ORBITAL CELLULITIS FOLLOWING DENTAL EXTRACTION*

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INTRODUCTION

THE DEVELOPMENT OF ORBITAL CELLULITIS FOLLOWING EXTRACTION OF TEETH has been recognized clinically in a number of studies.[1-7] Because of the large number of dental extractions performed in the United States each year (estimated at 50 million in the American Dental Association Bureau of Economic and Behavioral Research 1979 Survey of Services Rendered), it is important to recognize the process by which this may occur. Organisms from an odontogenic source may gain entrance to the orbit through local tissue planes, by hematogenous spread, or by involvement of the paranasal sinuses.[7-9] With the present widespread use of antibiotics the clinician rarely observes the contiguous spread of dental infection to the orbit. When this process does occur the use of antibiotics may slow the spread of infection so that the underlying disease process may not be recognized. In some cases, however, such secondary factors as the virulence of the organism, the general health of the patient, or a poor choice in initial antimicrobial therapy may dispose certain patients to a rapid spread of infection.

In four cases presented in this paper, all patients demonstrated elevated white blood cell counts and radiologic evidence of acute ipsilateral paranasal sinus infection. Fever was present in three patients. Meningitis developed in one. Possible predisposing factors were pregnancy with an upper respiratory tract infection in one patient, heroin addiction in another, and nephrotic syndrome with chronic antral infection in a third. The interval between dental extraction and development of orbital symptoms ranged from 2 hours to 13 days. The sequelae—subdural empyema

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and death, severe loss of vision, blindness with ptosis and exotropia—demonstrate the need for early diagnosis and the immediate institution of appropriate antimicrobial therapy and surgical drainage when indicated.

CASE REPORTS

CASE 1

A 19-year-old Caucasian woman was admitted to the hospital with pain, swelling, and redness of the left periorbital region. This was associated with diplopia and decreased visual acuity in the left eye. The patient was 38 weeks pregnant. She had been in excellent health until 2 weeks before admission, when symptoms of a mild upper respiratory tract infection developed. One week before admission, she began to experience pain in the left upper second molar. The tooth was extracted 5 days before admission. Within 2 hours after the tooth was extracted, the patient noticed pain and swelling about the left eye. She returned to her dentist the next day and was informed that she had had "an allergic reaction to novacaine." The dentist prescribed oral diphenhydramine hydrochloride (Benadryl). On the second day after extraction, the dentist prescribed oral penicillin. This had no effect. The patient's symptoms continued to worsen, and on the fifth day after extraction, she experienced diplopia and decreased visual acuity in the left eye.

On initial hospital examination, the patient was afebrile. There was left-sided periorbital edema in addition to significant proptosis and chemosis (Fig 1). The visual acuity was 20/20 in the right eye and 20/200 in the left eye. A left-sided afferent pupillary defect was noted. Extraocular muscle function of the left eye was reduced in all fields of gaze. Funduscopy revealed choroidal folds in the left eye. There was no evidence of infection at the dental extraction site. Sinus roentgenograms showed a pansinusitis on the left side (Fig 2). The white blood cell count was 12,700.

Therapy with intravenous ampicillin (1 gm every 6 hours) and oxacillin (1 gm every 6 hours) was begun, and a subperiosteal abscess located in the medial posterosuperior aspect of the left orbit was drained of a moderate amount of purulent material. Nongroupable beta-hemolytic *Streptococcus* was subsequently cultured from this material.

Postoperative recovery was dramatic. On the fourth day the intravenous antibiotics were discontinued. Oral dicloxacillin (500 mg every 6 hours) and ampicillin (500 mg every 6 hours) were prescribed. Visual acuity at discharge was 20/20 in both eyes and no afferent pupillary defect was present. The results of a follow-up examination 1 month later were completely normal (Fig 3). In the interim, the patient had delivered a healthy, full-term infant.

CASE 2

A 35-year-old black man was admitted to the hospital because of left-sided periorbital edema and tenderness. The patient was a chronic heroin addict undergoing treatment that included oral methadone (40 mg daily) therapy. Thirteen days

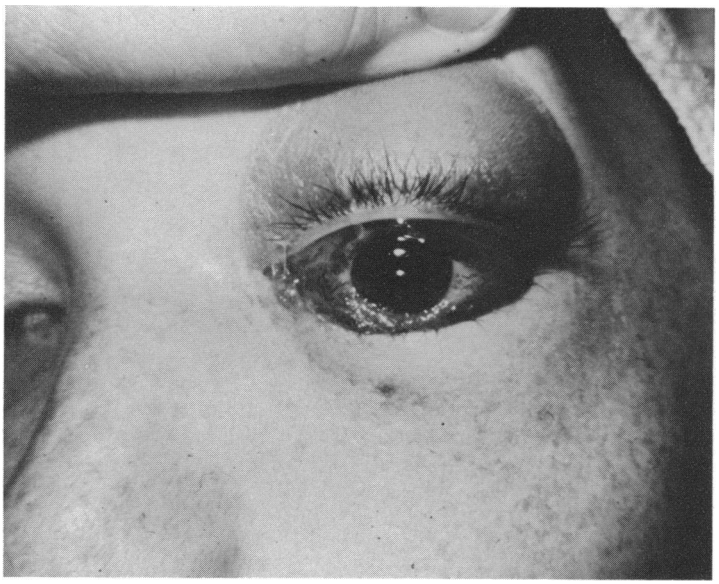

FIGURE 1
Case 1 with left-sided periorbital edema, proptosis, and chemosis.

before admission he was seen in the dental clinical for pain of 1 month's duration in the left upper third molar. A panorex roentgenogram showed irreversible pulpitis and periapical periodontitis (Fig 4). The tooth was extracted, and the patient was given oral penicillin (250 mg every 6 hours). Two days after the extraction the patient returned to his dentist complaining of a severe headache and purulent discharge from the left nostril. There was no evidence of infection at the extraction site. He was given propoxyphene hydrochloride (Darvon) for pain and dismissed. Thirteen days after the extraction he again returned to his dentist complaining of severe headaches, nasal drainage, and pain at the extraction site. No facial or periorbital edema was noted and the extraction site was unchanged. He was again dismissed. Later in the day, left-sided periorbital edema and tenderness developed; the patient was then admitted to the hospital.

On initial examination, the patient was alert and febrile (T = 100.7 F). There was extensive periorbital edema on the left side with significant chemosis and proptosis. Extraocular muscle function on the left side was reduced in all fields of gaze. The pupillary light reflexes were reported to be normal. There was pain on palpation over the left maxillary sinus. No evidence of infection was present at the site of dental extraction. Sinus roentgenograms revealed opacification of the left maxillary and sphenoid sinuses. The white blood cell count was 17,000 with a significant left shift.

