picture of a diffuse, purulent, lobular, bronchopneumonia. The general appearance of the cut section resembles closely that of an early stage of acute pneumonic phthisis, but on pressure of these yellowish surfaces with the knife, small pin-point like droplets of pus can be caused to exude. There is no old fibrous organized process and no cavitation. The mediastinal lymph nodes are swollen, gray and oedematous. There are no purulent areas nor caseation in the lymph nodes. The left lung has to be dissected off from the costal pleura. The cortex is partially torn upon delivering from the thoracic cavity. On section, it presents very much the same picture as the right, except that there is an area in the lower lobe which is more fibrous and is grayer color, shows more organization, and is much older than the rest of the process. Inspection of the abdomen reveals all the organs to be free from evidence of these lesions, except the spleen, which is diffusely spotted over with tubercle-like points varying in size from a pinhead to a pea, but on pressure exuding the pin-point like droplets of pus. There are a few very early areas in the right kidney, and the right adrenal has three such spots in its cortex. The abdominal organs are otherwise entirely negative in the gross.

Pathological Diagnosis—Pulmonary, systemic, cutaneous blastomycosis.

Case No. 1767. White, male, 32 years, married, Italian, fireman. Family and previous history negative. Dates present illness back three months. At that time, had sores on body, and also cough. Pulse range, 84-100. Temperature range, 97-100.2. Patient died after four weeks' residence.

Autopsy—Very emaciated man, apparently 34 years of age, with reddish brown hair and beard. Marked fungating lesions are seen over alae nasi, cheeks, forehead, and superior maxillary region, also on the right and left hands, both feet, and left lower leg. Those on the upper part of the body are the characteristic papillomatous lesions of blastomycosis. There are tattoo marks on both arms. There is no oedema of extremities. Small lesions on the penis are not characteristic. On opening the thorax, the diaphragm is seen to extend to the fifth rib on the right. The pericardium contains the normal amount of clear amber fluid. The cardiac apex is seen at the fifth rib, the left lung is bound completely to the costal pleura by old adhesions. The heart is normal in size, the left ventricle is empty, the mitral, the auricle, and ventricle are negative. The muscle is browner than normal, and shows no fatty changes. The right ventricle is empty. The tricuspid admits three fingers loosely. A small post-mortem and chicken-fat clot is in the right auricle and extending into the pulmonary vein. The pulmonary artery and veins are negative. The first part of the aorta shows moderate

nodular sclerosis. The semi-lunars are normal. The left and right coronaries show no sclerosis, and appear normal. The heart is negative, except for moderate hypertrophy of the left ventricle and moderate brown atrophy. On removing, the left lung does not crepitate to any great degree. The costal pleura is attached by strong fibrous bands. On palpitation, shotty nodules are felt throughout. On section, the parenchyma is filled with firm, yellow masses, rather more yellow than the usual tubercle. The center of the mass does not break down and form pus. The right lung is voluminous, and not adherent. It contains more air than the left. It is beset throughout by large, hard discrete nodules, from pea to hickory nut size. The spleen is normal in size and shows a post-mortem autolysis and digestion; it is otherwise normal. The liver is somewhat enlarged and slate colored. It is a good deal firmer than normal on section. It shows a moderate passive congestion and fibrosis, but no blastomycotic spots. The left adrenal is normal in size and shows no changes in the medulla. The left kidney is somewhat larger than usual. On section, it shows passive congestion, but no other lesion. The right kidney is in every way similar to the left. It shows passive congestion, but no other lesion.

Pathological Diagnosis—Pulmonary and cutaneous blastomycosis.

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“SURGICAL ASPECTS OF ABDOMINAL TUBERCULOSIS IN CHILDREN.”*

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ABDOMINAL tuberculosis in children like any disease in which the diagnosis is often difficult and for which different forms of treatment yield varying success, gets very easily into a confused state. Such is, I believe, the present condition of this disease. For the sake of clarification it seems wise to remind you of some of the fundamentals and to emphasize certain facts brought out by a study of cases occurring at the Children's Hospital.

In spite of repeated statements in the literature that tuberculosis of any form and particularly abdominal is rare in the first year of life, we have found it as frequent at this age as at any age up to twelve years.

* Read before the New England Pediatric Society, at Boston Medical Library, on May 13, 1921.

Though insufficient work has been done to prove what percentage of infections are due to the bovine type of tubercle bacilli, it is probable that this type is responsible for the majority. The lymphatic system of children is more prone to tuberculosis infection than that of an adult, or, to put it another way, the bovine type of bacillus may have a predelection for the lymphatics, and children, on account of their diet, are more often exposed to this type of infection.

