disease is superimposed upon the first infection because resulting immunization is an ineffective protection against tubercle bacilli in vast numbers.

Conclusions.

Latent tuberculous lesions of the lungs and tracheo-bronchial lymph nodes are revealed by radiological examination, and when they are accompanied by an active tuberculin reaction it may be assumed that they are not dormant or healed. These lesions, well known to pathologists, may have grave clinical significance. They are not infrequently the source of tuberculosis of meninges, bone, genito-urinary system, adrenals, and other organs; they may produce clinically recognizable pulmonary tuberculosis of the type of childhood, or, as an index of exposure to tuberculous infection, may be followed by the adult type of pulmonary disease.

Latent tuberculosis of the tracheo-bronchial lymph nodes of young children and latent apical tuberculosis of children during the second decade of life indicate the presence of grave infection, and may forewarn against clinically manifest disease. These latent lesions appear with great frequency in families exposed to open tuberculosis. All members of families in contact with tuberculosis should be given repeated tuberculin tests and radiological examinations even though they appear to be in good health. When these examinations reveal grave latent disease the well known measures directed to retard the progress of tuberculosis will prevent the development of manifest disease.

Tuberculous infection in childhood increases resistance to the disease, but is at the same time a source of danger. Recognition and control of latent infection are effective means of preventing tuberculosis.

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DISCUSSION.

Dr. R. G. CANTI (London) said that the question of the primary tuberculous focus in the lung in childhood had not received much attention in this country. He had reported in the *Quarterly Journal of Medicine* (1919) 84 consecutive *post-mortem* examinations on children under 10; 16 were tuberculous, and of these 10 had shown the primary lung focus. Further experience had convinced him that Ghon's theory of the relation of primary focus and glandular infection was tenable. He showed diagrams of the scheme of lymphatic drainage from the lung, and said it was possible to predict from the glands infected in a particular case the position of the primary focus in the lung. He supported this by diagrams of three cases. He believed it could be proved that the mediastinal glands were secondary to the lung focus, since where there was a lung focus there were tuberculous glands in the corresponding mediastinum, and where the mediastinal glands were tuberculous a tuberculous focus was present in the lung. Moreover, anatomical study had shown those glands to be tuberculous which would be expected to be so affected if the lung focus was primary. The changes in the glands never appeared older than those in the lung focus. He thought it was also proved that the lung focus was not secondary to one elsewhere in the body. In the majority of cases showing a lung affection no focus could be found outside the thorax. In most cases with a focus outside the thorax there was no lung lesion. If the lung infection was secondary to some other invasion—as, for example, in the abdomen—the type of tubercle bacilli would be bovine; tuberculous foci in the lung yielded the human type of bacillus. The human bacillus had no predilection for lung tissue; feeding experiments had shown that the lesions produced by human and

bovine bacilli were the same, the greatest concentration of infection being abdominal. This point had an interesting confirmation in the work of Scott in Hong-Kong. In conclusion, Dr. Canti showed, for Dr. Hadfield of Bristol, five museum specimens from five consecutive cases of children dying of tuberculosis in which Dr. Hadfield had been able to demonstrate the primary lung focus.

Dr. P. F. ARMAND-DELILLE (Paris) said that, contrary to general opinion, tuberculosis in children was found in the varied adult types. Most valuable information was to be obtained from comparing lung shadows and autopsy findings. He described his technique for the study of lungs *post mortem* in their proper relative positions, and showed how it was possible thus to find every important lesion and compare it with x-ray shadows. Investigation on these lines might reveal one of two conditions—tuberculosis apparently limited to the tracheo-bronchial glands, or associated with different forms of lung tuberculosis. In nearly every case careful examination indicated the primary lung lesions. These might be (1) diffuse miliary tuberculosis in its various forms; (2) localized lesions, usually broncho-pneumonic lobar infiltrations; (3) unilateral lesions of a whole lung; and (4) bilateral terminal lesions. His experience had convinced him that normal lungs showed x-ray hilum shadows which bore no relation to enlarged bronchial glands. Even when these glands became enormous caseous tumours their shadows might be lost in the larger shadow of the cardio-vascular apparatus. *Post-mortem* findings were gradually aiding in the interpretation of lung shadows from a prognostic point of view; it was possible to correlate progressive modification in the character of shadows with analogous anatomical evolution. Tuberculous bronchial gland tumours frequently occurred in the middle of the mediastinum, where their shadows could but rarely be observed because they were usually masked by the cardio-vascular apparatus. So-called "perihilar shadows" were not produced by the glands. The primary focus was seldom detectible in the x-ray picture, as it was often localized at the opposite pulmonary base behind the shadow of the heart, liver, stomach, or spleen. In lung lesions a study of well taken plates might reveal miliary tuberculosis before the characteristic signs and symptoms had appeared. It was difficult from x-rays alone to arrive at the accurate diagnosis of the type of pathological lesion present. A series of x-ray photographs during treatment showed progressively disappearing shadows, while tubercle bacilli were still found in the sputum. Dr. Armand-Delille illustrated his remarks with many beautiful slides of sections and comparative x-ray plates.

