Postoperative Parotitis

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As recently as 1955, Robinson [1] categorically stated that "surgical parotitis has practically vanished and prophylactic antibiotic therapy is probably the chief reason." In 1958 Brown, *et al.*,[2] stressed the "increased incidence of infections due to antibiotic resistance micrococci in hospitalized patients." They further stated that an "unemphasized aspect of this problem is the reappearance of postoperative parotitis as a complication of surgery."

Postoperative parotitis is an acute inflammation of the parotid gland. The organism responsible has been identified as an antibiotic resistant strain of hemolytic *Micrococcus pyogenes* var. *aureus* (*Staphylococcus aureus*).[3]

The pathogenesis of the disease is unknown; but there are four possible portals by which the organism may gain entry into the gland: (1) the lymphatics, (2) the blood stream, (3) the parotid duct, and (4) direct extension from contiguous tissues.[2] Gilchrist and McAndrews [4] believe that the infection is an ascending infection from the mouth by way of Stensen's duct. It has also been suggested that prolonged pressure on the gland by the anesthesiologist may be a contributing factor. In addition, White [5] associated the condition with poor oral hygiene and a dry mouth. Anesthesia has been incriminated as playing a significant role in the development of postoperative parotitis. Coughlin,[6] however, has shown that it can occur in patients who have not had general anesthesia and reports its occurrence in 12 patients who had received a spinal anesthetic.

The diagnosis is simple and easily made by finding an enlarged, tender parotid gland, usually on the fourth to sixth day following operation. The first symptom is most often localized pain and swelling of the gland. Occasionally pain may be experienced in the temporomandibular joint. An elevation of the temperature to 102° to 104° F. will occur within the first 24 hours. The leucocyte count will be acutely elevated. The mortality rate according to Gilchrist [4] varies from 30 to 60 per cent.

The following survey covers a five-year period at the Harbor General Hospital. During this time 12,166 surgical procedures were performed. The ages of the patients ranged from newborn infants to 100 years. Twenty-eight of these patients developed postoperative parotitis. Their ages ranged from 35 years to 92 years, with an average of 73 years. The anesthetic methods used in these 28 patients were local infiltration (3), regional epidural block (2), subarachnoid analgesia (9), and inhalation anesthesia (14) consisting of nitrous oxide—oxygen, ether and cyclopropane. Thirteen of the 14 patients receiving inhalation anesthesia had endotracheal anesthesia. All of the patients who developed parotitis had received either atropine or scopolamine for preoperative medication. In addition, two patients received subsequent doses of atropine during the surgical procedure.

The state of hydration prior to operation was considered poor in 17 patients, fair in eight patients and good in three patients. The duration of operation in those developing parotitis ranged from 65 minutes to 300 minutes. The average anesthesia and operating time was 185 minutes. Eighteen patients had nasogastric suction postoperatively.

The onset of parotitis varied from the first to the twenty-seventh postoperative day. The average date of onset was on the ninth postoperative day.

The gland on the right side was involved in

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TABLE 1. Surgical Procedures of Patients Who Developed Parotitis

	Number of Patients
Orthopedics	
Fractured hip	7
Fracture of femur	2
Amputation of leg	3
General Surgery	
Small bowel resection	3
Gastric resection	2
Hiatal hernia repair	2
Gastrostomy	1
Herniorrhaphy	4
Radical mastectomy	1
Other Procedures	
Spurapubic prostatectomy	1
Abdominal hysterectomy	1
Radium insertion in neck	1
Total	28

thirteen patients, left side in eight patients, and bilateral in seven patients.

The surgical procedures of the patients who developed parotitis are shown in table 1.

Treatment was varied and consisted of forcing fluid in ten patients, potassium iodide drops in one patient, incision with drainage in three patients and antibiotics. Because of the large number of antibiotics used in numerous combinations an evaluation of their relative value is not attempted. Petersdorf [7] believes that specific antibacterial therapy is still essential.

Radiation therapy in conjunction with antibiotics was used in 25 patients. This consisted primarily of giving 250 KVP radiation with ½ mm. or 1 mm. of copper or a Thoreus III filter, 50 cm. TSD. The amount of radiation to be given is debatable; but an air dose of 100 to 900 roentgens had been given.