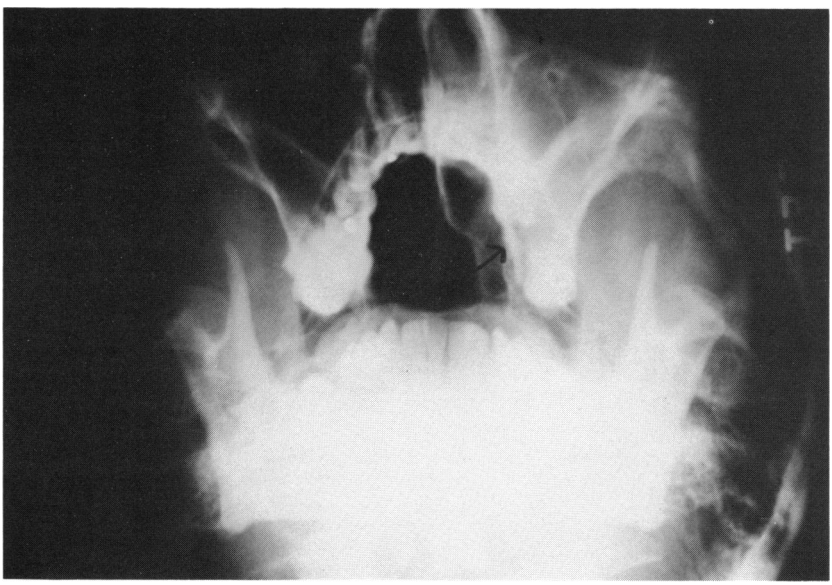

FIGURE 2
Sinus roentgenogram of same patient, showing opacification of left ethmoid and maxillary sinuses. *Arrow* indicates site of tooth extraction.

Therapy was begun with intramuscular ampicillin (500 mg every 6 hours) and warm compresses to the left orbit. The patient remained febrile, and the periorbital edema and chemosis increased. On the second day the left pupil reacted sluggishly. No change in the therapeutic regimen was undertaken. On the third day, there was no light perception in the left eye.

An otolaryngologist attempted to drain the orbital abscess through a small stab incision made in the supranasal aspect of the left orbit. A minimal amount of purulent material was recovered. In addition, a left nasoantral window was created, and 20 ml of purulent material was drained from the maxillary sinus. Cultures of this material subsequently grew penicillinase-producing *Staphylococcus aureus* and alpha-hemolytic *Streptococcus*. Postoperatively, the antibiotic regimen was changed to intravenous methicillin (1 gm every 4 hours) and oral erythromycin (500 mg every 6 hours). The patient's condition deteriorated over the next 4 days, with increasing periorbital edema and proptosis.

On the seventh day, he was seen in consultation. There was massive left-sided periorbital edema and proptosis (Fig 5). A large amount of foul-smelling purulent material was expelled through a drainage site that opened spontaneously in the left lower lid during examination.

In surgery, copious amounts of purulent material were evacuated from the left orbit through an incision made in the drainage site of the lower lid. Additional

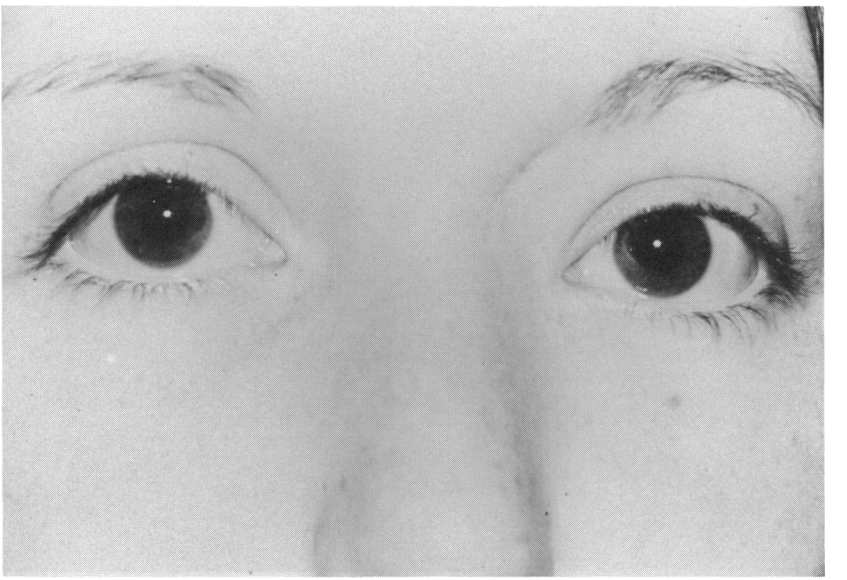

FIGURE 3
Same patient (case 1) 1 month after resolution of infection.

purulent material was evacuated through incisions made in the upper lid. Cultures of the material subsequently grew *Enteroacter aerogenes*, micrococci, and pneumococci. Postoperatively, the patient was given intravenous methicillin (2 gm every 4 hours), gentamicin (80 mg every 8 hours), and clindamycin (600 mg every 8 hours). His condition improved during a 10-day period, and the antibiotic regimen was changed to oral cloxacillin (500 mg every 6 hours). On discharge there was no light perception in the left eye. Visual acuity in the right eye was 20/20. Funduscopy of the left eye showed optic atrophy. In a follow-up examination 8 months later, ptosis and exotropia were noted on the left side. Cosmetic improvement was achieved by a left lateral rectus muscle recession and a left Fasanella-Servat procedure[10] (Fig 6).

CASE 3

A 21-year-old Caucasian man was admitted to the hospital with complaints of fever, lethargy, and right-sided periorbital edema. The patient had a nephrotic syndrome of unknown cause. During the month before his admission the patient had been taking oral penicillin for an abscess of the right upper second molar. The tooth was extracted and within 36 hours after extraction orbital edema developed on the right side and the patient became febrile (T = 102 F) and lethargic.

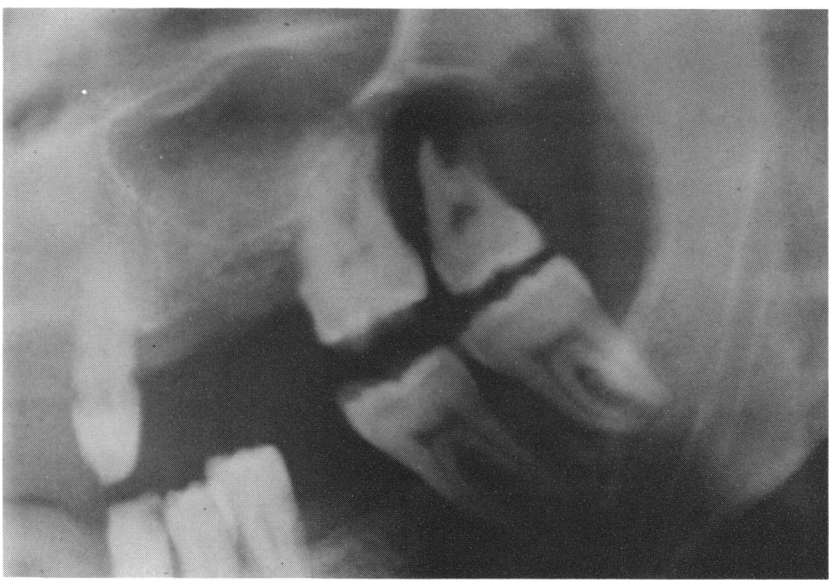

FIGURE 4
Panorex roentgenogram of case 2, showing pulpitis and periapical periodontitis of left upper third molar.

