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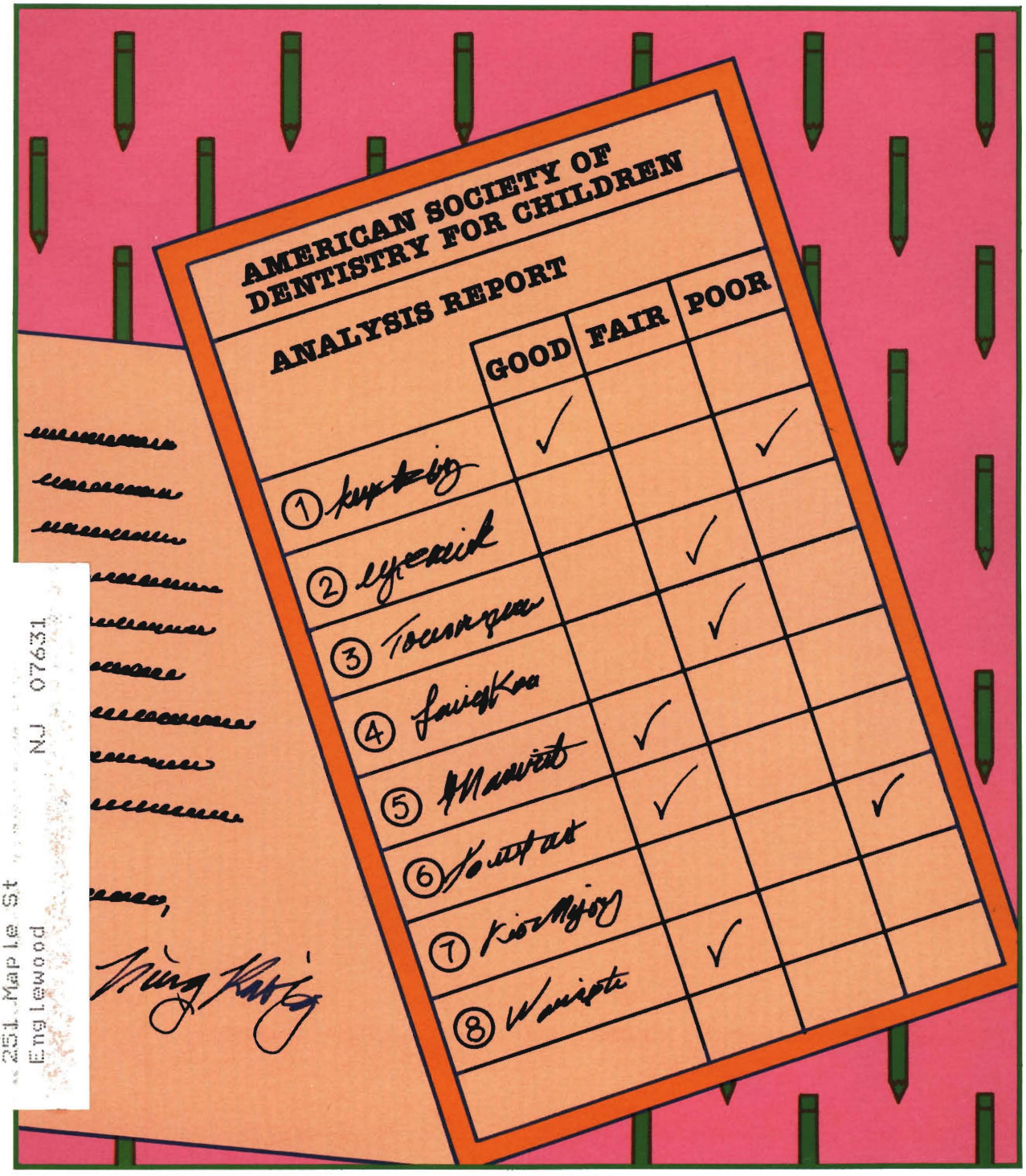
JANUARY-FEBRUARY 1985

JOURNAL OF DENTISTRY FOR CHILDREN



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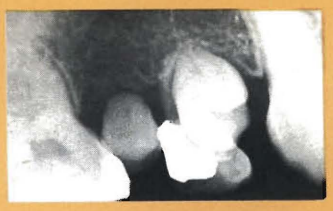

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JOURNAL OF DENTISTRY FOR CHILDREN

Volume 52 Number 1 January-February 1985

 Issued bimonthly—in January-February, March-April, May-June, July-August, September-October, and November-December—at 211 E. Chicago Ave., Suite 920, Chicago, Ill. Second class postage paid at Chicago, IL and additional mailing office. Subscription prices: within U.S.A. $40.00 per volume; Foreign (including Canada and Mexico) $50.00 per volume; to members of the ASDC $20.50 per volume (six issues per volume). Single copies $10.00. Member—American Association of Dental Editors.

All copy and manuscripts for the journal should be sent directly to the Editorial Office, 730 Blaney Drive, Dyer, Indiana 46311.

Prospective authors should consult "Information for Authors," which appears in the January and July issues. Reprints may be obtained from the Editorial Office.

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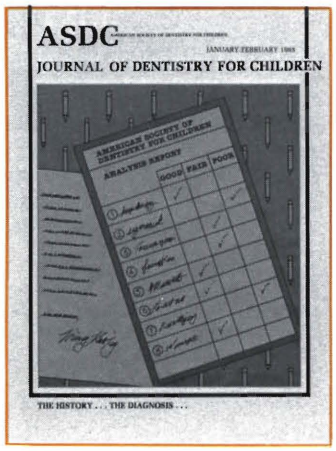
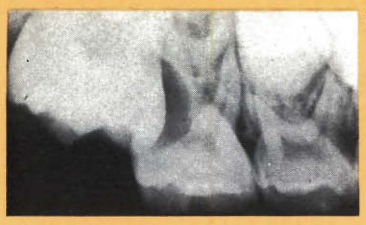
USPS #279-480



