

PAROTITIS AS A POSTOPERATIVE COMPLICATION¹

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IN the year, 1915, four of our patients developed parotitis and three of them died. This was considered a sufficient reason for making a study of the subject, which was done. The things that were learned from that study seemed to be valuable, because we have had only two cases of parotitis since, both postoperative, and neither one died.

First, we went over our records and found that in six thousand one hundred patients who had been operated on, seven had developed parotitis, and one patient developed parotitis and died while waiting for operation. A study of these cases was made which follows.

All of these eight patients had been in the same hospital, which had no special significance, we thought, because practically all of our operative work is done at this hospital. Five were males and three were females. All of these eight patients had had abdominal operations except one and he had an abdominal infection, peritonitis following appendicitis. Three were operated on for obstruction of the bowels, two were operated on for ulcer of the stomach, one had a panhysterectomy done, and two had peritonitis. Two of the patients had faecal fistulas and one had a colostomy when the parotitis developed. In five patients both glands were inflamed and in three patients the right gland only was affected. Five patients died while three recovered. Of the five that died three had abdominal conditions that might have caused death anyway, but in two of the five patients the

parotitis was evidently the principal cause of death.

In six of the patients there was no positive evidence of suppuration in the glands. Two recovered without suppuration and four died. Of the four that died, three died in such a short time after the parotitis developed that there was hardly time for suppuration to become manifest. In two of the patients suppuration became apparent and the glands were drained.

It was deemed significant that seven of these patients had had abdominal operations and the other one had an abdominal infection, peritonitis. The seven patients operated on had received scopolamine $\frac{1}{100}$ grain and morphine, $\frac{1}{8}$ grain as a preliminary hypodermic before the operation. As is well known scopolamine dries up the secretions of the mouth and salivary glands. But one patient had not been operated on and had not received scopolamine. So evidently scopolamine was not a factor in the production of parotitis in that patient. All of the patients had been on the Ochsner treatment and nothing had been administered by the mouth for several days. An effort had been made to supply these patients with sufficient water by administering salt solution and bicarbonate of soda solution per rectum.

After a study had been made of our case records a study was made of the literature. Nearly all of the cases of post-operative parotitis reported in the literature followed abdominal operations. This seemed to be significant.

¹Read before the Western Surgical Association, Chicago, December, 1918.

One of the most interesting articles we found was by H. B. Rolleston and M. W. B. Oliver.¹ They studied 1000 cases of gastric ulcer treated in St. George's Hospital in twenty years from 1889 to 1908. These patients were all treated medically and not surgically. In 470 patients treated by oral starvation there were 21 cases of parotitis or 4.5 per cent, while in 530 patients who were allowed something by mouth there were two cases of parotitis or 0.4 per cent. These two patients were on rectal feeding but had been allowed to suck a little ice. It should be noticed that these patients were suffering from an abdominal condition, gastric ulcer, that food and drink had been interdicted by the mouth in practically all of them, and none of them had been operated on. It is evident therefore that some factor other than something pertaining to a surgical operation was a cause of the trouble in these patients. The authors sum up their conclusions as follows:

"1. 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 recurrence.

"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."

Curiously enough there is an article in the same journal by W. S. Fenwick entitled, "The Prevention of Parotitis during Rectal Feeding."² He believes that the infection ascends Stenson's ducts. He tried various measures to keep the mouth clean during the rectal feeding but they were not successful. Then he made an effort to promote a continuous flow of saliva by having the patient chew horse radish, pieces of raw meat at intervals, or keep a pebble constantly in the mouth. He finally had the patients suck an India rubber teat about two inches long which produced the

desired effect. He said the patients sucked the teat for hours at a time and thus kept the mouth clean and moist. After he adopted this method he treated more than 300 cases of hæmatemesis by rectal alimentation without being troubled in a single instance by parotitis.

An article by Jacob Frank³ discusses the different theories and sums up as follows:

"1. That it is highly probable that secondary parotitis is due to an ascending infection of Stenson's duct.

"2. That the onset of this complication may be prevented by attending to the following details: (a) mouth carefully cleansed before and after operation; (b) everything used for the anæsthetic should be sterile; (c) the anæsthetizer should avoid pressure on the gland while attempting to elevate the jaws during anæsthesia.

"3. When the swelling does not show any tendency to decrease in size, in about four days, it is advisable not to wait for fluctuation, as the location of the pus is beneath the dense parotid fascia. Therefore, free incision and drainage should be resorted to early."