Seventeen of the 28 patients who developed parotitis died or a mortality of 60 per cent. The immediate cause of death was as follows: pneumonia, 9 patients; myocardial infarction and cachexia, 2 patients; cerebral vascular accident and overwhelming sepsis, 2 patients; congestive heart failure, 3 patients; and wound abscess and marked sepsis, 1 patient. Although no deaths were directly attributable to parotitis, the disease was a contributing factor in seven cases. During this same period of study, 51 nonsurgical patients developed parotitis with an overall mortality of 38 per cent, or a 27 per cent mortality related to the parotitis.

Discussion

Postoperative parotitis is not a vanishing disease as heretofore believed. In this series the incidence is approximately one in every 434 operations. This is an exceptionally high percentage and may reflect the economic, nutritional and social status of the patient so frequently seen in a county institution. With the increase in the average age of patients coming to operation, we may expect the incidence of postoperative parotitis to increase further.

The causative factor is difficult to ascertain, but certain general contributing factors may be postulated from this series. The administration of atropine and/or scopolamine, especially in the aged patient, may cause inhibition of the salivary secretion with a portal of entry for microorganisms being established via Stensen's duct. In conjunction with this hypothesis, the state of hydration becomes an important factor in this survey since 25 of the 28 patients were fair to poorly hydrated prior to operation.

Pressure upon the parotid gland may be an important etiologic factor; but if this is so, the onset of parotitis should be within the fourth to sixth postoperative days. Binder [8] has demonstrated a causal relationship between trauma to the parotid gland and the development of parotitis. The average onset in this series was on the ninth postoperative day. This would lend support to the theory that the infection was an ascending one and not a pressure initiated one.

Treatment

The method of treatment is controversial. Prophylaxis is the most effective treatment and attention should be directed to oral hygiene. Systemic dehydration should be corrected before operation and minimal usage of the belladonna drugs for preoperative medication. Supportive measures are important in the management of acute postoperative parotitis. Fluid and electrolyte replacement, local heat application and mouth care are essential. Furthermore, there is no reason to doubt the

value of adequate doses of properly selected antibiotics.

The theoretical bases for radiation therapy is somewhat uncertain; but an effect through the production of local vasodilation probably best fits the available data. Radiation should be considered as an ancillary rather than the principal therapy in the management of postoperative parotitis. Surgical drainage will be necessary in a few instances, but the need for hasty incision has not been demonstrated. Surgical drainage may be indicated if clinical improvement has not occurred within three to four days.

Summary

Postoperative parotitis occurred in 28 patients in this series of 12,166 cases or one out of every 434 operations with a mortality of 60 per cent.

References

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HEMOPHILIA With the administration of antihemophilic factor (AHF) from ox and pig blood, severe hemophiliacs may be made completely normal for a significant period of time, and major surgery safely undertaken. In their present form, the materials are antigenic and, therefore, can be used for only one period of treatment in any one hemophiliac. (*Ingram, G. I. C.: Major Surgery in Hemophilia; Use of Animal Antihemophilic Factor in Britain, Transfusion 2: 88 (Mar.–Apr.) 1962.*)

HEPATIC COMA Progressive electroencephalographic changes are noted in patients with impending or actual hepatic coma. These do not correlate well with studies of blood chemical constituents and vary more with changes in the level of consciousness. There is a continuum of changes from normal to theta, to pseudoparoxysmal, to delta activity. (*Silverman, D.: Some Observations on the EEG in Hepatic Coma, Electroencephalog. Clin. Neurophysiol. 14: 53 (Feb.) 1962.*)

SERUM SODIUM Electrolyte changes may occur during prolonged operations. Serum sodium was measured for 25 patients whose operations lasted over two hours. Eight had lowered and two had elevated sodium levels, exceeding 5 mEq./liter. The pertinent factors are type and amount of infusions, transfusions, sweating, irrigation of raw surfaces, manipulation of viscera, hyperpnea, fever, and activity of anti-diuretic hormone. (*Bradham, R. R., Gregorie, H. B., Jr., and Jackson, R. E.: Effect of Long Operations on Serum Sodium Concentrations, A.M.A. Arch. Surg. 84: 487 (May) 1962.*)