On initial examination, the patient appeared ill and lethargic but was responsive to verbal commands. The temperature was 100.4 F. There was marked right-sided periorbital edema, ptosis, proptosis, and chemosis. Abduction of the right eye was restricted. The pupillary light reflexes were normal. The results of funduscopy were normal in both eyes. Examination of the mouth revealed extensive dental caries. There was no evidence of infection at the site of the tooth extraction; several sutures were present in the tooth socket. Other findings of the physical examination were within normal limits. The white blood cell count was 21,700 with a significant left shift. The serum blood urea nitrogen level was 91 mg/100 ml; the creatinine level was 9 mg/100 dl. The cerebrospinal fluid was cloudy and contained 7800 neutrophils/ml. The cerebrospinal fluid protein and glucose levels were 216 mg/dl and 30 mg/dl, respectively. Roentgenograms showed opacification of the right maxillary, sphenoid, and ethmoid sinuses.

Therapy with intravenous chloramphenicol (1.5 gm every 6 hours) was begun. On the first day, a nasoantral window was created by an otolaryngologist, and a large amount of foul-smelling purulent material was evacuated from the maxillary sinus. The mucosa of the right maxillary sinus appeared chronically inflamed. Multiple cultures of the blood, cerebrospinal fluid, and purulent material from the paranasal sinuses showed no growth.

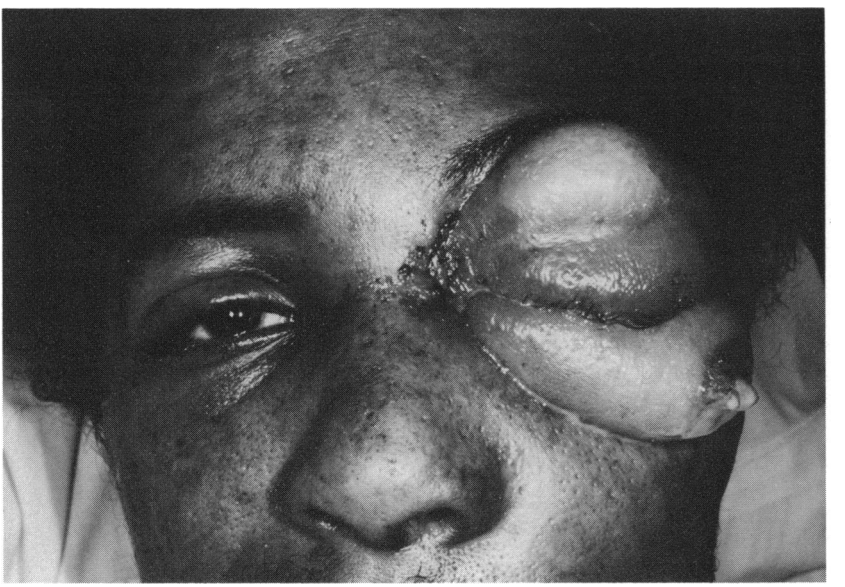

FIGURE 5
Same patient (case 2) with massive left-sided periorbital edema. Note drainage site in left lower lid.

During the next 7 days the orbital infection responded dramatically to an expanded antibiotic regimen of intravenous tobramycin (80 mg every 12 hours), nafcillin (1 gm every 4 hours), clindamycin (600 mg every 8 hours), and intrathecal gentamicin (1 dose). The neurologic status improved and there was a significant decrease in the right-sided periorbital edema and proptosis; however, the patient's condition was complicated by the onset of acute renal tubular necrosis with oliguria and circulatory volume overload with continuous peritoneal dialysis and digitalis. Adult respiratory distress syndrome developed and necessitated mechanical ventilation for 3 days.

On the eighth day of hospitalization, the patient complained of severe pain in the right eye. Examination showed a significant increase in the right-sided periorbital edema and proptosis. Chemosis was present, and induration of the right lower lid was noted. An attempt to drain this area through a tiny stab incision made in the right lower lid produced a small amount of purulent material. Examination of this material showed gram-positive cocci and rods, in addition to gram-negative rods. Subsequent cultures of this material revealed no growth. The patient did not improve and on the tenth day of hospitalization he was seen in ophthalmic consultation.

There was massive right-sided periorbital edema with proptosis and chemosis (Fig 7). Visual acuity in the right eye was limited to light perception. The right

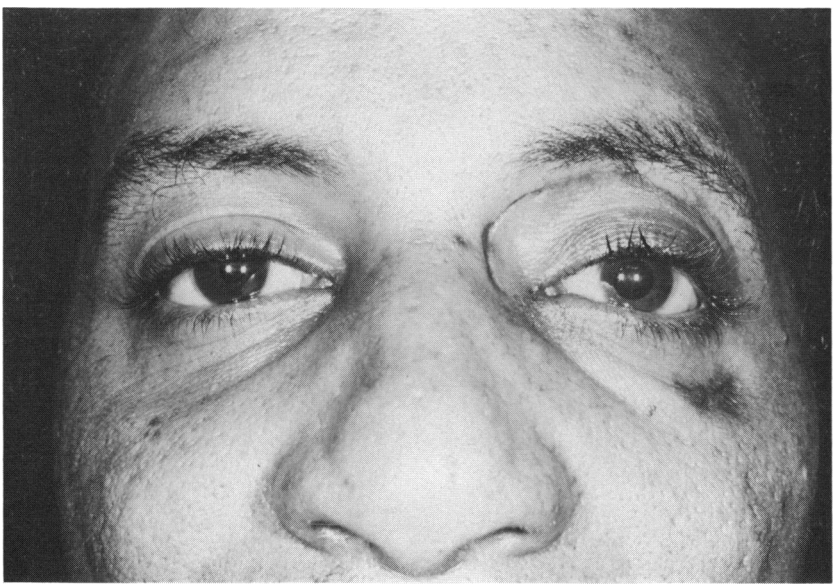

FIGURE 6
Same patient (case 2) 10 months after resolution of infection and 2 months after strabismus and ptosis surgery.

globe was frozen, and the pupil was nonreactive. The right nostril contained black necrotic material that appeared to be draining from the nasoantral window. Copious amounts of foul-smelling purulent material were drained through a 4.5-cm incision made in the supranasal aspect of the right orbit. Large amounts of foul-smelling, black, necrotic material were also drained from stab incisions made at the infratemporal, supratemporal, and infranasal margins of the orbit. A right external ethmoidectomy was performed and additional purulent material was recovered. Drains were then placed and the wounds closed (Fig 8). Cultures of the recovered purulent material subsequently grew microaerophilic *Streptococcus*.

