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# Communications

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## Parotid Enlargement in Bulimia

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The term *bulimia* refers to both a symptom and a disorder. The symptom consists of episodic bingeing, ie, consuming large amounts of high-calorie foods in short periods of time. Often the behavior is done in secret. Bulimia occurs in about 50 percent of patients with anorexia nervosa, where it is a poor prognostic sign,<sup>1</sup> and is seen as well in persons who are grossly overweight. Bulimia also occurs, however, in persons of normal weight and is often followed by self-induced vomiting to avoid weight gain.

Occasional bingeing, whether followed by vomiting, is a common practice among women in the United States and other Western cultures. A recent survey in Great Britain reported a prevalence of occasional bingeing of 21 percent in a large random sample of young women attending a family planning clinic.<sup>2</sup> In some individuals, however, bingeing becomes increasingly frequent, sometimes occurring many times each day for weeks or months at a stretch. Typically these patients self-

induce vomiting after almost every binge to avoid weight gain and may engage in other forms of purging as well (cathartics and diuretics). Such persons become preoccupied and obsessed with their bingeing and vomiting cycles, guilty over their secret and bizarre eating habits, intensely anxious over the increasing loss of control over their own behavior, and depressed as a consequence. Much social disruption and withdrawal are evident, and they cease to function adequately at work, school, and in other important life areas. This syndrome constitutes the disorder of bulimia.<sup>3</sup>

The prevalence of bulimia is not known, but its prevalence appears to have increased greatly in the United States over the last decade, giving rise to special clinics for its treatment in many large cities as well as to a number of self-help groups around the country. In the Eating Disorder Service at the University of Pennsylvania currently 15 to 20 new cases are seen each month. The condition occurs almost entirely in women, usually between the ages of 15 and 35 years.

In addition to the multiple emotional and adjustment problems these patients evidence, the following medical complications can occur: acute dilatation of the stomach from ingesting large amounts of food over brief periods of time, steatorrhea and related problems from the exces-

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sive use of cathartics, irritation of the throat with hoarseness from the repeated effects of acidic vomitus, erosion of the dental enamel from the vomitus, and multiple metabolic disturbances. The most serious of the latter is hypokalemia, again resulting from frequent vomiting. A final complication, the subject of this report, is enlargement of the salivary glands, especially the parotids. The enlargement may be unilateral or bilateral, is usually painless, and is often so slight that the patient may not be aware of the condition until a small tender enlargement is detected by the examining physician. On occasion, however, the swelling can be so extensive as to grossly distort the shape of the face, restrict movement of the head, produce pain, and be a major source of concern and embarrassment to the patient. Because of its relative rarity and the fact that most bulimic patients are highly secretive and defensive about their bizarre eating habits, the condition can present a diagnostic puzzle, as the present case illustrates.

### Case Report

When this patient was first seen, she was a 34-year-old married housewife and mother of two young children. She recalled occasional secret bingeing and vomiting from the age of 15 years. The frequency of bingeing and vomiting increased during periods of stress. Bingeing and vomiting became a daily practice when, in her early twenties, she was attending college but living at home in a conflict-ridden environment. When she was 23 years old she first became aware of pain and fullness at the angle of her jaw bilaterally. The swelling of her parotids waxed and waned over the next four years, the enlargement sometimes reaching 10 cm in diameter on both sides. This symptom was a source of great distress, as it gave her face a distorted appearance.

Over these years it did not occur to the patient that there might be a relationship between this condition and her bingeing and vomiting, behaviors she had been doing to some extent for eight years. During these four years many examinations and diagnostic procedures were carried out in a number of medical facilities in an effort to reach a diagnosis upon which appropriate treatment could

be instituted. Suspected diagnoses corresponded to the various medical conditions known to produce parotid enlargement such as obstruction of the ducts, local infection, viral diseases, allergic and autoallergic reactions, Sjögren's syndrome, Mikulicz's disease, uveoparotid fever, and so on.<sup>4</sup> Thus she had examinations of the oral cavity and oral pharynx, dental examinations, and laryngoscopy, the results of which were always negative. Roentgenograms of the chest, paranasal sinuses, and teeth were negative. Also negative were gastric washings, pancreatic enzyme studies, several sialograms, a variety of endocrinological studies, antinuclear antibody tests, and countless blood chemistries. Needle biopsy results of the parotids were normal without evidence of acute or chronic inflammation.

Over this four-year period, a variety of treatments were tried on an empirical basis, including antihistamine medications, phenylbutazone, neostigmine injections, and trials of steroids and antibiotics. A trial was also made with autologous vaccines prepared from her own saliva. As none of these brought relief, and the patient was becoming increasingly distressed and despondent over her refractory condition, a trial of radiation therapy was recommended and undertaken. The patient was now 27 years old.