It should be remembered that raw milk is not the only source of infection but that almost any of its products may carry it quite as well. For example, according to experiments done by the Department of Agriculture in Washington, the tubercle bacillus may remain virile in butter for a period of ninety days while cheese, ice-cream and other products of tuberculous milk are capable of disseminating the disease.

It is probable that most of the abdominal tuberculosis of children comes through the intestinal tract. The extension of tuberculosis from the female genitals which Mayo finds so common in the adult, so far as I know, does not occur in childhood. Both anatomically and physiologically, the right lower quadrant is conducive to the development of infection, for it is here that the intestines are best supplied with lymphatics and it is here that their contents move more slowly. Such being the case, it is not surprising that we find the right lower quadrant the common starting point for the tuberculous process. The tubercle bacilli find their way to the lymphatics of the mesentery either from a minute ulceration in the intestinal mucosa or by penetration of the mucosa without leaving any trace, in the same way that they penetrate the tonsil to the cervical glands without leaving demonstrable lesion behind.

The infection either becomes localized in the mesenteric glands giving what is termed, *Tabes mesenterica*, or it may extend to the peritoneal surface developing miliary tubercles and fluid. This combination is commonly known as the ascitic type of the disease. It would be preferable to refer to this as the ascitic stage in the disease rather than the type. Thirdly, one finds patients with no fluid but with coils of the intestines adherent to one another and to the parietal peritoneum. This is commonly re-

ferred to as the plastic type or stage. The pathologists tell us that this is nature's method of curing the disease. I am not quite sure that they are right in considering this a curative stage for two reasons; one, that it has been many times demonstrated at secondary operations on patients who had recovered from tuberculous peritonitis, that few adhesions exist, and secondly, that a fatal termination is the common result in patients suffering from a marked degree of plastic peritonitis, even with no other lesions in the body.

The problem of diagnosis varies to a certain extent with the stage of the disease. One rather surprising feature stands out, however, at all times; namely, that though the disease is by nature more or less chronic, the symptoms for which these children seek relief are in most cases acute and recurrent. Recurrence of moderately acute symptoms of short duration is perhaps the most uniform feature.

Where the infection is confined to the mesenteric glands of the lower ileum and caecum and these are caseous and broken down, to differentiate from an appendix abscess is frequently impossible. The symptoms are acute, vomiting has taken place, there is tenderness over the appendix region associated with involuntary muscle spasm and rigidity. The temperature is between 100 F. and 102 F. Under such circumstances time is usually not taken to ascertain the result of a Von Pirquet reaction and the white count may be only suggestive. When the glands are less broken down but still large enough to be readily palpable and with less tenderness than one would expect from an appendicitis and more mobility of the mass, a correct diagnosis may be made. But simply hyperplasia of the mesenteric glands must be remembered in the differential diagnosis. Again, where the glands involved are those of the mesentery of the ileum further removed from the ileo-caecal junction, a mass may develop in the mid-line just below the umbilicus which may be differentiated from an appendix abscess by its feel, mobility and position. When time is taken for a Von Pirquet reaction, it is a great aid to diagnosis, a negative reaction with reliable tuberculin, practically ruling out tuberculosis except in children who have an overwhelming infection. The only cases of abdominal tuberculosis which I have seen with a negative Von Pirquet reaction have been in infants having in the abdo-

men large tubercular masses and the peritoneal cavity practically obliterated by adhesions. The ascitic stage of the disease offers fewer difficulties in diagnosis. The symptoms simulate less closely other diseases. The feature usually noticed is the gradually increasing size of the abdomen. Ascites from other causes is perhaps more rare in the child than in the adult, and one should be able to demonstrate its cause. Rare conditions such as large ovarian, mesenteric or omental cysts must be considered and are sometimes difficult to differentiate. We have had five such cases referred to us with the diagnosis of tuberculous peritonitis, some of which have been correctly diagnosed as cystic only after the child was etherized before operation. Signs of abdominal fluid with small palpable masses of mesenteric glands associated with a positive Von Pirquet, and lack of the extreme muscular rigidity and symptom of a diffuse septic peritonitis make the diagnosis of tuberculous peritonitis possible.