VOMITING AFTER OPERATIONS ON THE STOMACH.

BY

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VOMITING coming on within a day or two of an operation on the stomach, whether a gastro-jejunostomy, partial resection, or other surgical procedure, is not uncommon, and may be serious or even fatal. In some cases it is merely due to the anaesthetic, or to blood in the stomach. In that event it is likely soon to cease. Repeated vomiting, that persists for days and puts the patient's life in danger, may be due to one of the following causes:

1. *Vicious Circle*.—Years ago this was frequent and often fatal; nowadays, in good hands, it is seldom seen. It is due, of course, to a bad lie of the efferent loop of jejunum; the afferent loop may have been too long, or the efferent loop may fall away from the opening in the stomach so sharply that the duodenal contents re-enter the stomach and cannot find a way out. The vomit is copious and contains abundant bile. Prevention is easy. It is only necessary to use a short afferent loop of jejunum in the operation of gastro-jejunostomy, and to insert a stitch, after completing the anastomosis, to anchor the efferent loop to the transverse mesocolon an inch or two to the

right of the hole in the mesocolon, and this complication will not be seen.

2. *Infection of the Suture Line.*—Several French surgeons maintain that vomiting beginning a couple of days after the operation may be due to infection of the peritoneum around the suture line. It is quite possible that this may be true, but I have no recollection of seeing it.

3. *Adhesion of the Suture Line.*—I am persuaded that the commonest cause of persistent and sometimes dangerous vomiting after gastric operations is a sticking together of the anterior and posterior suture lines in the mucosa. Of course, a careless surgeon may actually sew them together, at any rate at a point or two, but apart from this gross technical error it is possible for the cut edges to adhere. If this occurs there will be copious and persistent vomiting, which differs from that of vicious circle in that no bile is brought up. The bile cannot enter the stomach, and the gastric contents cannot leave it. I first learnt of this danger after performing gastro-jejunostomy for pyloric ulcer.

The patient, a woman, vomited about twice a day for three days, bringing up all she took and much gastric juice besides, but no bile. I reopened the abdomen, and found the stomach very large and tense, but the jejunum empty. On opening the jejunum and inserting a finger it was perfectly plain that the anterior and posterior edges at the anastomosis had stuck together. It required some little force to separate them, but they were not bridged by a stitch. After various other complications she recovered.

A few weeks after a patient with cancer, on whom I had performed a partial resection by the Polya technique, began to vomit the day after operation, and brought up all she was given, and much gastric juice besides, for three days. I reopened, and found a huge tensely distended stomach and an empty jejunum. Here also exploration with a finger inside showed that the suture line had adhered tightly, but there was no stitch bridging it. She died.

These two cases brought to my mind other patients at an earlier date, who gave me great anxiety for a few days by persistent vomiting, which then stopped. It is quite likely, of course, that sufficient pressure might be developed in the stomach to force the passage; in fact, in both my cases the stomach was extraordinarily tense. If that happened the condition would be relieved.

In my opinion, then, adherence of the suture line is a not uncommon, and potentially very serious, complication of gastric surgery. Fortunately it is easily prevented. Ever since the experience related above I have inserted a piece of corrugated rubber dam, a little larger than a postage stamp, within the lumen of the anastomosis, fixing it by a fine catgut stitch to the mucosa of the stomach so as to overlap the suture line and point the way to the efferent loop of jejunum. I have had no trouble with vomiting after stomach operations since adopting this device. If it be objected that one has to take the clamps off before completing the anterior suture line, in order to insert the piece of dam, the reply is that that ought to be done in any case to make certain that there is no bleeding.

Intestinal anastomoses can be made safe and pervious by the same device.

THE ETIOLOGY AND TREATMENT OF PSORIASIS.

