After the removal of the sac, gradually the sinus from the cystic duct contracted and at the same time the kinking, or other cause of the occlusion of the common duct, was removed and the bile passed into the intestine and continues to do so, as shown by the colour of the faeces.

The age of my patient was less than usually is the case where the gall bladder is much dilated. The rate of dilatation was much greater, as usually the process is a slow one. Twenty-five pints were drawn off within four or five weeks of any swelling being observed, and not more than seven months after the fall which most probably set up the condition.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, June 17th, 1905, p. 1316.

SECONDARY PAROTITIS DUE TO ORAL STARVATION IN THE MEDICAL TREATMENT OF GASTRIC ULCER.

BY

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THE occurrence of secondary or symptomatic parotitis is well recognized as a result of abdominal diseases, especially after laparotomy for perforated gastric ulcer and in cases obviously infected, such as appendicitis, in fevers, in gastric ulcer, and in some other conditions. Stephen Paget first drew attention to the parotitis secondary to abdominal lesions in 1886, and in the following year he collected 101 cases, 50 of which were associated with some morbid condition of the generative organs. In 1904 the etiology and pathology of secondary parotitis were fully discussed by Bucknall and by Tebbs. Bucknall brought forward histological evidence in support of the conclusion that parotitis is invariably due to an ascending infection of Stenson's duct depending on oral sepsis, and that its onset may be prevented by appropriate measures. Tebbs, on the other hand, considered that in the majority of cases the path of infection is the blood stream, and not the duct from the mouth.

In the present communication we desire to direct special attention to the occurrence of secondary parotitis in the course of gastric ulcer treated medically by oral starvation so as to ensure absolute rest to the stomach. One of us (H. D. R.), after trials with other methods, such as Lenhart's and the various modifications of oral feeding, believes that the safest method as regards the ulcer, and the one most likely to be followed by permanent cure, is the somewhat Spartan one of giving nothing, not even water, by the mouth, and supplying 3 or 4 pints daily of water or (in recent years) of saline solution containing sugar, or of albumen water, by the rectum until deep tenderness over the stomach has disappeared, nutrient enemata being given in special circumstances only. The reason for depriving the patient of water by the mouth, even in sips, is of course that, since it is not absorbed by the stomach, its expulsion necessitates peristaltic action, and thus interferes with healing of the ulcer and, as has happened in some cases, may give rise to haematemesis.

The drawbacks to this method of treatment are the discomfort due to thirst and the occasional occurrence of parotitis, to which we wish to draw special attention. During eleven years' work in the wards of St. George's Hospital one of us (H. D. R.) has employed this method of treatment in the great majority of acute gastric ulcers and of chronic gastric ulcers accompanied by haemorrhage; during this period secondary parotitis has supervened in 9 cases of gastric ulcer under his care. It may be mentioned that special attention has always been paid to the mouth in the way of ordering antiseptic mouth washes in these cases, so as to avoid this complication. Such an incidence of secondary parotitis in cases of gastric ulcer treated medically by oral starvation appears to be exceptional, if we may judge from the scanty references to this subject in medical literature and from conversations with some other hospital physicians. The largest published collection of cases is that given by Tebbs—

namely, 15 cases among the cases of gastric ulcer treated medically in St. George's Hospital between the years 1890 and 1904. These cases are included in our series of 23 cases collected from the notes of cases in the hospital during the rather longer period of the twenty years 1889–1908. Paget's original paper in 1886, which summarized 60 cases of secondary parotitis, contained one case of acute gastric ulcer with suppurative parotitis, the patient surviving but never recovering her "health of mind or body." In 1898 Hone reported a fatal case of suppurative parotitis in a patient treated by starvation for gastric ulcer, and collected 8 recorded cases—Hawthorne 2 cases, Donkin 3 cases, E. A. Barton 2 cases, Nicholson 1 case. In his textbook Osler mentions that he has seen 3 cases after gastric ulcer, but as no details are given we have not counted these as due solely to oral starvation. Campbell Howard states that in fifteen years at the Johns Hopkins Hospital there were 76 cases of ulcer of the stomach with 1 fatal case of suppurative parotitis (previously recorded by Atkinson).