Postoperatively, the orbital infection resolved during a 3-week period. Peritoneal dialysis was continued for 6 weeks. On discharge, visual acuity in the right eye was limited to bare light perception. The patient did not return for follow-up examination.

CASE 4

A 12-year-old black boy was seen by an otolaryngologist because of swelling and tenderness in the right periorbital region. Seven days earlier, he had undergone extraction of the right upper first molar. Two days after extraction, right-sided periorbital edema and tenderness developed. On initial examination, the patient

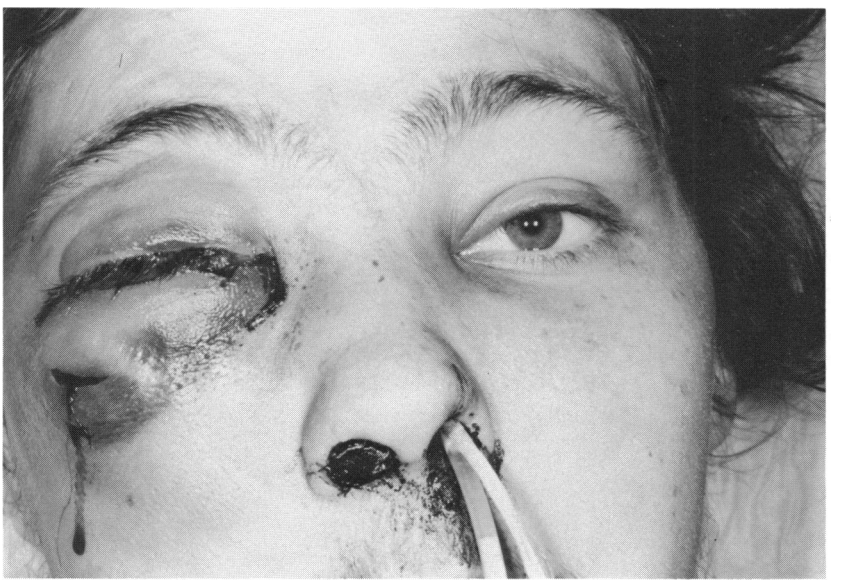

FIGURE 7
Case 3 with right-sided periorbital edema. Note small drainage incision in right lower lid.

was alert and febrile (T = 101 F). There was extensive nonfluctuant edema in the right periorbital region. Visual acuity and extraocular muscle function were normal. The site of the dental extraction showed no evidence of infection, and the remainder of the physical examination was reported to be within normal limits. The white blood cell count was 10,700 with a slight left shift. Computerized axial tomography revealed the presence of right-sided pansinusitis (Fig 9).

A diagnosis of periorbital cellulitis was made. Blood, nasal, and pharyngeal cultures were obtained, and intravenous cephalothin therapy (1 gm every 4 hours) was started. On the second day of hospitalization the patient's right-sided periorbital edema increased. In addition, right-sided proptosis and chemosis were noted. The fever increased to 103 F. Blood, nasal, and pharyngeal cultures showed no growth. On the third day, the patient became lethargic. Signs of meningeal irritation were present, and deep tendon reflexes were absent. Computerized axial tomography revealed a soft tissue density in the right retroorbital region with displacement of the optic nerve laterally, a 10-mm right-sided proptosis, and a 4-mm shift of midline cerebral structures to the left.

The patient was then seen in ophthalmic consultation. He was severely obtunded, reacting only to pain. There was massive right-sided periorbital edema and proptosis, as well as chemosis, subconjunctival hemorrhage, and exposure keratopathy of the right eye. The right pupil was 7 mm and fixed; the left pupil

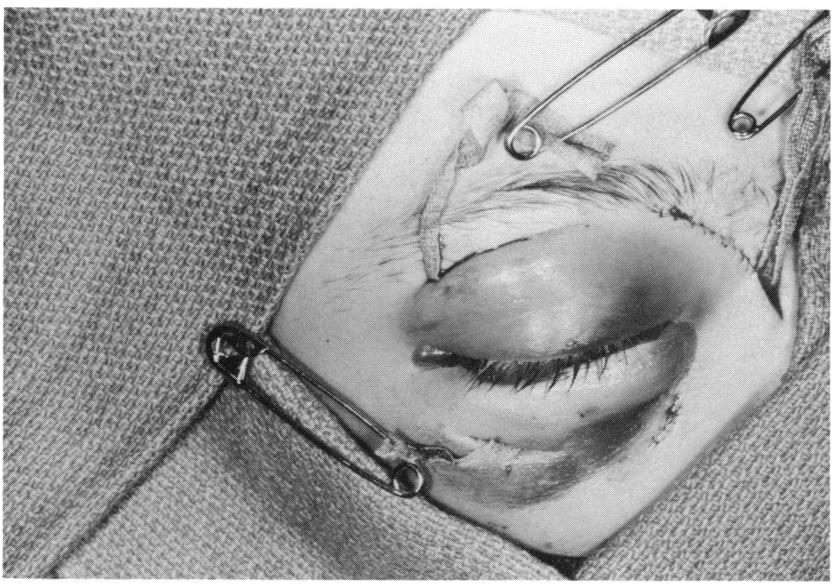

FIGURE 8
Surgical photograph of same patient (case 3). Note multiple drainage sites.

was 5 mm and reacted sluggishly to light. Funduscopy of the right eye showed papilledema.

In surgery a 4.5-cm Lynch incision was made and a large orbital abscess with profuse purulent material was drained. Cultures of this material subsequently grew microaerophilic *Streptococcus*.

Postoperatively, the antibiotic regimen was changed to parenteral gentamicin (75 mg every 6 hours), clindamycin (600 mg every 6 hours), methicillin (1 gm every 6 hours), and chloramphenicol (1 gm every 6 hours). The neurologic status continued to deteriorate. An electroencephalogram revealed electrocortical silence. Computerized axial tomography showed a 13-mm shift of the cerebral midline structures to the left. The patient required mechanical ventilation, and hypotension necessitated a dopamine drip for maintenance of blood pressure. Diabetes insipidus developed, complicating fluid management. On the seventh day of hospitalization, the patient suffered cardiopulmonary arrest and died.