Radiation therapy consisted of 15 MeV delivered via a Betatron in the area of the left parotid in four fractions over a three-day period for a total dose of 720 rad. There was a rapid regression of the swelling from an approximately  $7 \times 7$  cm mass to a barely detectable enlargement at the angle of the jaw. Similar radiation was applied to the right parotid area, which responded in a similar fashion. Over the next ten months, however, the swelling and associated discomfort gradually returned. The patient was still secretly bingeing and vomiting. A second course of radiation was undertaken, 200 rad being delivered to each side by means of a 10-MeV electron beam. Again in a matter of days there was a favorable response. The swelling again returned over the next six months. A third and final course of radiotherapy was applied to both sides, 1,500 rad being delivered over seven fractions on this occasion. Virtually all the swelling disappeared following this treatment.

The patient continued to binge and vomit over

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**Contraindications:** Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyl dopa therapy has been associated with liver disorders (see Warnings); hypersensitivity to any component, including sulfites (see Precautions).

**Warnings: It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyl dopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions.** With prolonged methyl dopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyl dopa. If a positive Coombs test develops during methyl dopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood. At the start of methyl dopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyl dopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyl dopa, the drug should not be reinstated. When methyl dopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyl dopa is stopped.

Should the need for transfusion arise in a patient receiving methyl dopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, occasionally with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever, abnormalities in liver function tests or jaundice appear, stop therapy with methyl dopa. If caused by methyl dopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyl dopa should not be reinstated in such patients. Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

**Pregnancy and Nursing:** Use of any drug in women who are or may become pregnant or intend to nurse requires that anticipated benefits be weighed against possible risks; possibility of fetal injury or injury to a nursing infant cannot be excluded. Methyl dopa crosses the placental barrier, appears in cord blood, and appears in breast milk.

**Precautions:** Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). Sulfites have been reported to cause severe allergic reactions in certain susceptible individuals, particularly patients with asthma. Oral Suspension ALDOMET and Injection ALDOMET Ester HCl contain sodium bisulfite; Tablets ALDOMET contain no sulfites. Methyl dopa may interfere with measurement of: urinary uric acid by the phosphotungstate method, serum creatinine by the alkaline picrate method, and SGOT by colorimetric methods. A paradoxical pressor response has been reported with intravenous use. Since methyl dopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyl dopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyl dopa or its metabolites.

Stop drug if involuntary choreoathetoid movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyl dopa because the drug is removed by this procedure.

**Adverse Reactions:** *Nervous System/Psychiatric:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetoid movements; psychic disturbances, including nightmares and reversible mild psychoses or depression. *Cardiovascular:* Bradycardia, prolonged carotid sinus hypersensitivity, aggravation of angina pectoris. Paradoxical pressor response with intravenous use. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyl dopa if edema progresses or signs of heart failure appear). *Digestive:* Nausea, vomiting, distention, constipation, flatulence, diarrhea, colitis, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis. *Hepatic:* Abnormal liver function tests, jaundice, liver disorders. *Hematologic:* Positive Coombs test, hemolytic anemia. Bone marrow depression, leukopenia, granulocytopenia, thrombocytopenia. Positive tests for antinuclear antibody, LE cells, and rheumatoid factor. *Allergic:* Drug-related fever, lupus-like syndrome, myocarditis, pericarditis. *Skin:* Rash as in eczema or lichenoid eruption; toxic epidermal necrolysis. *Respiratory:* Nasal stuffiness. *Metabolic:* Rise in BUN. *Urogenital:* Breast enlargement, gynecomasia, lactation, amenorrhea, impotence, decreased libido. *Endocrine:* Hyperprolactinemia. *Musculoskeletal:* Mild arthralgia, with or without joint swelling; myalgia.

**Note:** Initial adult oral dosage should be limited to 500 mg daily in divided doses when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third months of therapy; increased dosage or adding a diuretic frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

For more detailed information, consult your MSD Representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486

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## PAROTID ENLARGEMENT IN BULIMIA

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the following six years, although with somewhat less frequency than previously. Only minimal, painless swelling of the parotids occurred. During this period she was married and bore two normal children. As the eating disorder was a continuing source of anxiety and guilt to her, she finally confided in her husband about the problem and sought psychiatric help. It was at this time that she learned of the likely relationship between her eating habits and the parotid swelling. Currently she is being treated for her psychiatric disorder.

### Comment

At the Eating Disorder Service enlargement of the parotids occurs in about 10 percent of the patients with bulimia, occurring more often in those who binge and vomit one or more times every day. About 50 percent of these show elevated serum amylase levels, a proportion in keeping with the report of Levin et al.<sup>5</sup> We have detected no amylase elevations in those without parotid swelling.