From the study of our cases we are inclined to agree with Frank's conclusion, that the cause is usually an ascending infection from the mouth. All of our patients had been on the Ochsner treatment and fluids had been withheld by the mouth. Two of our patients had a fistula of the small intestine which allowed the liquid contents of the small intestine to escape before the fluid portion was absorbed. Both of these patients died. We became convinced that the parotid glands in our patients had become inactive because nothing had been taken into the mouth to excite activity of the gland, and that this inactivity predisposed the gland to infection. We also became convinced that our patients had not received enough fluid and this further contributed to inactivity of the parotid glands and dryness of the mouth. A study of the practical results of trying to give fluids per rectum convinced us that the patient did not get near as much fluid by that method of administration as we had tried to make ourselves believe that he did. When we put good

¹Brit. M. J., 1919, May 29.

²Brit. M. J., 1909, May 29.

³Surg., Gynec. & Obst., 1912, xiv, 469.

honest nurses in charge of the proctoclysis and received honest reports we found that about half of the patients retained very little, if any, of the fluid. The remaining half of the patients retained perhaps half of the amount of the fluid administered. After two or three days' administration the rectum usually becomes irritable and retains none of the fluid. At least this has been my experience, and I would like to know whether I am alone in this experience.

So we decided that our first problem was to keep the parotid gland actively secreting and passing a current of secretion down Stenson's duct into the mouth, and our second problem was to keep the patient's body supplied with sufficient fluid.

In solving the first problem we tried the use of chewing gum but it did not seem to work well. Perhaps the ingredients of the brands of chewing gum defeated our purpose. At any rate we discarded chewing gum after a trial with it. Then we tried letting the patients chew on a rubber nipple following the suggestion made by the English author quoted above. While we were doing this a colleague called attention to the fact that acids excited the parotid gland secretion, as evidenced by the diagnostic measure of having a patient suspected of having mumps try to eat a sour pickle, and suggested the use of acids. We then tried putting a lemon drop in the nipple. Finally we gave the patients old-fashioned lemon stick candy to suck and that gave the best results. Whenever it is deemed best to withhold food and drink by the mouth for a time as in stomach operations, peritonitis, or paralytic ileus, for example, the patient is given a stick of lemon candy to suck but not to eat. It excites a flow of saliva and keeps the parotid gland active. Observation has seemed to show that there is not enough secretion swallowed at one time to excite peristalsis of the stomach or bowels. At least the patients did not complain of peristaltic pains while using the candy.

The second problem of supplying the patient with sufficient fluid was solved by following the suggestion of Kanavel¹ and putting the hypodermoclysis needles in the axillæ,

and leaving them *in situ* as long as necessary in order to save the patient the pain of reinsertion. A pint of normal salt solution may be given every three or four hours in this manner, and you know that the patient's body receives the entire amount of the fluid. We have left the needles in four or five days and the patients did not complain of discomfort. Incidentally, we believe that in our practice this measure has saved lives in other conditions as well as postoperative parotitis. Sometimes we keep the solution going continuously through the needles into the tissues for several hours.

If the inflammation has not been prevented by the above means, a free incision with adequate drainage should be instituted early, in about four days, if the inflammation is not subsiding by that time. The capsule or fascia covering the gland is so dense that it takes a long time for the pus to work through and give evidence of its presence on the surface of the skin. It usually works along beneath the fascia and breaks into the external auditory canal.

Vilray P. Blair² makes a plea for the early institution of surgical treatment on the third or fourth day, and shows a cut of the incision he uses. He claims that this incision heals spontaneously with a hardly noticeable scar. This incision has been used by the writer a few times and the results so far corroborate the claims of Blair.

CONCLUSIONS

1. Postoperative parotitis is more apt to occur after abdominal operations than operations on any other part of the body.
2. Its development is favored by a dry condition of the mouth and a lack of fluids in the body.
3. The infection usually ascends through Stenson's duct.
4. In patients whose abdominal condition makes it necessary to withhold food and drink from the mouth and stomach for a time, prophylactic treatment should be instituted.
5. The mouth should be kept clean and moist by its own secretions and the body should be abundantly supplied with water.

¹Surg., Gynec. & Obst., 1916, xxiii, 483.

²Med. & Surg., 1917, March

6. A good way to excite the secretions of the mouth and to keep a current of saliva flowing *down* Stenson's duct is to allow the patient to *suck* on a stick of lemon candy after operation.

7. A very accurate and effective way to supply the body with fluid is to administer salt solution by hypodermoclysis. If this is done according to Kanavel's directions, there

will be no pain in the administration and very little discomfort.

8. If the prophylactic treatment fails and parotitis develops and the inflammation is increasing, or is no better by the third or fourth day, the gland should be uncovered by a free incision and punctured in several places with blunt forceps and the incision packed with wet sterile gauze as suggested by Blair.