THE COVER
The cover symbolizes the report of Wells International, who conducted a survey of the entire structure, operation, and objectives of ASDC, during the last six months. The report in its entirety is printed in this issue.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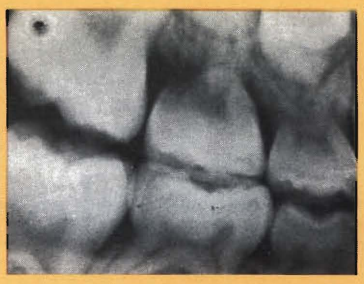
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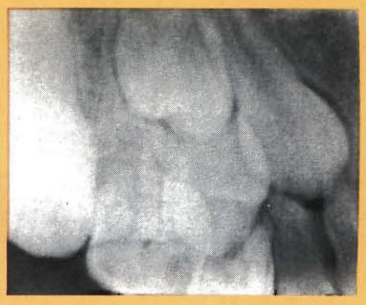
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CASE REPORTS

NUTRITION

Assessment of the autistic patient's dental needs and ability to undergo dental examination

Behavior

Oariona Lowe, DDS
Robert Lindemann, DDS, MAEd

Autism represents one of the most bizarre, severely incapacitating developmental disabilities diagnosed today. Autism is a very complex disorder of language, socialization, and perceptual motor difficulties.[1] The term autism was first used by Bleuler, in 1911, to label a particular withdrawal behavior disorder observed in schizophrenic patients.[2] Kanner, however, first described the syndrome of autism, in 1943, defining three prominent characteristics associated with it.[3] The pathognomonic feature was extreme aloneness and the child's inability to relate to people in the ordinary way. The second characteristic was an anxiously obsessive desire for the preservation of sameness, thereby restricting the arena for spontaneous activity. Kanner's third feature was failure to use language for purposes of communication. The autistic child suffers primarily from an impairment of his cognitive and perceptual functions; the consequence is a limited ability to understand and communicate, and to learn to participate in social relationships.[4]

A universally accepted definition of autism has not been established. Currently, the only way to diagnose autism is by observation of the child's behavior. The syndrome of autism is, therefore, behaviorally defined.

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Ritvo and Freeman defined the autistic syndrome for the National Society of Autistic Children as follows:

- ☐ Onset prior to thirty months of age.
- ☐ Disturbances in the developmental rate of the appearance of physical, social, and language skills.
- ☐ Abnormal responses to sensations.
- ☐ Speech and language are absent or delayed.
- ☐ Abnormal ways of relating to people, events, and objects.[4]

Rutter and Barak, in 1971, outlined four basic criteria which they felt must be met before the diagnosis of autism is made.[5] These are:

- ☐ Failure to develop interpersonal relationships.
- ☐ Delay in speech and language development.
- ☐ Ritualistic, stereotypic and compulsive phenomena.
- ☐ Onset before the age of thirty months.

The failure to achieve a universal common definition has added to difficulties in assessment of autistic children.[6] Diagnosis of the syndrome before the age of thirty months is necessary for two reasons. Ongoing studies of autistic children show that their behavior patterns tend to change markedly as they grow older. There is a lack of attachment and a relative failure of bonding that is most marked in the first five years. Autistic children do not display the parental attention-seeking and clinging that normal children show. After age five, many of the social impairments that have resulted from the child's lack of cooperative group-play, his failure to make personal relationships, and his lack of empathy and failure to perceive other people's feelings and responses become evident. Autistic children seldom play cooperatively and their play behavior, even in isolation, tends to be abnormal or repetitive, and does not invite participation by other children.[7] The pervasiveness of speech and language deficits in autistic children tends to isolate them further from meaningful social contacts. The same is true for the stereotyped, repetitive, self-stimulative behavior of some of these children. The speech disorder involves the basic process of language development. Autistic speech can be characterized as repetitious, echolalic, or parrotlike with pronominal reversal. Failure to use language is a prominent part of the autistic syndrome.

Autistic children show a great irregularity in the age at which the sequential motor and mental development progresses. The strange and bizarre behavior of autistic children is due in part to their peculiar mobility patterns. The deviant mobility may involve the hands, lower extremities, trunk, or the entire body. Self-stimulation, repetitive play, and sitting for hours staring into space are behaviors frequently described.[8] They may also display self-injurious behavior, such as extremity biting, scratching, or face-slapping.[9]

	Autistics	Nonautistics
Male	15	13
Female	5	7
Age range	3-30 years	3-30 years
Institutionalized	11	4
Noninstitutionalized	9	16
Speech		
Verbal	12	19
Nonverbal	8	1

Figure. Patient characteristics of the study population.

Furthermore, responses to Rimland's diagnostic questions for autism indicated that after 5.5 years of age, the child's behavior pattern tended to become more diffuse.[10]

The perceptual disturbance characteristic of many autistics is manifested by either a lack of responsiveness or an exaggerated reaction to sensory stimuli (visual, auditory, olfactory, vestibular, and proprioceptive). They may show both a heightened awareness and sensitivity to sensory stimuli, such as preoccupation with spinning objects, overresponse or nonresponse to varying levels of sound, and attention to inanimate objects by repetitive sniffing or licking.

It has been suggested that autism is the earliest development of psychosis and may be an early manifestation of schizophrenia. The age of onset of childhood psychoses follows a markedly bipolar distribution with one peak in infancy and another in adolescence.[11] Autism correlates with the former peak, childhood schizophrenia with the latter. In addition, delusions and hallucinations are characteristic of schizophrenic children, but rarely are seen in autistic individuals.[12]

When autism was first recognized as a disease entity, attempts were made to differentiate it from mental retardation. The physical characteristics of autistics including their "normal" faces suggested the presence of intelligence, yet markedly retarded. Studies have shown, however, that, unlike mentally retarded children, autistic children have a particular cognitive deficit that involves language and central coding processes. It has been found though, that autism may coexist with mental retardation.