The mechanism of parotid swelling in patients with bulimia is not clear. Intense repeated stimulation of the salivary glands from repeated episodes of binge eating (work hypertrophy) would seem to be the most likely cause. Indeed, persistent parotid swelling has been observed in several women who ingested large amounts of starch on a daily basis.<sup>6</sup> However, parotid swelling also has been described in cases of repeated, surreptitious self-induced vomiting.<sup>7</sup> Russell<sup>8</sup> described a woman with bulimia whose chronic salivary gland swelling subsided when she could bring vomiting under control. Possibly the mechanism here involves the irritating effects of the vomitus on the opening and lining of the salivary ducts. Although most Eating Disorder Service patients with parotid swelling both binge and vomit, patients with swelling are seen who self-induce vomiting without bingeing as well as occasional patients who binge without vomiting. Thus both behaviors probably play a role.

Bulimia was without doubt the cause of the bilateral parotid enlargement in the patient described above. Its onset corresponded to an increment in the frequency of her bingeing and

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**Azo Gantrisin®**

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Before prescribing, please consult complete product information, a summary of which follows:

**INDICATIONS:** Initial treatment of uncomplicated urinary tract infections caused by susceptible strains of *Escherichia coli*, *Klebsiella* species, *Enterobacter* species, *Proteus mirabilis*, *Proteus vulgaris* and *Staphylococcus aureus* when relief of pain, burning or urgency is needed during first 2 days of therapy. Azo Gantrisin treatment not to exceed 2 days. Evidence lacking that sulfisoxazole plus phenazopyridine HCl better than sulfisoxazole alone after 2 days. Treatment beyond 2 days should only be continued with Gantrisin (sulfisoxazole/Roche). (See DOSAGE AND ADMINISTRATION.) **Important Note:** Coordinate *in vitro* sulfonamide sensitivity tests with bacteriologic and clinical response. With ongoing therapy, add aminobenzoic acid to culture media. Increasing resistance of organisms may limit sulfonamide usefulness. As identical doses produce wide variations, measure blood levels in patients receiving sulfonamides for serious infections: 12 to 15 mg/100 ml is optimal; adverse reactions are more frequent above 20 mg/100 ml.

**CONTRAINDICATIONS:** Children under 12; known sensitivity to either component; pregnancy at term and during nursing period; in glomerulonephritis, severe hepatitis, uremia and pyelonephritis of pregnancy with gastrointestinal disturbances.

**WARNINGS:** Sulfonamides are bacteriostatic; organisms causing common infections are often resistant. A streptococcal group A streptococci or prevent sequelae like rheumatic fever and glomerulonephritis. Deaths from hypersensitivity reactions, hepatocellular necrosis, agranulocytosis, aplastic anemia and other blood dyscrasias have been reported. Sore throat, fever, pallor, purpura or jaundice may be early signs of serious blood disorders. Perform blood counts and renal function tests.

**PRECAUTIONS: General:** Use with caution in patients with impaired renal or hepatic function, severe allergy, bronchial asthma. Hemolysis may occur in glucose-6-phosphate dehydrogenase-deficient individuals.

The more soluble sulfonamides are associated with fewer renal complications. Maintain adequate fluid intake to prevent crystalluria and stone formation.

**Information for Patients:** Maintain adequate fluid intake; urine will turn reddish-orange. **Laboratory Tests:** Perform urinalysis with careful microscopic examination at least once a week and regular blood counts after 2 weeks therapy; measure blood levels in patients with serious infection (see INDICATIONS). **Drug Interactions:** Sulfonamides may displace oral anticoagulants from plasma protein binding sites, increasing anticoagulant effect. Can also displace methotrexate. **Drug Laboratory Test Interactions:** May affect liver function tests in hepatitis.

**Carcinogenesis, Mutagenesis, Impairment of Fertility: Carcinogenesis:** Azo Gantrisin has not undergone adequate trials relating to carcinogenicity; each component, however, has been evaluated separately. Rats appear especially susceptible to goitrogenic effects of sulfonamides; long-term administration has resulted in thyroid malignancies in this species. Long-term administration of phenazopyridine HCl has induced neoplasia in rats (large intestine) and mice (liver). No association between phenazopyridine HCl and human neoplasia reported; adequate epidemiological studies have not been conducted. **Mutagenesis:** No studies available. **Impairment of Fertility:** The components of Azo Gantrisin have been evaluated in animal reproduction studies. In rats given 800 mg/kg/day sulfisoxazole, there were no effects on mating behavior, conception rate or fertility index. Fertility was not affected in a two-litter study of rats given 50 mg/kg/day phenazopyridine.

