or swallowing. She has noticed no hoarseness, undue perspiration,

palpitation or dyspnœa. Claims to have lost about ten pounds in weight in the past year and has felt decidedly weaker. No history of insomnia. Appetite is good. Menstrual history normal.

From the midline of the neck in the region of the thyroid cartilage, extending to the left of the midline, there is a tumor the size of a hen's egg. It is not attached to the skin. There is no inflammation of the skin. The general consistency of this tumor is hard. There is, however, one soft area about the size of a walnut which apparently fluctuates. The tumor is not tender. It apparently moves with deglutition.

September 23, 1925, Doctor Owen, through a collar incision about three inches long, exposed the mass, which appeared to be cystic. Rupture occurred while attempting enucleation; cystic and colloidal matter escaped into the wound. The capsule of the tumor extended up to the hyoid bone beneath the sternum and beneath the sterno-hyoid muscle. It was impossible to remove the entire capsule, but as much as possible was tied and severed. Part of the stump of the capsule was left above and part below the cystic portion of the tumor.

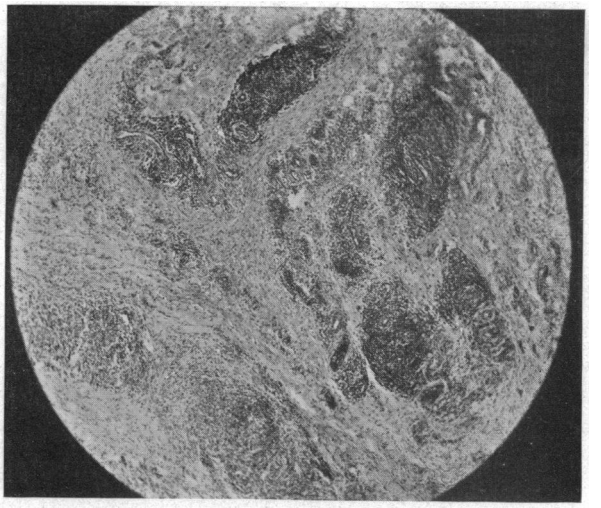

FIG. 1.—Part of the cyst wall (low power) showing aggregations of lymphocytes. Thick-walled vessels are visible. The dark spots are engorged vessels.

March 1, 1926, the patient was readmitted to the hospital with a recurrence at the site of the operation. The size of the present tumor was about two centimetres in diameter, slightly to the left of the midline of the neck. The tumor was round, hard and adherent. A second operation was performed March 2, 1926, through the scar of the former operation. Two small tumor masses were dissected out, one cystic; the second a hard fibrous mass. Both were attached to tracheal rings.

The excised portion of the cyst removed at the first operation had a wall varying in thickness from 0.5–0.3 mm. and was irregularly loculated. There were several low projections on the inner surface. Microscopically the wall consisted of parallel bands of fibrous tissue in which were embedded dense masses of lymphocytes. Some of these closely resembled lymphoid tissue, *i.e.,* they showed fairly defined reticulo-endothelial cells surrounded by lymphocytes. Others, especially the small perivascular groups, were clearly of inflammatory origin. The inflammation extended into the surrounding muscle which was infiltrated by small round cells and, in many places, invaded by fibrous tissue. The blood-vessels were thick-walled and for the most part dilated. Here and there in the cyst wall small vesicles lined by cuboidal cells were seen. Their structure recalled that of the thyroid, but they contained no colloid. Although not typical, they were interpreted as rudimentary thyroid vesicles which are often seen in the walls of branchiogenic and thyroglossal cysts. No characteristic epithelium could be found lining the cyst.

This was hardly surprising in view of the size of the cyst and the inflammation of its walls, but it is unfortunate as it prevents exact diagnosis.

The diagnosis made at the time was that of branchial or possibly thyroglossal cyst. The grounds on which this conclusion was based were: (*a*) The characteristic fibrous tissue wall; (*b*) the presence of lymphoid tissue; (*c*) the presence of rudimentary thyroid tissue. The lymphoid tissue may have been formed simply in response to the invasion by carcinoma cells and the rudimentary thyroid vesicles might be interpreted as a part of the growth. Nevertheless, it is clear that a cyst existed for some considerable time before the malignant growth. Had this been a cyst of the thyroid gland itself, it seems impossible that thyroid tissue should not have been found in its immediate neighborhood, but none was present. Its situation and character do not correspond to any form of cyst and therefore we adhere to the original diagnosis of a cyst derived from embryonic rests, probably from the pharyngeal pouch corresponding to the third branchial cleft, *i.e.*, the thymopharyngeal duct.

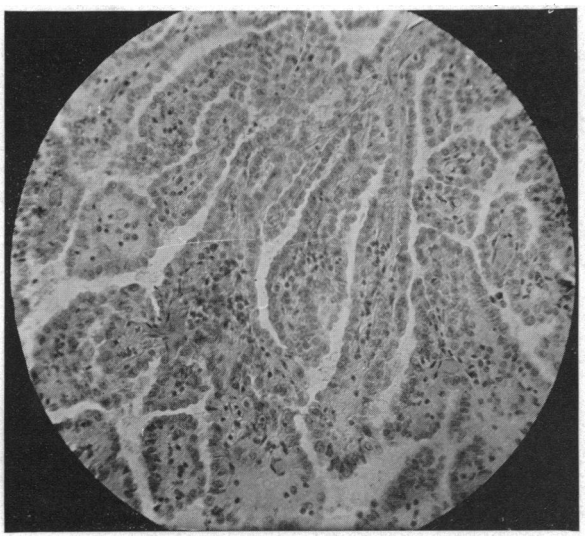

FIG. 2.—High power view of tumor showing the formation of papillæ.