In the twenty years—1889 to 1908—there were 1,000 cases of gastric ulcer treated by medical measures in St. George's Hospital. As we are anxious to confine our investigation to the influence of oral starvation in the causation of secondary parotitis, we have not included any cases of gastric ulcer which were operated upon in the following statistics. Secondary parotitis has been shown to occur in a considerable proportion of cases of gastric ulcer after operation (English, Tebbs), and may then be associated with, and possibly related to, peritoneal infection. Of the 1,000 cases, 530 received food, water, or both, by the mouth from the outset of treatment, whilst the remaining 470 cases were not allowed anything by the mouth for some days after admission. Among the 1,000 cases there were 23 cases of parotitis, or 2.3 per cent., all in women; in no case was any patient affected twice with this complication. Of these 23 cases complete oral starvation was employed in 21; the two remaining cases were on rectal feeding but in addition were allowed to suck ice. In other words, the percentage of secondary parotitis in 470 cases treated by rigid oral starvation was 4.5, whilst in the 530 cases allowed something, even though only water, by the mouth it was 0.4. Haematemesis had occurred, either before admission or while in the hospital, in 16 of the 23 cases of gastric ulcer complicated by parotitis. It is thus clear that exclusive rectal feeding, or rather oral starvation, is a much more constant antecedent than haematemesis. Attention is drawn to this point because Hone attached more importance to antecedent haematemesis than to oral starvation as a factor in the causation of this form of parotitis. Probably haematemesis is likely to be a factor in the production of parotitis only in so far as it leads to treatment by oral starvation. There is no evidence that secondary parotitis depends on the occurrence in the parotid gland of thrombosis such as might be favoured by a post-haemorrhagic leucocytosis.

Of the 23 cases, the parotitis was confined to the left side in 9 cases, and in 2 cases the left side was affected first and the right side later; in 6 cases the right side only was affected, and in 1 case the right side was affected first and the left side subsequently; in 5 cases the side affected was not noted. On two occasions secondary parotitis occurred in two patients about the same time; but, apart from this, there was no evidence that it occurred in epidemic form. Of the 23 cases, suppuration occurred in 4, 2 of which were fatal; in one of these two the suppuration spread diffusely into the tissues of the neck, and was indistinguishable from Ludwig's angina. Suppurative parotitis in the course of gastric ulcer is a grave complication, for out of 8 cases of suppurative parotitis (4 from St. George's Hospital, and cases recorded by Paget, Hone, Hawthorne, and Barton) 3 proved fatal. Among the remaining 19 patients of gastric ulcer, with non-suppurative parotitis, in St. George's Hospital, 2 died.

In conclusion, we have collected 34 cases of secondary parotitis in cases of gastric ulcer, 23 from St. George's Hospital and 11 published by others (Paget 1, Hone 9, Atkinson 1). From the consideration of the exceptional number observed at St. George's Hospital, an event which we cannot explain, it appears (1) that secondary parotitis may complicate cases of gastric ulcer treated medically by oral starvation; (2) that it occurs ten and a half times—

more frequently in such cases of gastric ulcer than in cases allowed fluid by the mouth; (3) that it is an outcome of the dry condition of the mouth, and that mouth-washes do not prevent its occurrence; (4) that it is more often unilateral than bilateral; (5) that suppuration occurs in about one-fourth of the cases, and that this constitutes a grave complication.

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THE PREVENTION OF PAROTITIS DURING RECTAL FEEDING.

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THE necessary prohibition of food and water by the mouth after severe haematemesis is not infrequently followed by inflammation of the parotid glands, which usually develops on the fourth day after the haemorrhage. The side first affected is determined to a great extent by the position assumed by the patient, the gland which is most frequently in contact with the pillow being first attacked. An examination of the pus obtained from the abscess shows a variety of micro-organisms, the most constant of which—the *Staphylococcus pyogenes aureus* and *Micrococcus lanceolatus*—are also abundantly present in the thick secretions of the mouth. The impossibility of keeping the buccal cavity absolutely clean in these cases permits an ascending infection of Stenson's ducts, with consequent inflammation of the glandular tissues.

When I first adopted the use of large nutrient enemata of peptonized milk (15 to 20 fl. oz.), some ten years ago, and trusted to the usual methods of cleansing the mouth, a very large number of cases developed suppurative parotitis. An effort was then made to promote a continuous secretion of saliva, with the object of irrigating the ducts, and thus of preventing an ascending infection, and with this object the patients were directed to chew horse-radish, pellitory, or pieces of raw meat at intervals, or to keep a pebble constantly in the mouth. Eventually it was found that an india-rubber teat about 2 in. in length met all the requirements of the case, and that patients were quite content to suck it for hours at a time, with the result that the mouth remained quite clean and moist. When not in use the teat is kept in a weak solution of Condy's fluid.