Patients suffering from the plastic stage of the disease may have a comparatively long story of digestive disturbances, being below par and not gaining in weight. They also usually have a story of recurrent attacks of abdominal pain and come to the surgeon only when these attacks become severe or are associated with vomiting and the other signs of obstruction, or possibly when a faecal fistula develops at the umbilicus. As we see patients in this stage not only are they more acutely sick than those in the other stages, but also, in their poor development and emaciation, show more evidence of the chronic malady from which they are suffering. Among our plastic cases, five suffered from complete obstruction, two had perforation with septic peritonitis superimposed on its tubercular lesion, two had fistulae and one had mesenteric thrombosis with gangrene of a foot or more of ileum.

Perhaps it is a little arbitrary to divide abdominal tuberculosis of children into these three distinct stages. It should, however, facilitate the discussion of the problem providing one remembers that any stage may overlap one or both the other two in a given patient. Furthermore, the diagnosis, prognosis and treatment, all vary according to the extent of the process and without making this distinction, the whole problem becomes hopelessly confused.

The history of the treatment of these forms of tuberculosis is interesting. They were first practically all considered hopeless. Then there was a time when surgery was resorted to for most of the cases with some gratifying results. Later it was found that surgery was by no means a cure-all and that many patients recovered as the result of out-door life, proper hygiene, rest and a nourishing diet. The pendulum then swung back too far the other way and surgery was omitted almost entirely. Today, one will find very varying points of view. It seems to the writer however, that there is sufficient data at hand to warrant establishment of more uniformity of treatment than at present exists.

First in importance should be placed proper hygiene, diet according to digestive ability of patient and out-door life. Any treatment disregarding these principles is unsound. Surgery as an aid to treatment in selected cases is of unquestioned value, but the case should be logically selected as far as the difficulties of diagnosis make that possible. Kerley advocates operating only when the amount of fluid is sufficient to interfere with respiration or the heart or when obstruction is imminent. This teaching does not seem to me to be based on either the pathological course of the disease or practical results. As Kerley himself states, the diseased glands may at any time be the starting place for localized or general tuberculosis and probably responsible for a considerable portion of the cases of tuberculous peritonitis. Therefore, when one can make a diagnosis of localized tuberculous mesenteric adenitis, the logical procedure is to remove the infected glands. Likewise, when one finds localized caseous glands on an erroneous diagnosis of appendicitis, they should be removed. As a practical matter this has been done many times with gratifying results both immediate and lasting. There are a considerable number of such cases reported in the literature with good immediate results and eight of my own cases followed from one to eight years have remained apparently well. With this procedure there has been no immediate mortality but of course it must be understood that the number of cases for which it is suitable is limited and no one would advocate attempting to remove widely disseminated mesenteric tuberculous glands. Even in such cases however, it is logical when there

are only a few that show a tendency to break into the peritoneal cavity to prevent this by their removal and by so much reduce the chance of a peritonitis or general tuberculosis.

With regard to the large single tuberculous mesenteric abscesses, it is my practice to incise them, evacuate the pus, wipe out the cavity with iodine and close the incision in the mesentery as well as the abdomen without drainage. Three patients thus treated have been followed over four years and all appear well. Two other similar cases were treated by drainage; one died and one is improved but has some digestive disturbance and a ventral hernia.

In the ascitic stage of the disease the merits of surgical interference or the reasons for resorting to it are not so clear. Many patients, however, who are not improved before laparotomy turn the corner and start to improve soon after and go on to ultimate recovery. When laparotomy is beneficial to such cases it is probably due to the hyperaemia produced by the evacuation of the fluid and letting air into the peritoneal cavity. That this takes place has been proved experimentally. My colleague, Dr. Mixter, has advocated the injection of air or nitrogen into the peritoneal cavity to replace the fluid. Just what the effects of this procedure are has not been demonstrated experimentally and the practical results have not been striking enough yet to persuade me to substitute it for the simpler procedure. Nineteen patients with tuberculous ascites have been followed for periods of from three to twelve years. Of four who had no operative interference, three are apparently well and one dead. Eight had laparotomies with the injection of either air or nitrogen. Six of these patients are apparently well and two are dead. Of seven patients who were subjected to simple laparotomy with the evacuation of fluid, six are apparently well and the seventh has a tuberculous keratitis but otherwise appears improved.

The plastic stage of the disease presents not only a much more difficult problem for the doctor but a much poorer outlook for the patient. Generally speaking when obstruction has taken place from extensive plastic peritonitis, a fatal termination may be expected and the exceptions to this rule are not as frequent as one would wish. In my private practice are two cases of obstruction relieved by operation.