Postmortem examination revealed the presence of a large subdural emphyema overlying the right hemisphere (Fig 10). Eighty milliliters of purulent material was recovered from this area. Cultures of this material grew microaerophilic *Streptococcus*. Tentorial herniation and compression of the midbrain were present. A soft, yellowish, necrotic area was present on the orbital surface of the frontal bone. Histologic examination of the supraorbital dura overlying this area

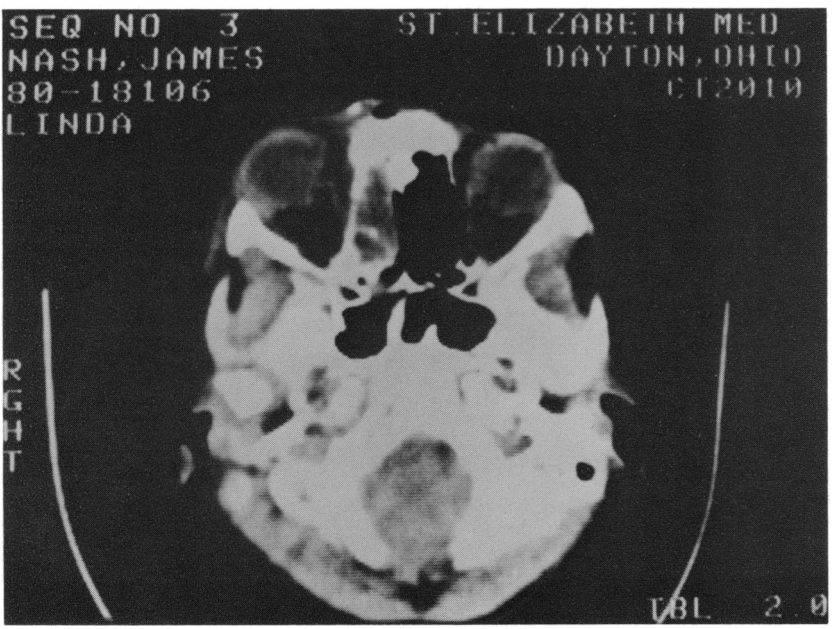

FIGURE 9
Computerized axial tomographic scan of case 4, showing opacification of right ethmoid sinus.

showed severe necrotizing inflammation with many organisms and disruption of the dura. Microscopic examination of the meninges overlying the cerebral hemispheres revealed marked congestion and a low-grade inflammatory response. Twenty milliliters of purulent material was recovered from the maxillary sinus. The frontal sinus contained no purulent material. The dural venous sinuses were normal. Sensitivity testing of the isolated organism (microaerophilic *Streptococcus*) revealed resistance to penicillin, ampicillin, gentamicin, and cephalosporins, and sensitivity to tetracycline, erythromycin, and clindamycin.

DISCUSSION

Before antibiotics were available, a thorough working knowledge of regional head and neck anatomy was required for the successful treatment of a spreading dental infection. Classical anatomical studies published in the 1930s, by Dingman[11] and others,[12-14] analyzed the fascial compartments in the head and neck wherein infections may gain entrance to the orbit via several pathways. Purulent material from an apical periodontal abscess travel in the path of least resistance. The buccal cortical plate of

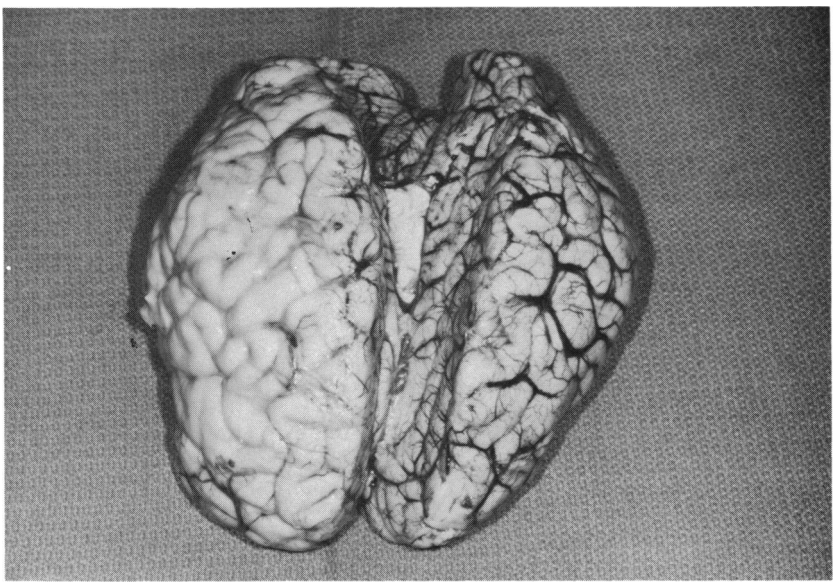

FIGURE 10
Postmortem photograph of brain of same patient (case 4), showing subdural empyema on right.

the alveolar process overlying the maxillary teeth is very thin; thus, most abscesses penetrate buccally.[14] An abscess originating in the maxillary molars may penetrate the buccal cortical plate above the origin of the buccinator muscle and spread to the soft tissues of the cheek.[1,15]

Orbital involvement may then ensure, either by direct spread through local tissue planes or by an ascending facial thrombophlebitis.[1,8,15] The ascension of a facial thrombophlebitis to the orbit is facilitated by the absence of valves in the ophthalmic veins, resulting in an extensive two-way communication between the facial and orbital venous networks. Sicher and DuBrul[8] have described the process by which orbital cellulitis may preceed the development of cavernous sinus thrombosis while the septic thrombophlebitis spreads sequentially through the facial and superior ophthalmic veins. About 7% of cases of cavernous sinus thrombosis are of dental origin.[16] The most extensive communication between the facial and ophthalmic veins occurs at the inner canthal region above the medial palpebral ligament. An abscess of the first maxillary premolar or canine tooth is in close proximity to this region and should be handled with great care.[15]

Infections of the maxillary molars may also spread posteriorly into the infratemporal and pterygopalatine fossae.[1,2,7,15] Purulent material may then extend along the tuberosity of the maxilla and thus gain access to the orbit through the inferior orbital fissure.[1,15] This fissure is closed by a strong fascia and smooth muscle fibers; however, an opening is often found at its lateral aspect.[17]

Because of the intimate anatomic relationship between the paranasal sinuses and the orbit, paranasal sinusitis is the most common cause of orbital cellulitis.[18-21] In the adult, more than 50% of the orbital circumference is surrounded by sinus cavities. Venous blood flows freely between the ophthalmic and ethmoid veins.[22] An extensive plexus of veins near the nasolacrimal duct communicates freely with venous plexuses of the turbinates, the linings of the sinuses, and the ophthalmic veins.[23] In addition, there can be congenital dehiscences in the medial and superior walls of the orbit.[24]

It is, therefore, not surprising that odontogenic infections most commonly reach the orbit through the paranasal sinuses.[1] As many as 20% of all cases of sinusitis have been attributed to dental origin.[25] The apices of the maxillary molars and premolars are in close apposition with the floor of the maxillary sinus. Occasionally the apices of these teeth are in direct contact with the mucous membrane of the sinus. During extraction of an abscessed tooth, the floor of the sinus may be fractured, resulting in innoculation of the antrum with purulent material. In 1927, Hempstead[26] reviewed 385 cases of maxillary sinusitis. In 22.5% of these cases, the infection was attributed to extraction of abscessed molars. In 16% of the cases, a fistula extended through the alveolar process into the maxillary antrum.