Swallow reported that dental care for the autistic child can be an exhausting experience for the dentist.[13] When given the behavioral traits of autism, this is readily understood. It requires constant repetition and patience in dealing with bizarre behavioral patterns; hence, treatment of the autistic child is rarely undertaken by the general dentist. The problems of treatment include lack of cooperation, and inappropriate patient/dentist

interaction. In a previous study, Braff indicated that the autistic patient can be treated, if adequately sedated.[14] This generalization, however, has not been made from a carefully controlled study of autistic patients. Rutter reported that autistic children become disturbed by dental treatment and may cause the dentist stress by their actions. There is very little information in the dental literature concerning treatment of the autistic patient, however, and reference to the dental manifestations and needs of these patients has been for the most part, anecdotal. Reaction to dental appointments and the oral health status of these patients have yet to be identified and quantified.

The bizarre behavioral manifestations of the autistic syndrome and the common reasons why dental needs of handicapped persons are not met, has stimulated the authors' interest in studying the autistic population.

This study was undertaken to:

- ☐ Determine the ease of performing a dental examination on autistic patients on the first appointment.
- ☐ Evaluate the dental needs of these patients.
- ☐ Determine whether utilization of certain behavior management tools could be successfully used on the autistic patient, to accomplish an initial oral examination and to make acceptable bite-wing radiographs.

Our hypothesis predicts that only a small percentage of autistic patients will allow an initial oral examination, including bite-wing radiographs.

MATERIALS AND METHODS

Forty patients were randomly selected for this study from patients attending the UCLA Pediatric Dental Clinic. Twenty autistic patients and twenty nonautistic age-matched controls were selected, ranging in age from three to thirty years (12.5 years was the mean age for both groups) (Figure). The autistic patients all had a medical diagnosis of autism, were either institutionalized or living at home, and showed a wide range of verbal ability. The nonautistic population consisted of two mentally retarded, one cerebral palsy, two schizophrenics, and fifteen normal patients (patients with no medical handicaps).

In an attempt to standardize the examination procedure, the Chief Pediatric Dental Resident made the oral examinations and bite-wing radiographs for each subject. All patients were seen on an outpatient basis, the medical and social history was reviewed, and a clinical examination performed. The criteria for a successful clinical examination included:

- ☐ Complete and thorough oral examination, which included intraoral evaluation of the condition of the gingiva; detection of any caries lesions as confirmed with a dental explorer; examination of the floor of the mouth, soft and hard palate, tongue, and posterior tonsillar areas; and extraoral evaluation of the facial structures.
- ☐ The second criterion for a successful clinical examination was to obtain clinically acceptable bite-wing radiographs where interproximal caries could be detected.

All forty patients were examined using common behavior management tools for the clinical-examination phase of this study. The behavior management tools used were positive reinforcement and the technique of tell, show, and do. Negative reinforcement was used if positive reinforcement was unsuccessful. In addition, the effect of familiarization with the dental environment was evaluated by observing the patient over a number of visits.

Standard techniques of positive reinforcement included praising the patient for his accepted behaviors, either orally ("good boy/girl"), or physically (rubbing his/her hands or face, or patting the shoulder or back), or by telling the patient that he will get a reward (food) after the examination or procedure is completed. Negative reinforcement was supplied by removing a stimulus to increase the frequency or intensity of a behavior (e.g. "you won't get lunch if you don't open your mouth, or you won't get to go home unless you let me finish the procedure"). Negative reinforcement was used only if positive reinforcement was unsuccessful. The technique of tell, show, and do is a common modeling and rehearsal procedure of telling the patient what is going to happen, what he will feel, or do, showing the child what should be done, and then attempting the procedure.[15,16]

In addition to the routine oral examination, the oral hygiene, plaque, and caries index for each patient were observed. The Klein and Palmer Caries Index was used to record the decayed, missing and filled permanent and primary teeth.[17,18] The Greene and Vermillion OHI-S Index was used to score debris and calculus.[15] Plaque was scored by the Silnes and Löe Plaque Index.[19]

Modifications of the Simplified Greene and Vermillion Oral Hygiene Index were made by the author to include the primary dentition for debris and calculus. Facial surfaces of the maxillary right and left second primary molars, facial surfaces of the maxillary right primary central incisor, the lingual surfaces of the man-

Table 1 ☐ Comparison of oral and bite-wing examination on autistic and nonautistic patients.

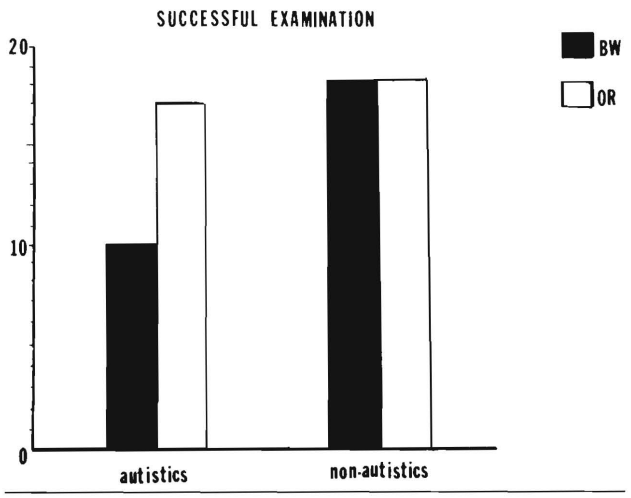

Table 2 ☐ Def of autistic and nonautistic patients on inital examination.

def (KLEIN & PALMER)

AUTISTIC	N	MEAN	SEM	P
NO	10	5.0	1.70	< 0.05
YES	10	0.87	0.61	

T_0 = BASELINE

Table 3 ☐ Def of autistic and nonautistic patients on recall examination.

AUTISTIC	N	MEAN	SEM	P
NO	10	5.6	1.82	
YES	10	1.37	1.10	N.S.

T_1 = recall

$p < 0.05$

dibular right and left primary central incisor were evaluated for soft debris or stain. Debris was scored from 0 to 3 using the following criteria:

0 = No debris or stain.
1 = Soft debris covering not more than one third of the tooth surface, or the presence of extrinsic stains without other debris regardless of the surface covered.
2 = Soft debris covering more than one third, but not more than two thirds of the exposed tooth surfaces.
3 = Soft debris covering more than two thirds of the exposed tooth surface.