**Pregnancy: Teratogenic Effects:** Pregnancy Category C. The components of Azo Gantrisin have been evaluated. At 800 mg/kg/day sulfisoxazole was nonteratogenic in rats and rabbits, with no perinatal or postnatal effects in rats. In two other studies, cleft palates developed in rats and mice after 500 to 1000 mg/kg/day sulfisoxazole. No congenital malformations developed in rats given 50 mg/kg/day phenazopyridine. As there are no satisfactory animal or human studies, it is not known whether Azo Gantrisin can cause fetal harm or affect reproduction capacity. Use during pregnancy only if the potential benefit justifies the potential risk to the fetus. **Nonteratogenic Effects, Nursing Mothers and Pediatric Use:** See CONTRAINDICATIONS.

**ADVERSE REACTIONS: Allergic:** Anaphylaxis, generalized allergic reactions, angioneurotic edema, arteritis and vasculitis, myocarditis, serum sickness, conjunctival and scleral injection, periarteritis nodosa, systemic lupus erythematosus. **Cardiovascular:** Tachycardia, palpitations, syncope, cyanosis. **Dermatologic:** Rash, urticaria, pruritus, erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis, exfoliative dermatitis, photosensitivity. **Endocrine:** Goiter production, diuresis, hypoglycemia. Cross-sensitivity with some goitrogens, diuretics (acetazolamide and the thiazides) and oral hypoglycemic agents may exist. **Gastrointestinal:** Nausea, emesis, abdominal pain, anorexia, diarrhea, glossitis, stomatitis, flatulence, salivary gland enlargement, G.I. hemorrhage, pseudomembranous enterocolitis, melena, pancreatitis, hepatic dysfunction, jaundice, hepatocellular necrosis. **Genitourinary:** Crystalluria, hematuria, BUN and creatinine elevation, nephritis and toxic nephrosis with oliguria and anuria, acute renal failure, urinary retention. **Hematologic:** Leukopenia, agranulocytosis, aplastic anemia, thrombocytopenia, purpura, hemolytic anemia, anemia, eosinophilia, clotting disorders including hypoprothrombinemia and hypofibrinogenemia, sulfhemoglobinemia, methemoglobinemia. **Musculoskeletal:** Arthralgia, chest pain, myalgia. **Neurologic:** Headache, dizziness, peripheral neuritis, paresthesia, convulsions, tinnitus, vertigo, ataxia, intracranial hypertension. **Psychiatric:** Psychosis, hallucinations, disorientation, depression, anxiety. **Miscellaneous:** Edema (including periorbital), pyrexia, drowsiness, weakness, fatigue, lassitude, rigors, flushing, hearing loss, insomnia, pneumonitis.

**OVERDOSAGE: Signs:** Anorexia, colic, nausea, vomiting, dizziness, drowsiness, unconsciousness; possibly pyrexia, hematuria, crystalluria. Blood dyscrasias and jaundice may occur later. **Treatment:** Institute gastric lavage or emesis; force oral fluids; administer intravenous fluids if urine output is low with normal renal function. Monitor blood counts and appropriate blood chemistries, including electrolytes. In cyanosis, consider methemoglobinemia and treat with intravenous 1% methylene blue. Institute specific therapy for blood dyscrasias or jaundice.

**DOSAGE AND ADMINISTRATION:** Azo Gantrisin is intended for the acute, painful phase of urinary tract infections. The recommended dosage in adults is 4 to 6 tablets initially, followed by 2 tablets four times daily for up to 2 days. Treatment with Azo Gantrisin should not exceed 2 days. Treatment beyond 2 days should only be continued with Gantrisin (sulfisoxazole/Roche).

**HOW SUPPLIED:** Tablets, each containing 0.5 Gm sulfisoxazole/Roche and 50 mg phenazopyridine HCl—bottles of 100 and 500.



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vomiting at the age of 23 years. It persisted for the years during which she was bulimic until treated symptomatically with radiation. Her salivary glands seem less responsive to binging and vomiting since her third and most intense course of radiation. Since learning of the likely relationship between her eating habits and the parotid enlargement, however, she has become aware that swelling tends to occur three to six days after a severe bulimic episode.

Persistent, bilateral enlargement of the parotid glands in a young woman without apparent cause should suggest bulimia to the physician, especially if other signs and symptoms of the bulimic disorder are present, such as erosion of the dental enamel, hypokalemia, major adjustment problems, or depression. Because of the secretive nature of the disorder and the guarded manner of these patients, eliciting a history of the binge-vomiting behavior is not always easy. The patient's trust and confidence must be won and the matter of her eating habits approached with tact and understanding. Once the diagnosis is made, a program of treatment that often includes behavioral, cognitive, psychodynamic, and pharmacologic components is often successful in resolving the disorder.<sup>9,10</sup>

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