In all of the cases detailed in the present study, development of orbital cellulitis followed extraction of an ipsilateral maxillary molar (Table I). Three of the four patients were febrile on admission. All had elevated white blood cell counts. Roentgenographic evidence of paranasal sinus infection was present on admission in all cases. In each patient, it is probable that infection reached the orbit via the paranasal sinuses.

The time interval between extraction of the tooth and onset of orbital symptoms ranged from 2 hours (patient 1) to 13 days (patient 2). Severe headaches and purulent nasal discharge developed within 2 days in the second patient, indicating that infection had spread to the paranasal sinuses. In cases 1 and 3, the spread of the infection to the orbit was particularly rapid. Case 3 had evidence of chronically inflamed mucosa in the maxillary sinus. In addition, his debilitated state of health was a likely contributing factor in the accelerated spread of the infection not only to

TABLE I: CLINICAL SUMMARIES OF PATIENTS WITH ORBITAL CELLULITIS FOLLOWING DENTAL EXTRACTION

	CASE 1	CASE 2	CASE 3	CASE 4
Tooth extracted	Left upper 2nd molar	Left upper 3rd molar	Right upper 2nd molar	Right upper 1st molar
Interval from extraction to onset of orbital symptoms	2 hours	13 days	36 hours	2 days
Findings on admission:				
Temperature	Afebrile	100.7 F	100.4 F	101 F
White blood cell count	12,700	17,000	21,700	10,700
Sinus involvement	Left pansinusitis	Left maxillary and sphenoid	Right maxillary, sphenoid, and ethmoid	Right pansinusitis
Procedures	Day 1: drainage of orbital abscess	Day 3: stab incision upper lid Day 7: drainage of orbital abscess	Day 1: nasoantral window Day 8: stab incision lower lid Day 10: drainage of orbital abscess; external ethmoidectomy	Day 3: drainage of orbital abscess
Predisposing factors	Pregnancy, upper respiratory infection	Heroin addiction	Renal disease, chronic antral inflammation	None
Organisms isolated	Nongroupable beta-hemolytic *Streptococcus*	Maxillary sinus: *S aureus*, alpha-hemolytic *Streptococcus* Orbit: *Enterobacter aerogenes*, micrococci, pneumococci	Microaerophilic *Streptococcus*	Microaerophilic *Streptococcus*
Outcome	Complete recovery	Complete loss of vision, ptosis, exotropia	Severe loss of vision	Subdural empyema, death

the orbit, but also to the meninges. In case 1, orbital symptoms developed only 2 hours after extraction. Her 2-week history of coryza preceding the dental extraction may have contributed to a rapid spread of infection through an already inflamed antral mucosa. The pregnancy itself may have predisposed her to the original odontogenic infection. The rate of occurrence of gingivitis during pregnancy has been reported at from 30% to 100%, reaching a peak during the 8th month of gestation.[9]

In cases 3 and 4, infection spread to involve the central nervous system. In case 4, a subdural empyema developed approximately 10 days after dental extraction; in case 3, meningitis developed in the patient 36 hours after extraction. In 1945, Haymaker[7] analyzed 28 cases of fatal central nervous system infections following dental extraction. Spread of the odontogenic infection to the intracranial cavity was attributed to direct extension in 17 cases and to hematogenous spread in 11 cases. His series included six cases of intraorbital abscess. In three of these cases, the orbital abscess was directly involved in the subsequent intracranial spread of infection. In two cases, cavernous sinus thrombosis occurred. In the remaining case, purulent material from the orbital infection penetrated the frontal bone and frontal sinus, ultimately gaining entrance to the subdural space. A similar process appears to have occurred in case 4. The orbital infection spread upward to involve the orbital roof. Subsequent disruption of the dura overlying the orbital portion of the frontal bone allowed organisms to invade the subdural space.

These cases demonstrate the importance of appropriate antimicrobial therapy and the implementation of adequate and timely surgical drainage when indicated. In case 4, the infecting organism was resistant to the initial antibiotic employed and drainage of the orbital abscess was delayed, allowing the infection to penetrate the orbital roof and invade the subdural space. A similar situation occurred in case 2, in which the infecting organism also was resistant to the initial antibiotic employed. In general, when Gram staining and cultures of the infecting organisms are unavailable, multiple-drug regimens should be used. Agents should be administered intravenously and in high doses. Initial therapy should include a penicillinase-resistant penicillin. Ampicillin should be included in patients less than 4 years old to provide coverage against *Hemophilis influenze*.[27] The clinician should be aware that a patient with orbital cellulitis may respond dramatically to initial antimicrobial therapy, only to relapse several days later after an orbital abscess has formed, as occurred in case 3. After 7 days of antimicrobial therapy, the patient's orbital signs had almost returned to normal; however, within 24 hours the periorbital edema, proptosis, and chemosis returned, and the visual acuity in the eye was severely impaired.

Deciding when intensive antimicrobial therapy must be augmented with surgical drainage requires astute clinical judgment. Duke-Elder[28] and Dingman[11] have emphasized the difficulties inherent in making this decision. If drainage is attempted prematurely, a localized infection may be disseminated throughout the orbital tissues. Conversely, if left undrained, a true orbital abscess that has not responded to medical therapy may result in loss of vision, cavernous sinus thrombosis, meningitis, subdural empyema, or brain abscess. If the clinical situation is ambiguous and a decision is made to delay surgery, the ophthalmological findings must be followed meticulously. B-scan ultrasonography and computerized axial tomography of the orbit may aid in the detection of an abscess[29,30]; however, careful observation of the eye should take precedence in influencing the decision to attempt surgical drainage. The presence of choroidal folds may represent traction on the optic nerve.[31-33] Increasing proptosis and chemosis associated with extraocular muscle impairment are general indications for surgery. Surgical drainage should be employed before impairment of vision develops.

It is essential that any surgical drainage be established adequately: small stab incisions proved inadequate in cases 2 and 3. In general, a large incision should be used. This can be made over the region of a suspected abscess or in the supranasal quadrant of the orbit (Lynch incision). Additional incisions can be made if needed. Drains should be placed in the wounds and advanced a small distance each day.