Calculus was scored similarly for both permanent and primary teeth:

0 = No calculus present.
1 = Supragingival calculus covering not more than one third of the exposed tooth surface.
2 = Supragingival calculus covering more than one third, but not more than two thirds of the exposed tooth surface or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth or both.
3 = Supragingival calculus covering more than two thirds of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portions of the tooth or both.

Using the Silnes and Löe Plaque Index, the clinical presence of plaque was noted and classified according to the following criteria:

0 = No plaque in the gingival area.
1 = A film adhering to the free gingival margin and adjacent area of tooth. Plaque was identified by running a probe across the tooth surface.
2 = Moderate accumulation of soft debris within the gingival pocket on the gingival margin and/or adjacent tooth surface which can be seen by the naked eye.
3 = Abundance of soft matter within the gingival pocket and/or gingival margin and adjacent tooth surface.

For this index the teeth in the left upper quadrant of the mouth were examined. The mesial, lingual, buccal and distal surfaces of numbers 9, 10, 11, 12, 13, and 14 or correspondingly, F, G, H, I, and J were scored. The scores for the various areas of each tooth were added together and divided by 4 to determine the plaque index for the tooth; then totaling all the indices and dividing by the number of teeth in the quadrant, the plaque index was established.

The oral hygiene status, as derived from the combined score of the OHI-S and PI.I, was assessed and used to compare the autistics to the controls. Within the autistic group, the noninstitutionalized and institutionalized patients were also compared.

When the oral examination was completed, bite-wing radiographs were attempted, to aid in the identification of progressive caries lesions. Bite-wing examinations were performed using the standard paralleling radiographic technique and bite-wing tabs.

Table 4 ☐ DMF of autistic and nonautistic patients on initial examination.

DMF (KLEIN & PALMER)

AUTISTIC	N	MEAN	SEM	P
NO	10	3.0	1.68	
YES	10	4.3	1.44	N.S.

T_0 = BASELINE

Table 5 ☐ DMF of autistic and nonautistic patients on recall examination.

AUTISTIC	N	MEAN	SEM	P
NO	10	3.0	1.68	
YES	10	4.8	1.72	N.S.

T_1 = recall

$p < 0.05$

Table 6 ☐ Oral hygiene index indicating the presence of more debris and calculus in the autistic population.

OHI - S (GREENE & VERMILLION)

AUTISTIC	N	MEAN	S.D.
NO	20	1.06	0.90
YES	20	1.56	1.33

Table 7 ☐ Plaque index mean showing an increased amount of plaque found in the autistic group.

PLAQUE INDEX (SILNESS & LÖE)

AUTISTIC	N	MEAN	S.D.
NO	20	0.59	0.31
YES	20	0.81	0.60

RESULTS

It was demonstrated that a successful oral examination was obtained on seventeen of the twenty (85 percent) of the autistic patients examined on the first appointment (Table 1). In the nonautistic population, successful oral examinations were achieved on eighteen of the twenty patients (90 percent) examined on the first dental visit (Table 1). Negative reinforcement had to be used on eight of the autistic patients.

In the nonautistic population, the management tools used on the patients were primarily positive reinforcement and tell, show, and do, as in the autistic population. Negative reinforcement was used on only two of these patients.

For the bite-wing examination, clinically acceptable bite-wing radiographs were obtained on ten of the twenty (50 percent) autistic patients (Table 1). Again, the behavior management tools of positive reinforcement, tell, show, and do were used on all the patients; negative reinforcement was used on ten of these patients for successful radiographs. The other ten patients, of whom radiographs were not obtained, were rescheduled and other means of controlling their behavior were used.

For the nonautistic population, a clinically acceptable bite-wing radiograph was obtained on 18 (90 percent) of the 20 patients examined. Negative reinforcement was used on only two of these patients.

When comparing the caries index in patients with primary dentition (def), it was found that on initial examination, the autistic patients demonstrated a significantly higher caries index than the ten control patients (Table 2). On recall examination, no significant difference was demonstrated in either group (Table 3). The caries index in patients with permanent dentition (DMF) showed both the autistic group and control group to be comparable. The DMF, both at baseline and recall, was not found to be statistically significant at $p < .05$ (Tables 4 and 5, respectively).

Table 6 represents the oral hygiene index. The autistic group demonstrated a higher OHI-S mean than the nonautistic group, indicating the presence of more debris and calculus. The OHI-S score, although higher, was not found to be statistically significant at $p < .05$. Table 7 shows the plaque index values. A higher plaque mean was also noted with the autistic group, indicating the presence of more plaque deposition. Again, although this score was higher than the nonautistic group, the difference was not significant. It was also observed that the oral hygiene status (OHI-S plus P1.I) was better in the noninstitutionalized autistic, although not statistically significant. The noninstitutionalized autis-

tics were younger patients (mean age 6.7 years, noninstitutionalized versus 18.2 years, institutionalized).

DISCUSSION

This study shows that:

- ☐ Successfful initial oral examination and bite-wing radiographs were achieved on 50 percent of the autistic patients examined.
- ☐ The autistic patients revealed a lower oral hygiene status than the controls, but a comparable caries index.
- ☐ Successful management of these patients in the dental environment includes reinforcing positive behaviors, utilizing the technique of tell, show, and do, and using negative reinforcers when necessary.

In performing the bite-wing examination, one of the major problems in obtaining adequate radiographs is in preventing patient movement during the x-ray exposure. This is especially true with the autistics, because many of these patients were unable to attend to directions and many would not sit still. The use of negative reinforcers aided in the success of obtaining a clinically acceptable bite-wing radiograph. Also, by showing the patient what is expected of him (modeling and tell, show, and do) and in some instances holding his mouth closed, acceptable radiographs were obtained. This was also true of the nonautistic patient.