Since this simple device was adopted I have treated more than 300 cases of haematemesis by rectal alimentation, lasting from ten days to seven weeks, without being troubled in a single instance by parotitis. It is only in hospital practice, where the mouth is very foul at the time of admission, that the gland occasionally becomes inflamed before any measures can be taken to prevent it.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

OVERDOSE OF EUCALYPTUS OIL.

THE taking of so large a dose as 3v or 3vj of eucalyptus oil is not of frequent occurrence. It may, therefore, be of interest to record the following facts:

About 1 a.m. on March 3rd, 1909, W. A., a man of about 55 to 60 years of age, a railway engine driver, came home from his work. He had a slight cold, and was in the habit about that time of taking six drops of eucalyptus oil on sugar, followed by what he called a tablespoonful of cod-liver oil. I was shown the spoon, and it was more like a small ladle, and I should say it would hold from

3v to 3vj. He took his six drops of eucalyptus, and put the bottle back on the shelf. He then took down in the dark what he thought was the cod-liver oil bottle, standing close to the other, but which was the same bottle containing eucalyptus oil, and poured himself out sufficient to fill the capacious tablespoon mentioned. No doubt the fumes of the six drops were still in his nose, covering the other, for it was not till he had swallowed the eucalyptus oil that by the warm feeling of it in the stomach he realized it was not cod-liver oil. He then told his wife of his mistake, but went to bed and fell asleep.

About 4.30 or 5 a.m. he wakened and felt very sick. He did not, however, vomit for some little time. When I saw him, about 8.45 a.m., he was so sick that he could not move his head or limbs, nor so much as lift up his eyelids to look at me. He had been vomiting a straw-coloured, watery, mucous fluid, which smelled strongly of eucalyptus. His skin was pale and even grey in colour. He was very cold, and could not be warmed up. His pulse was not hurried, nor small in the wave, nor high in its tension; it seemed a natural pulse of medium tension inclining to the soft side. The pupils were equal and medium in size, and reacted well to light.

By 3.30 p.m. he was very much better. He was not sick, had no headache, had a good healthy colour, and a moist clean tongue. His pulse was as before. He had slept very heavily. He had had some hiccup, breathing was never sighing.

On March 6th and 7th he still was shaken in his nerves, and did not feel "tone" enough to return to the footplate. He, however, was able to resume his usual employment in a day or two after that. He is now in his usual health.

Greenock.

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PETROLEUM IN FAVUS.

ON February 18th a woman brought to my surgery her two boys, aged respectively 6 and 10 years, both suffering from tinea favosa. The younger first began to show evidences of the disease, and it was ascertained that he had a habit while at his grandmother's house of frequently handling a cat, from which tufts of hair were falling out. His mother, beyond cropping his hair and applying some simple ointment, took very little further notice of the eruption until the elder began to show traces of the same disease, and whereas it spread but slowly in the younger boy's scalp, it spread very rapidly in that of the elder.

When I saw them, yellow scabs in size from a penny downwards to mere specks and having a mousy smell were thickly scattered all over their scalps, especially on the crown. The areas underneath the scabs were suppurating, and from these areas hair roots treated with caustic potash under an $\frac{1}{8}$ objective showed the parasite. I cut the hair shorter and began to depilate; but this proved so very painful, especially in the case of the roots on the outskirts of the suppurating areas, that I desisted. The usual watery antiseptic lotions (very likely from no fault in themselves) were unavailing to stop the spreading margins, although I persevered for a week or so, and one day I soaked the scalps in turpentine, in the hope that this spirituous antiseptic, by its greater penetrating quality would succeed where the watery antiseptics had failed. On the following day the skin was very inflamed and tender, and only olive oil was applied until these symptoms somewhat abated. I decided to try ordinary petroleum as less irritating but yet possessing equal, if not superior, penetrating properties. My object at first was mainly to limit the spreading margins, but after two or three applications I could see that the petroleum possessed curative properties also, and henceforth no other treatment was tried. The scalps were washed daily with soap and warm water, and after careful and thorough drying ordinary petroleum, purchased at 7d. a gallon for a stove, was allowed to soak for several minutes into the skin, gentle friction being used meanwhile. A suitably fitting cotton cap as the only dressing was worn under the ordinary cap. Little or no irritation from the petroleum was observable, and in less than a month from the first petroleum bath bald patches alone remained to indicate the spots where the disease had been. They were perfectly healed.

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