The high incidence of dental extraction makes it important to recognize the possibility of infection that may spread to the orbit, and the potential consequences of such an infection must not be underestimated. The one fatality occurred in a previously healthy 12-year-old boy. No apparent predisposing factors were uncovered that should have rendered him susceptible to such a rapid spread of infection. This demonstrates the importance of careful choice in initial antimicrobial therapy and close monitoring of physical signs once therapy has been instituted.

ACKNOWLEDGMENTS

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DISCUSSION

DR R. R. WALLER. I appreciate very much the opportunity to discuss this informative and beautifully presented paper. There are a variety of mishaps affecting the visual system which occur while in the dental office. Most injuries, fortunately, are not serious, only transiently affect vision and can be prevented by wearing protective eyewear. Doctor Robert Hales, in 1970, reported several cases of ocular injuries sustained in the dental office, including corneal abrasions from an explosion of an ampule of local anesthetic; chemical keratitis from pumice, embedded corneal foreign bodies of alloy materials, and the resultant recurrent corneal erosions which often follow. Corneal perforation was also reported in his paper when, during the course of filling a tooth of a 10-year-old girl, a double-ended excavation instrument which was caught in a gauze, flipped into the air, and impaled in the child's eye. Blaxter and Britten in 1961 and Cooper in 1962 reported transient amaurosis, diplopia, and pupil dilation following mandibular nerve block with procaine. The probable explanation was that an intra-arterial injection had taken place and because of anastomosis between the internal and external carotid system, the anesthetic agent reached the eye. This latter explanation makes sense, especially in those situations where the middle meningeal artery is anomalously the major blood supply to the orbit.

Doctor Bullock's paper reminds us of yet another complication of dental surgery which in contrast is not an immediate complication, but rather a delayed event of days or weeks and it can be devastating. The entrance of organisms through local tissue planes is probably the least common route of entry for the pathogen in these cases in the 1980s. On the other hand, entry of organisms by hematogenous route appears more plausible. Doctor Bullock pointed out the presence of rich anastomoses between facial and ophthalmic veins and mentions the more than chance relationship between dental disease and cavernous sinus thrombosis. That rich vascular anastomoses between the blood supply to the sinuses and the orbit also exist is evidenced by reports in the literature detecting immediately within the retinal vessels, choroidal circulation, and conjunctival vessels, the presence of depo-steroid, following injection into the nasal cavity for treatment of allergic polyps. The common relationship between paranasal sinusitis and orbital cellulitis is beautifully documented in this paper, in that all four patients presented with radiologic evidence of paranasal sinusitis on the side of dental extraction. It is also striking that all patients had upper teeth extracted, no lower teech, and we are reminded that the apices of the maxillary premolars and molars can be in contact with the maxillary sinus mucosa.

The principles of management of orbital cellulitis have been emphasized and include hospitalization, close monitoring, and vigorous (as well as appropriate)

antibiotic therapy. This is not the time for outpatient care. Orbital cellulitis should be a true ophthalmic emergency. We prefer to evaluate separately the signs of orbital inflammation (chemosis, lid swelling, erythema, proptosis, displacement, strabismus, and visual disturbance) recording trends in the medical record at least every 12 hours, and more frequently if necessary. If the clinical condition deteriorates, one must reevaluate medical therapy, but deterioration should mean abscess until proven otherwise, and this means emergency evaluation with computed tomography (CT) or ultrasound. CT examination appears to more precisely identify the smooth, dome-shaped, more readily drained subperiosteal abscess confined by the periorbita adjacent to the affected sinus. Perhaps Doctor Bullock would comment on efficacy of CT *vs* ultrasound in this situation. Abscesses should be drained externally through adequate incisions, not small stab wounds. We believe the establishment of excellent drainage from the contiguous sinus into the nasopharynx if the abscess is in the peripheral orbital space.

Doctor Bullock has reminded us of the importance of early detection and aggressive management of orbital cellulitis. This can avoid the serious ocular complications of ophthalmoplegia and visual loss, and the tragic complications of meningitis and death. I have enjoyed very much having the opportunity to discuss this excellent paper.

Dr Leonard Apt. Several years ago we experienced a small epidemic of eye and central nervous system complications following dental surgery in the colony of monkeys at the UCLA Medical Center. The complications included orbital cellulitis, retrobulbar abscess, orbital apex syndrome, superior orbital fissure syndrome, and brain abscess followed by death.

After a monkey escaped from his cage and mutilated the leg of a caretaker, the staff veterinarians decided to remove or saw off the canine teeth of the monkeys to prevent further mishaps. Most often the tooth was cut off at the gum level, the nerve destroyed, and the canal packed in the manner used by dentists to preserve a tooth.

Ocular infections followed these procedures. The baboon shown on the projected slide developed orbital cellulitis a few days after dental surgery. Orbital swelling was followed by proptosis, a hyperemic fixed globe, and systemic signs and symptoms of infection and toxicity. The dental consultant ordered systemic chloramphenicol therapy. Little improvement was seen after 1 week. Orbital incision and drainage was attempted. No frank pus was obtained. Routine cultures of the scant aspirate were negative. Cultures for anaerobic bacteria were not taken by the attending veterinarian. A change of antibiotic therapy to methicillin and gentamicin was recommended. The baboon's condition improved temporarily, but later in the week he was found dead in his cage. A brain abscess was found at autopsy.

In recent years at the Jules Stein Eye Institute we have seen two adults who developed severe orbital cellulitis following dental extraction. Both patients were diabetic. One patient, a dentist, responded well to antibiotic therapy. The other patient died when the markedly disturbed metabolic state, complicated by the

infection, could not be controlled. These events suggest that diabetes may be another predisposing factor contributing to the occurrence of orbital infection after dental procedurs. I suspect that the spread of infection to adjacent structures (soft tissue, sinuses, etc) and the eye occurs more frequently than surmised but is not reported because improvement occurs with prompt antibiotic therapy.

One intriguing aspect of this subject is why eye complications and infections elsewhere in the body do not occur more frequently after dental procedures since transient bacteremia is not uncommon. In reported studies the incidence of bacteremia following extractions has ranged from 15% to 85%. The higher incidences are found more often in the studies that included culture procedures for anaerobic (normal in the mouth) as well as aerobic bacteria. Perhaps the reason for negative culture reports in cases of eye and other body infections following dental extraction is the failure to use appropriate culture methods for both groups of bacteria. Failure to identify the causative microorganisms deters the choice of proper antibiotic therapy.

From dental studies we learn that transient bacteremia sometimes occurs after seemingly innocuous manipulation around teech, even in patients with healthy-appearing gingiva. For example, it may occur after tooth brushing, vigorous flossing, and the use of Stimudents and the Water Pik. The incidence of bacteremia increasese if periodontal disease exists.