The pieces of tissue removed at the second operation microscopically revealed a typical papillary adenocarcinoma. The growth consisted of cuboidal cells with pale vesicular nuclei and irregular, ill defined, often vacuolated protoplasm. The cells sometimes formed vesicles which did not contain colloid. More often they had a papillary arrangement around a central core of connective tissue. Occasionally a number of these papillæ were contained in one large vesicle (Fig. 2). In a few areas the tumor cells had become much more irregular and stained very deeply, suggesting added malignancy. A severe inflammatory reaction was present and numerous polymorphs in all stages of degeneration were found among the tumor cells. Lymphocytes were also found in abundance. Lymph-glands, as well as areas of less well-defined lymphoid tissue, and the surrounding connective tissue were invaded by the growth. No normal thyroid tissue was seen.

DOCTOR OWEN remarked that this case raises the question of the origin of papillary adenocarcinoma in the thyroid region. If one follows the teaching of the standard text-books, it is generally considered to arise in the gland itself, but in this case the thyroid appeared perfectly healthy. The tumor was found at the site of the previous operation and at the actual spot where a portion of the cyst wall had been left behind. One must therefore assume an origin from the wall itself.

It seemed just possible that they were dealing with a carcinoma of the thyroglossal duct. But in vain had he searched the literature for any record of such a phenomenon. He had come across no single description of any malignant tumor arising in the thyroglossal duct or from a thyroglossal cyst. Delafield and Prudden [4] suggest that a squamous-celled carcinoma might develop in such a situation and one other author mentions the possibility,[5] but none describe an actual growth. Nor could he find any example of a papillary adenocarcinoma arising from a branchiogenic cyst. In all the recorded cases the tumor is squamous-celled. Some other origin for our tumor had to be sought.

As long ago as 1857 Schlüter [12] described a somewhat similar growth in the side of the neck which had no connection with the thyroid and which he supposed to be derived from an accessory thyroid gland. Madelung,[10] Jores,[7] Kapsammer [8] and others describe papillary adenomas and carcinomas which they all attribute to accessory thyroids. A very able account of these tumors has been given by Billings and Paul.[2] They have collected and analyzed all the recorded cases (34 in number) of aberrant thyroids and the growths arising from them. Papillary cyst-adenoma and carcinoma have been reported fifteen times. Some of these bear a fairly close resemblance to our tumor. Three authors, Kapsammer, Barker [1] and Hinterstoisser [6] describe partly cystic tumors, the cysts having fibrous walls as in our case. They do not, however, mention the presence of lymphoid tissue in them. Kapsammer's case is particularly interesting for he gives a figure of his tumor, which seems identical with ours except that it was much larger. His tumor was adherent to the thyroid gland, but had otherwise no connection with it.

If one studies these authors a good case is made out for affirming that this type of papillary adenoma or adenocarcinoma always arises in aberrant thyroid tissue. It would seem to originate but rarely, if ever, from the thyroid itself and never from midline embryonic structures such as thyroglossal rudiments. But in this case if one supposes such an origin is to be explained away the cyst which was certainly present before the carcinoma, and in which the carcinoma began. Of course cysts may and do arise in aberrant thyroids, but in that case one would expect to find colloid-containing vesicles grouped somewhere in the wall. The vesicles in the sections were extremely sparse, were never arranged in any kind of group, and none contained colloid. It might be held that the carcinoma had produced the cyst, but in view of the history and the findings at the first operation this seems scarcely tenable. He fully admits that the histological character of the cells suggests a thyroid origin rather than an origin from embryonic rests, but the clinical course of the tumor points in the reverse direction. He therefore proposed a compromise. He would suggest that tumors of this type take their origin not from the lining epithelium of the branchial or thyroglossal cysts, but from the rudimentary thyroid tissue present in their wall. This satisfies the histological conclusions of Kapsammer and Jores, with which he was in

agreement, and at the same time accounts for the situation and mode of growth of these tumors and the microscopical findings in the cyst wall.

It would be absurd to draw any final conclusions from the study of one case. He brought this material forward in the hope that others will be tempted to follow up the question and look for preëxisting embryonic rests in patients with papillary adenoma or adenocarcinoma of the thyroid.

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POST-OPERATVE MASSIVE COLLAPSE OF THE LUNG

DOCTORS W. P. HEARN and LOUIS H. CLERF read a paper with the above title, for which see page 54.

DR. ISIDOR RAVDIN said that from the Brigham Clinic in Boston, Doctor Scott has reported the entire literature on this subject. There is a definite mortality. The cases he had had all got well without bronchoscopic treatment. He had one patient with complete massive atelectasis following operation for chronic appendicitis. He was extremely ill for five or six days. He thought he was too ill to take the chance of any bronchoscopic treatment. The patient got well and in fifteen or sixteen days was ready to leave the hospital. This condition is frequently confused with pneumonia. If the patient shows signs of pulmonary complications, look at the location of the apex beat. It is always deflected toward the affected side in collapse of the lung.