BY

J. H. HEANEY, M.D.

THE baffling nature of the cause of psoriasis is illustrated by the fact that one dermatologist has even called in the aid of embryology to show an analogy with the scales of lizards. He suggests that psoriasis is a "hark back" defence against cold, since it occurs almost invariably on the elbows and knees, which are the most thinly protected.

The most obvious proposition is that psoriasis is a parasitic disease, but there is no cultural or microscopic confirmation of this. The circumstantial evidence consists in (1) well authenticated cases of direct infection; (2) the marginal method of extension; (3) an observed connexion with osteoarthritis; (4) the fact that psoriasis resembles rheumatism in seasonal incidence and tendency to recurrence; and (5) the curative action of parasitocidal medicaments.

There can be no doubt that poison attacks the skin by way of the blood stream, just as it appears certain that

seborrhoea, in the beginning at least, is a more or less widely spread local infection connected with the bottle bacillus, and later associated with certain cocci. Confirmation of this is afforded by the sudden outbursts of activity in old-standing cases, by the symmetrical distribution of the lesions in acute cases, and by acute forms accompanied by asthma and headache, the last named suggesting sensitization. The evidence adduced in favour of other causes is so unconvincing that it might almost be cited in favour of the parasitic theory.

It has been suggested that psoriasis is a stage of seborrhoeic dermatitis, and the case of a patient, now aged 50, who has been under observation for some years will illustrate what is meant. The disease began as a profuse dandruff in early youth; later greasy scaly patches appeared, and still later dry patches on the scalp, followed by dry "spots" here and there on the body. Dry patches subsequently developed on the elbows and lumbar region, and still later extensive dry scaly patches on both shins. This, with two acute attacks of "spots" on the face and hands, extended over the age period 14 to 50. There is now baldness of the seborrhoeic character and very little dandruff, a psoriatic patch on the lumbar region, some involvement over the elbows and shins, and a few patches more truly seborrhoeic in type here and there on the body.

Beginning on the head and extending downwards, this is a classical case of seborrhoea, but if the history and the uppermost lesions are ignored there remains a typical psoriasis of the back, elbows, and shins, extending upwards or not at all. Some say that the two diseases may coexist. At first sight the view that psoriasis is a stage of seborrhoea appears temptingly obvious. What more likely than that a seborrhoeic dermatitis is the forerunner of psoriasis, sometimes in the same subject, as possibly in the case described, or that a seborrhoeic ancestor may have descendants with bald heads and a typical psoriasis beginning below? It is not disputed that a tendency to baldness is hereditary, and it is generally conceded that there is a predisposing disposition to psoriasis.

If it be taken for granted, then, that one of the factors in seborrhoea becomes "fixed" in the blood, and later may cause a psoriasis, which is it? The vegetable parasites to which the bottle bacillus belongs do not usually, if ever, set up blood states, but the cocci do. Moreover, a vaccine of the bottle bacilli has no effect on psoriasis. On the other hand, chrysarobin has a special lethal effect on vegetable parasites and an almost specific action on psoriasis. Used locally in various strengths it is stimulant or irritant; it may stimulate growth of hair or cause a dermatitis. It has a special action on primitive vegetable organisms, and kills the spores of ringworm. Two local actions stand out therapeutically, the stimulant and the parasitocidal. Recently a patient with chronic psoriasis came under observation. The eruption was almost universal, and the irritation and fall of scales were such that it was feared chrysarobin might precipitate a pityriasis rubra. No treatment had any effect until chrysarobin was prescribed. It is difficult to think of a stimulant effect here. There remains the parasitocidal action and the further possible effect of absorption into the blood stream.

Whitla wrote forty years ago that chrysarobin ointment rubbed into one side of the body alone cured the lesions on the opposite side; such an event could only happen through the agency of the blood. How does chrysarobin act in the blood—directly, as salvarsan does, or indirectly? If indirectly, it is possible that, since chrysarobin taken by the mouth induces a rise of temperature, it might give rise to a copula and complement of its own. The complement, not being specific, might unite with the copula of the disease—another instance of complement deviation—and in that way, or partly in that way, might effect a cure. This would presume a previous deficiency of complement in the blood. Apart from psoriasis, if it were found that chrysarobin was a ready and efficient means of producing complement, its use might become greatly extended.

On the other hand, chrysarobin may act directly on the "poison" both in the blood and in the skin lesions. It is found that the typically seborrhoeic lesions respond better to mercury and tar, but that the drier and more