It must be emphasized, then, that the techniques commonly used in treating nonautistic patients could be utilized in treating the autistic patient. The dentist, however, should be prepared to employ a wide variety of approaches and modifications of examination in a manner flexible enough to shift with changing patient needs.

It was observed that the oral hygiene status of the autistic patient was lower than the nonautistic. This may be due to the fact that autistic patients cannot motorically accomplish toothbrushing, and they do not understand the importance of oral hygiene. More emphasis on training should be directed to assist the autistic patient in maintaining an acceptable level of oral hygiene. It was also demonstrated that the oral hygiene level was better in the noninstitutionalized, younger autistic patient. This may be due to the fact that the younger patient, living at home, receives more individualized attention than the older institutionalized autistic. This includes helping the child to brush his teeth. The younger child is also easier to manage than the older patient. The younger patient is physically smaller and easier to restrain. The older patient, due to his large size, is more difficult to restrain physically and may have developed defenses.

The syndrome of autism involves multiple perceptual and cognitive impairments that lead to the development of extreme behavior maladjustments. When considering management of the autistic child's problem behavior, it is essential to bring this issue of bizarre behaviors into realistic focus. The behavior of the autistic patient may be very difficult to understand and there is a great variability in the syndrome and in the abnormal behavior exemplified. Establishing communication between a dentist and the autistic patient is undoubtedly the most dominant factor in successful treatment. It is emphasized that the role of communication between patient and dentist becomes more evident when hindered by some form of handicap. Communicating with the autistic patients in this study was extremely difficult.

Recent studies report that there is a growing interest on the part of the dental profession in treating the patient with a disability. A strong correlation between experience and willingness to accept the autistic patient provided strong support for continuing educational programs on treatment of these patients.

CONCLUSION

Dental treatment of the handicapped patient is very challenging. The ability to manage an autistic patient in the dental environment for an initial oral examination and bite-wing radiographs has been discussed. Although behavior management tools have been used successfully, these patients may need to be evaluated for sedation or general anesthesia for dental treatment. The dental needs identified for autistic patients in this study are limited to the population investigated. Further studies are suggested to quantify these needs.

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HYDROXYZINE

Hydroxyzine may be of some value for children with cerebral palsy because of its reported effect of decreasing skeletal motor activity. The author has found, however, that another medication (diazepam) is more effective for these children. Another personal observation is that hydroxyzine seems to be beneficial in treating hyperactive and autistic children. Unfortunately, there have been no reports to-date on the usage of this medication for these children.

In general, therefore, it can be said that hydroxyzine is most useful in alleviating manifestations of anxiety and tension in situations wherein the causative stress is a temporary one, such as the dental situation. Hydroxyzine is recommended as the initial medication, often as a preventive medication, in a program of preoperative sedation for the apprehensive child. The dosages can be modified at the next dental appointment if the drug is not entirely successful after the first trial, or a different anxiety agent may be employed at the discretion of the practitioner.

This discussion brings into focus the expectations that one has for a particular medication. If a child has been uncooperative at the time of injection and fidgety or perhaps whimpering throughout the restorative procedure, can one expect the medication to combat or control all of these problems? Hydroxyzine in adequate dosages may or may not overcome uncooperativeness at the time of injection, but it should minimize the problem in the child's mind. The medication should combat whimpering or fidgeting during restorative procedures. The patient should assume a quiescent state. If this expectation is not fulfilled, then the premedication cannot be considered a success.

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Clinic

A survey of 200 pediatric dental general anesthesia cases

Daniel John Enger, DDS
Arthur P. Mourino, DDS, MSD

INTRODUCTION AND REVIEW OF THE LITERATURE

Management of the child is a major component in children's dentistry. The majority of children can be adequately treated with behavior modification techniques such as Show, Tell and Do. Some children might require nitrous oxide analgesia and sedation to complete their treatment. Some children require hospitalization to complete their treatment. These are children who have extensive dental decay, are management problems, or are medically compromised.[1-6]

Complete oral rehabilitation for children with the aid of general anesthesia is an accepted procedure in most hospitals. Although the administration of a general anesthetic is relatively safe, complications can occur. Life-threatening complications, such as allergic reactions and bronchospasms, usually occur when the anesthetic is administered. Non-life-threatening complications are usually manifested later, even though they may have been initiated during the administration of the anes-

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thetic.[7] This study will evaluate complications which occur most often after the administration of a general anesthetic: nausea with vomiting, fever (oral temperature above 101° F degrees), sore throat and pharyngitis, lip swelling, and delayed or prolonged recovery.

Nausea with vomiting may be caused by the swallowing of blood from extraction sites or from individual differences in postoperative drug metabolism. The highly nervous, apprehensive child who expects to be sick afterwards because of a previous anesthetic experience or tales told to him by family or friends might also be prone to nausea with vomiting.[8]

An increased temperature may be caused by tissue trauma; bacteremia; drugs, such as atropine sulfate; anesthetic depression of tracheobronchial cilia promoting stasis of secretions and resulting in the release of leukocyte toxins that raise the setting of the hypothalmic thermostat; pulmonary infection; atelectasis; hot lights in the operating room with heavy draping and poor ventilation around the patient; and dehydration.[2] The body temperature of a patient usually decreases during anesthesia, mostly because of the air-conditioned rooms. During prolonged anesthesia, the temperature of the patient will increase. A young child will become dehydrated, if he has been fasting for eight hours. This will result in an elevated temperature. A temperature rise in a young child will increase the chances for vomiting and convulsions.[3]

Sore throat and pharyngitis are common occurrences during the postoperative period. These complications can be due to several factors: traumatic intubation, with the blade of the laryngoscope cutting or irritating the pharyngeal wall; prolonged coughing on an endotracheal tube or an airway; using an endotracheal tube that is too large or overinflating the cuff, causing damage to the pharyngeal wall and adenoidal tissue; nonsterile airways and tubes and vigorous pharyngeal suctioning during anesthesia or during the postoperative period.[9]

Postoperative swelling may be due to soft tissue trauma, infection, or allergic reaction.[10] The swelling may be immediate or delayed in onset, and treatment will depend on the etiologic factors involved.