One had only a small amount of adhesions and the obstruction was due to a band of omentum adherent to a caseous mesenteric gland. The other was an infant with recurrent partial obstruction who showed considerable plasticity but some fluid. These cases though obstructive I have not included among the plastic cases.

At the Children's Hospital there have been twenty-three cases of the plastic stage. Seven of these who had no operation are all dead. On three an astomosis was performed to try and relieve obstruction. Two of these patients died before leaving the hospital and the third was temporarily relieved but died five months later. Of the remaining thirteen who had exploratory laparotomies performed, eight are dead, one is somewhat improved and four are apparently well. In this stage of the disease the fact that no cases recovered who were not operated on and a few recovered who were, does not lead me to the conclusion that the operation was the cause of the recovery or that it was beneficial to the patient in any way, except as establishing a diagnosis and causing proper hygienic treatment to be carried out. That gaining entrance to an extremely small portion of the peritoneal cavity through an incision in the abdominal wall can cure extensive plastic peritonitis is not logical. On the other hand, it is difficult to see a child die from obstruction without attempting to relieve it and surgeons will probably continue to perform many unsuccessful operations for this condition with an occasional gratifying one until it becomes possible to avoid the condition existing.

I have purposely refrained from the consideration of treatment by tuberculin, x-ray, Kromayer light and heliotherapy as it does not seem that any of these are as yet on a firm or lasting basis. It is only fair to state that the results of heliotherapy published by Rollier, Hyde and others seem hopeful. A few cases in which I have seen their technique attempted here, have not been successful.

To sum up:

1. The diagnosis of primary abdominal tuberculosis in children is often difficult.
2. The variety of its types of manifestation has led to confusion in its treatment.
3. Hygiene, diet and out-door life is the foundation of all treatment.

4. In the stage of localized mesenteric adenitis, surgical excision is logical, successful, and probably prevents the other stages developing.

5. In the ascitic stage laparotomy with evacuation of fluid is to be recommended when the patient fails to respond to medical treatment after a reasonable length of time.

6. The prognosis in the extensive adhesive stage is poor with any treatment and operation is resorted to only for the hope of relieving obstruction in which it has been occasionally successful.

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by the interesting work of Dr. Leon White,* and also, by Dr. Sluder's remarkable monograph on the subject published in 1918. It is possible, too, that some unwariness has been stimulated by Dr. White's lucid explanation and simplification of a fairly difficult region. To read Dr. Sluder's masterly work on this subject, one somehow gets the impression that visual defects from the post-ethmoidal sphenoid region are quite common, and that these regions ought to be more frequently surgically exposed, even when the conditions are such that the operation would necessarily have to be classified as exploratory in character. Yet the difficulties are such that they should not be allowed to sink into a position of obscurity, nor should any stone be left unturned in search for accurate early diagnosis.

The cases in which this condition may become dominant are in the group called hyperplastic sphenoiditis by Dr. Sluder, Onodi, and others. In this condition there may be no purulent material to be shown in the sphenoid by x-ray, or by direct inspection of the nose, yet hyperplastic changes in the sphenoidal walls may extend to involve the optic, or other neighboring nerves in certain individuals whose anatomical relations in these regions do not provide sufficient protection against such a process.

Thus we may be confronted with a condition of failing vision which may be (labeled) hyperplastic sphenoiditis, and therefore ready to have an exploratory post-ethmoidal operation and yet not objectively differentiable from several other ocular or cerebral conditions, which may give somewhat similar defects. In such a situation any method of examination which may contribute information, should be well studied and quickly available.

It is the value of perimetry in this connection that I wish especially to emphasize, since I find no stress laid upon it either by Dr. Sluder or by Dr. White. This was vividly brought to my attention some ten years ago, when I first began to study this subject. I was asked to take a field on a case in Dr. Cushing's clinic, neurological clinic, then at the Johns Hopkins Hospital. The patient had just had, at another hospital, an enteration of the post-ethmoidal region, on account of a failure of vision in the left eye.

* White: "Retrolbulbar Neuritis from Posterior Accessory Sinus Disease," *Annals of Otolaryngology and Laryngology*.

THE VALUE OF QUANTITATIVE PERIMETRY IN THE STUDY OF POST-ETHMOIDAL SPHENOIDAL SINUSITIS CAUSING VISUAL DEFECTS.

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RECENTLY much interest has been stimulated concerning the relation of post-ethmoidal sphenoidal pathology to retrolbulbar neuritis,