Complications from transient bacteremia after dental extraction is averted in the healthy individual mainly by the body's immune system. Certain conditions, however, predispose a person to infection, and therefore antibiotic prophylaxis should be considered before dental treatment. This group would include patients who are chemically ill, debilitated, or immunosuppressed, patients with metabolic disorders such as the nephrotic syndrome (described by Doctor Bullock this morning), and patients and diabetes as mentioned by me earlier. For years, cardiologists have given antibiotics prophylactically to patients with congenital or acquired heart disease to avoid bacterial endocarditis. Recently orthopedic surgeons have recommended that patients with prosthetic hips receive prophylactic antibiotic therapy because there have been some cases of infection in quiet, healed joints after dental procedures. Similarly, I would seriously consider giving antibiotics prophylactically to a patient who had an intraocular lens implant. The artificial lens could act as a nidus for circulating microorganisms released into the bloodstream during dental treatment.

I have enjoyed Doctor Bullock's presentation and Doctor Waller's remarks. I appreciate the opportunity to share my thoughts and experience on this important subject.

Dr F. C. Blodi. I think we are all indebted to Doctor Bullock for calling our attention again to the pathogenesis of orbital cellulitis. I would like to emphasize three points:

1) It is interesting that most of these patients are quite young and the same is borne out in Doctor Bullock's series. I am not sure why because you would expect

that many more older patients get a tooth extraction and yet most of these patients are children.

2) It is not necessarily a pansinusitis that produces the orbital cellulitis. Doctor Bullock has mentioned that. I vividly remember a case which is still in the courts. A young child was managed, again poorly, by a pediatrician but no ophthalmic consultant had been called in. An otolaryngologist on repeated examinations did not find a pansinusitis.

3) For the third point, I would like to stress what Doctor Waller has already mentioned: management of this condition is obviously an emergency. There is no question about that, but the great question remains when and where to drain. The list of indications for drainage was shown in Doctor Bullock's paper and is a little bit vague. When is the motility somewhat reduced and when is the condition getting worse? This reminds me of medieval mysticism. We can follow the changes in the orbit nowadays with computerized tomography scans and ultrasound (preferably with both) very well. These patients have to be examined daily and the minute an abscess forms, it has to be drained. These wild stab incisions, not knowning where and when to drain, is 19th century medicine.

Dr William Jarrett. I think Doctor Bullock has done us a service by calling attention to this now rare disease. Although rare today, it was something that was quite common in the past, and it illustrates the changes in the disease patterns that come about with advances in medicine, in this case antibiotics. The widespread use of antibiotics, for example, completely changed the nature of otolaryngology as a specialty; acute mastoiditis with resultant intracranial abscess was a common and serious complication in the past but these are now rarely seen, as are cases of orbital cellulitis. Most residents will go through their training without ever seeing such a case, and a presentation such as Doctor Bullock's is needed to raise the level of our consciousness in order to make the correct diagnosis.

I have seen several such cases in my own practice; none of mine were the result of dental extraction, but all were due to pansinusitis. I think the important lesson to be learned is to think about this disease when you see such a patient. When a disease becomes uncommon, the diagnosis may be missed initially, with potentially disastrous results.

I cannot stress too much the importance of draining an orbital abscess, as Doctor Blodi pointed out. It is hard to know exactly when to intervene surgically. If just a cellulitis is present, drainage doesn't do any good and may indeed spread the infection. But if a subperiosteal orbital abscess develops, drainage becomes manditory. When drainage is done, an adequate incision down through the periosteum is needed, or the case won't be cured.

Again, I think it is important for us to be exposed to these cases and to know that they do occur even in this day of widespread antibiotic usage.

Dr Melvin Alper. To carry this group of complications further, Doctor Apt has referred to the bacteremia that occurs following dental intervention. I recently had a physician patient who came in with an occlusion of the central retinal artery.

He was running a slight fever. We had him evaluated. He had gone to a dentist about 2 weeks previous to the occluded vessel. He thought he had had a viral syndrome. We studied him and found that he had prolapse of the mitral valve (Barlow's) syndrome. He had developed vegetative endocarditis and had thrown an embolism which had occluded the central retinal artery. This is another etiology to add to your long list of ocular complications from visits to the dentists' office.

Dr Ronald Burde. I think the only point I would like to make is that the list that Dr Dan Jones gives for treating is for bugs that are assumed to be anaerobic for the most part and that what we are dealing with here are bugs that may be microaerobic, microaerophilic, or may be totally aerobic. I would ask Doctor Bullock to address the fact that in fact when one is dealing with a source that may have this type of organism, might one better use a different group of antibiotics or add at least one that is more specific for anaerobic organisms? I believe if we use only penicillin and gentamicin in these cases, we will miss treating the offending organism. That, of course, does not obviate the need for drainage of the abscess, but it may help us with the prevention of hematogenous spread or contiguous spread to the central nervous system.

Dr William H. Spencer. Nobody has mentioned anything about phycomycotic infection. Phycomycotic orbital cellulitis may follow dental extraction, particularly in individuals who are acidotic and diabetic. I notice that in your recommendations for detecting possible causative organisms you included various forms of culture but do not suggest biopsy. I would like to emphasize that phycomycotic infection is rarely detectable by culture alone. A biopsy of the infected tissue has to be taken before these organisms can be shown. I wonder if you would consider adding biopsy to your list of things that might be done to diagnose the infectious agent?

Dr John Bullock. I really appreciate everyone's comments. Doctor Waller mentioned ultrasound *vs* computed tomography (CT). I have had no experience with ultrasound in these cases, I have used only CT. He also mentioned drainage externally or internally into the nasopharynx, and I have no experience with internal drainage. I've approached them all externally as I have shown. Doctor Apt questions: "Why does this occur; there is so much infection in dental extractions, so why don't more patients get this." In my four cases I think that the number one answer is delay in diagnosis and treatment. All these patients were neglected both by the dentist and by the originally treating pediatricians and ear, nose, and throat doctors, and I think that the infections rapidly got out ot hand. I think it emphasizes the importance of early, proper management. Doctor Blodi mentioned, "how does one decide when to do surgical drainage." I see a lot of cases of orbital cellulitis and my feeling is that most of them do not need surgical drainage. Most of them just need antibiotic therapy.; I think it is wrong to rush to surgery and I think most of these patients should be managed for at least 24 hours

with massive doses of appropriate antibiotics and then if the patient is worsening, in spite of antibiotic therapy, I think drainage should then be done. I appreciate Doctor Jarrett's comments and in terms of Doctor Burde's comment about antibiotics, the list I showed included both aerobic and anaerobic organisms. In Doctor Dan Jones' chapter in the Duane series, he mentioned that the treatment of choice for anaerobic infections is massive doses of penicillin G.