Delayed or prolonged recovery is infrequent, but by no means nonexistent. Recovery may be prolonged, if heavy premedication or anesthesia has been used. Delayed recovery may also be attributed to hypotension or hypoxia. Some patients physiologically eliminate anesthetics slowly which may also lead to delayed recovery time.[8]

Nasotracheal intubation is often the method of choice for dental surgery. It is better tolerated than the oral tube during the recovery period, and does not restrict the operating field during dental surgery. A major disadvantage is that it can cause trauma and dislodgement of adenoidal tissue. The dislodged tissue could be carried into the trachea. Damage to the adenoidal tissue can increase the risk of postoperative infection, bleeding and sore throat.[4,10,11]

Medications are used preoperatively primarily to sedate the patient, afford a smooth induction and prevent secretion and vagal activity.[12] The choice and dosage of preanesthetic drugs depend upon the anesthesiologist's goals.[12] The most common anesthetics used in pediatric dental general anesthesia include halothane (fluorthane) and ethrane (enflurane).[13] Halothane was introduced in the late 1950's and has become widely used and accepted as a reliable anesthetic agent.[8,13-15] It is nonflammable, nonirritating, and a potent anesthetic agent.[8,13,15] Since it is relatively insoluble, the induction is rapid compared to more soluble agents such as ether and methoxyflurane.[8,13-15] Other advantages include a pleasant fruity odor, moderate muscle relaxation and a smooth rapid recovery.[8,13-15] It is, however, a respiratory and cardiac depressant, so the depth of anesthesia must be carefully monitored by the anesthesiologist.[8,13-15] Repeated exposure of the patient to halothane is contraindicated due to the possibility of liver damage.[8,13,14] It has also been reported that the repeated administration of halothane can increase nausea with vomiting during the recovery period.[8,13,14]

Ethrane was first introduced clinically in the early 1960's, and released for general use in 1972.[8,13,14] It is a stable, nonflammable liquid, and somewhat less volatile than halothane. Ethrane produces a rapid onset of anesthesia and a rapid recovery.[8,13,14] Ventilatory depression parallels anesthetic depth, as does muscle relaxation.[8,13,14] Ethrane is metabolized in the liver and produces free serum fluoride at levels far lower than methoxyflurane. A contraindication to ethrane is a patient with renal failure.[8,13-15] There is also the possibility of central nervous system excitation or seizure activity, when using this anesthetic.

The present study was designed to report the results of 200 cases of complete oral rehabilitation. The patient's preoperative physical status, the role of anesthetics, length of surgery, and traumatic intubation were evaluated to determine whether a relationship exists between these variables and postoperative complications. This study also evaluated whether there was a

Table 1 ☐ Sex, race and age of patients at Richmond Eye and Ear and Children's Hospital.

		Richmond Eye and Ear Hospital			Children's Hospital			Total
Sex	Race	No.	Age	(years)	No.	Age	(years)	Av. age
			Mean	Range		Mean	Range	
Male	Black	22	9.4	1-32	22	7.2	2-18	8.6
	White	33	10.0	1-44	29	7.7	2-17	
Female	Black	18	7.5	2-19	24	7.0	2-17	6.9
	White	27	7.0	2-52	25	6.4	2-20	

Table 2 ☐ ASA specifications for Richmond Eye and Ear (REEH) and Children's Hospital (CH).

	ASA specification	REEH No. of cases	CH	Total No.	Percentage
1	Normal healthy patient	64	44	108	54.0
2	Patient with mild systemic disease	28	51	79	39.5
3	Patient with severe systemic disease but not incapacitating	8	5	13	6.5
4	Patient with an incapacitating disease that is a threat to life	0	0	0	0
5	Patient not expected to survive without operation	0	0	0	0

relationship between the distance traveled and the method of payment for treatment with the patient's return for a six-month recall visit.

METHOD AND MATERIALS

Two hundred patients were included in this study. General anesthesia was administered at two hospitals in Richmond, Virginia: the Richmond Eye and Ear Hospital (REEH), a private hospital, and Children's Hospital (CH), a private teaching hospital. At Richmond Eye and Ear Hospital, anesthesiologists administered the general anesthetic and were primarily responsible for the patient. In cases done at Children's Hospital, nurse anesthetists from the Department of Anesthesiology at the Medical College of Virginia administered the anesthetics and were primarily responsible for the patient. A hundred patients were treated at each hospital, during a five-year period from January 1977 through November 1982. Dental procedures at Richmond Eye and Ear and at Children's Hospital were performed by pedodontists and postgraduate students.

The patient's physical status was described according to the American Society of Anesthesia (ASA) specifications:

- ☐ A normal healthy patient.
- ☐ A patient with mild systemic disease.
- ☐ A patient with a severe systemic disease that limits activity, but is not incapacitating.
- ☐ A patient with an incapacitating systemic disease that is a constant threat to life.
- ☐ A patient not expected to survive twenty-four hours without the operation.

Routine laboratory tests, consisting of a complete blood count, SMAC, PT, PTT, and urinalysis, were ordered for all patients. A physical examination by a pediatrician (Richmond Eye and Ear Hospital) or pediatric resident (Children's Hospital) was done prior to surgery.

On the morning of surgery, an appropriate preoperative medication was administered a half hour to an hour before surgery. An IV was started when possible and general anesthesia was induced. In instances when an IV could not be started, general anesthesia was induced with a mask. Nasotracheal intubation was done on all 200 patients using either cuffed or uncuffed tubes. The intubation was registered as being traumatic or atraumatic. Intubations were considered atraumatic if they were accomplished in two or fewer attempts. Following intubation, a general anesthetic, either halothane or ethrane, was given in combination with nitrous oxide and oxygen. The patient was draped and a gauze throat pack was placed to occlude the pharynx. Prophylaxis and topical fluoride treatments were performed and rubber dams were placed on all teeth requiring restorations. When the dental treatment was completed the oral cavity was thoroughly cleansed and the posterior pharynx and mouth suctioned prior to extubation. Patients were sent to the recovery room breathing on their own and when stable, transferred to their hospital room. The patients were discharged, usually on the following day when they were alert, ambulatory and with stable vital signs.

Complications occurring postoperatively were considered, if they occurred from the time of admission to the recovery room until the patient was discharged. In this study, a Chi Square test was used to compare postoperative complications versus:

- ☐ Preoperative physical status.
- ☐ Trauma introduced during surgery.
- ☐ Anesthetics used.

A Chi Square Test was also used in comparing patients returning for recall versus distance traveled and type of payment received. Finally, an unpaired t-test was used to compare complications versus length of surgery.

Table 3 ☐ Reasons for admission: Richmond Eye and Ear Hospital (REEH) and Children's Hospital (CH).

Patients may have been admitted for more than one reason.

Reasons:	REEH Number	CH Number	Total Number
1. Rampant caries (etiology not bottle) syndrome)	55	47	102
2. Rampant caries (etiology bottle syndrome)	25	46	71
3. Management problem	67	37	104
4. Medically compromised child	31	51	82
5. Family lives far from source of care	3	3	6
6. Extensive treatment	3	0	3
7. Other	0	5	5

Table 4 ☐ The dental procedures rendered under general anesthesia.

	REEH	CH	Total
Amalgams	506	471	977
Extractions	383	362	745
Stainless steel crowns	347	252	599
Composite resins	147	99	246
Formocresol pulpotomies	78	64	142
Gutta percha root canals	7	5	12
Gingival surgery	5	5	10
Space maintainers	5	3	8
Scaling and root planing	5	2	7
Zinc oxide eugenol pulpectomies	4	0	4
Frenectomies	2	0	2

RESULTS

Two hundred patients ranging in age from one year to fifty-two years were included in this study (Table 1). The average age of all males treated at both hospitals was 8.6 and for females was 6.9. There were 106 males and 94 females, of which 86 were black and 114 were Caucasian.

The ASA specifications are listed in Table 2. Of the 200 patients, 108 (54 percent) were in ASA 1, seventy-nine (39.5 percent) were in ASA 2, and thirteen (6.5 percent) were in ASA 3. Richmond Eye and Ear Hospital had sixty-four in ASA 1, and twenty-eight in ASA 2, and eight in ASA 3. Children's Hospital had forty-four in ASA 1, fifty-one in ASA 2 and five in ASA 3. There was no statistically significant relationship between the patient's preoperative physical status and the complications occurring postoperatively.

Reasons for admission are listed in Table 3. The majority of the patients had more than one reason for hospitalization. The most common reasons for admission were: rampant caries (etiology—nutrition and lack of oral hygiene of nursing bottle caries), management problems, and medically compromised children requiring hospitalization.

Of the 200 cases, 190 (95 percent) were atraumatic intubations; and ten (5 percent), traumatic. Of the traumatic cases four were in the one to three-year age-group and six were in the four-to-six-year age-group. There was no significance in the trauma introduced during intubation versus postoperative complications.

At Richmond Eye and Ear, seventy-four (74 percent) patients were given halothane and twenty-six (26 percent) patients were given ethrane. At Children's Hospital eighty-two (82 percent) were given halothane and eighteen (18 percent) patients were given ethrane. For the 200 patients, halothane was given in 156 (78 percent) of the patients and ethrane in forty-four (22 percent) of the patients. There was no significance observed, when comparing anesthetics used versus postoperative complications.

Table 4 shows the dental procedures accomplished for the 200 children while under general anesthesia. The most frequent dental procedures completed were amalgams, extractions, stainless steel crowns, composite resins and formocresol pulpotomies.

Table 5 shows the average time to complete the dental treatment, average time in the recovery room and the average number of days in the hospital. At Richmond Eye and Ear Hospital, the average length of surgery was 209 minutes (three hours and twenty-nine minutes), the average number of minutes in the recovery room was forty-three minutes and the average number of days in the hospital was 2.04. At Children's Hospital, the average length of surgery was 179 minutes (two hours and fifty-nine minutes), the average time in the recovery room was sixty-three minutes and the average number of days in the hospital was 2.18. There was no significance in comparing length of surgery versus postoperative complications.

Table 6 lists the complications which occurred postoperatively. Nausea with vomiting was the most com-

Table 5 ☐ The average time to complete dental treatment, the average time in the recovery room, and the average days in the hospital for Richmond Eye and Ear Hospital (REEH) and Children's Hospital (CH).

	Av. Time of surgery	Range	Av. time in recovery room	Range	Av. days in hospital	Range
REEH	209 min	55-360 min	43 min	15-130 min	2.04	1-5
CH	179 min	75-280 min	63 min	30-230 min	2.18	1-11

Table 6 ☐ Complications at Richmond Eye and Ear Hospital (REEH) and Children's Hospital (CH).
Patient may have more than one complication postoperatively.

	REEH	Day of surgery	Day after surgery	CH	Day of surgery	Day after surgery
Nausea with vomiting	41	39	2	30	27	3
Increased oral temp. (above 101°)	8	8	0	15	13	2
Cough, sore throat	7	6	1	8	8	0
Bleeding	2	2	0	6	6	0
Lower lip swelling	2	1	1	1	1	0
Delayed recovery	2	2	0	1	1	0
Total	62	58	4	61	56	5

Table 7 ☐ Distribution by age of patients and complications at Richmond Eye and Ear Hospital (REEH) and Children's Hospital (CH).

	REEH				CH			
Age	Patients		Complications		Complications		Total	%
	No.	%	No.	%	No.	%		
1-5	107	53.5	33	60.0	24	44.4	57	52.3
6-10	36	18.0	7	12.7	18	33.3	25	23.0
11-15	29	14.5	6	10.9	7	13.0	13	11.9
16-20	16	8.0	3	5.5	5	9.3	8	7.3
21-25	6	3.0	5	9.1	0	0	5	4.6
26 and older	6	3.0	1	1.8	0	0	1	.9
Total	200	100	55	100	54	100	109	100

mon complication in seventy-one of 200 patients (35.5 percent). This was followed by increased oral temperature in twenty-three of 200 patients (11.5 percent), and cough and sore throat in fifteen of 200 patients (7.5 percent). On the day of surgery, 114 of 123 (92.7 percent) complications occurred, while nine of 123 (7.3 percent) complications occurred on the day following surgery. (Patients may have had more than one complication occurring postoperatively). There were no deaths.

Table 7 lists the number of patients by age with complications. Some type of complication was seen postoperatively in 109 (54.5 percent) of the 200 patients. The majority of the postoperative complications occurred in the age-range of one to five years (52.3 percent) followed by the age-range six to ten years (23 percent).

Table 8 compares the patients returning for a six-month recall versus the distance traveled. One hundred and four patients (52 percent) received routine follow-up care. For the 200 patients, fifty-three of eighty-two (65 percent) in the city of Richmond returned for recall, and fifty-one of 118 (43 percent) outside of Richmond returned for recall. Patients living in the City of Richmond had a tendency to return for recall and this was significant at $p < .025$ for Richmond Eye and Ear and Children's Hospital.

Table 9 shows the type of payment received for treatment, versus the patients returning from recall. At Richmond Eye and Ear Hospital whether the patient returned for recall was not significant with the type of payment received. At Children's Hospital, medicaid and no-charge patients significantly ($p < .05$) returned for recall. For the 200 patients, sixty-three of 200 (31.5 percent) were Medicaid, seventy-five of 200 (37.5 percent) were private, forty-four of 200 (22 percent) were not charged, and eighteen of 200 (9 percent) were insurance. The patients returning for recall were: thirty-four of sixty-three (54 percent) of the Medicaid patients, thirty-four of seventy-five (45 percent) of the private patients, twenty-seven of forty-four (61 percent) of the patients not charged, and nine of eighteen (50 percent) of the insurance patients.

DISCUSSION

After general anesthesia for dental procedures, postoperative care must be provided. While most patients are ready for discharge on the same day, or on the morning after the surgery, those who develop postoperative complications such as fever, respiratory or cardiovascular complications should remain in the hospital for longer periods of observation.

The type of anesthesia, either halothane or ethrane, resulted in an acceptably low incidence of complications in our analysis. This is also attributed to the fact that the majority of cases were classified as ASA 1 or ASA 2. These are the normal healthy patients and the patients with mild systemic disease.

The majority of postoperative problems were nausea with vomiting, fever, and sore throat in children under the age of five. A possible cause for the high incidence of fever might be due to dehydration, because IV fluids were discontinued in the recovery room due to the patient's agitation. Although these complications may not cause a lengthy stay in the hospital, they warrant the concern of the attending pedodontist. Prescribing anti-emetics for nausea with vomiting, ice chips for sore

Table 8 ☐ Comparison of patients returning for a six-month recall vs. distance traveled at Richmond Eye and Ear Hospital (REEH) and Children's Hospital (CH).

REEH	In City of Richmond	Outside of Richmond	Total
Returned for recall	22	25	47
Did not return for recall	12	41	53
Subtotal	34	66	100
CH			
Returned for recall	31	26	57
Did not return for recall	17	26	43
Subtotal	48	52	100
Total	82	118	200

Table 9 ☐ Comparison of the type of payment received for treatment versus the patients returning for recall at Richmond Eye and Ear Hospital (REEH) and Children's Hospital (CH).

	No Charge	Private	Medicaid	Insurance	Total
REEH					
Returned for recall	0	28	14	5	47
Did not return for recall	0	30	17	6	53
Subtotal	0	58	31	11	100
*CH					
Returned for recall	27	6	20	4	57
Did not return for recall	17	11	12	3	43
Subtotal	44	17	32	7	100
Total	44	75	63	18	200

*Policy at Children's Hospital required no payment for treatment of clinic patients who were unable to pay. This was in effect until April, 1982.

throat and Tylenol for fever postoperatively, might alleviate some of these complications.

There were two patients from Richmond Eye and Ear Hospital who stayed longer than two days. A six-year-old child with congenital heart disease stayed three days because of an elevated temperature, coughing, and a chest full of secretions, and a four-year-old with cerebral palsy stayed five days because of postoperative bleeding from extraction sites. At Children's Hospital, there were six patients who stayed longer than two days. Two eighteen-year-olds with cerebral palsy and seizure disorders stayed three days. One eighteen-year-old had a grand mal seizure, elevated temperature, and coughing, and the other eighteen-year-old had postoperative bleeding from extraction sites. A four-year-old with cerebral palsy and mental retardation, and a three-year-old with sickle cell anemia stayed four days. The four-year-old had a croupy cough and hoarseness and the three-year-old had an elevated temperature. A four-year-old with congenital heart disease stayed five days because of a croupy cough; and a twelve-year-old with seizures stayed eleven days because of his seizures. A neurological workup was done during this extended stay. More children were likely to have an increased stay at Children's Hospital, because the hospital caters to the child who is medically compromised.

Children's Hospital is an independent, nonprofit specialty hospital, serving children through age nineteen. On-site pediatric surgical and medical services include orthopedics, urology, plastic surgery, eye, ear, and dentistry. Prior to April of 1982, patients treated at Children's Hospital were not required to pay for dental care requiring general anesthesia. Since then, all patients receive a financial interview, to determine the type and amount of payment to be charged. Medicaid and no-charge patients at Children's Hospital showed a high significance for returning for recall appointments. The children are seen by other medical specialties, thus improving the chances that they will return for a recall evaluation. Most private patients at Children's Hospital did not return for recall. These patients were followed postoperatively at the Medical College of Virginia Dental School, by the attending pedodontist.

Both hospitals reported as significant that more patients returned for recall who lived within the city of Richmond than outside. Patients from outside of Richmond were more likely to return to their referring dentist. This could be important when deciding upon the mode of treatment for patients who live a distance from the treatment center. Space maintaining procedures, such as lingual arches and band and loops, should be questioned, if the patient's return to the clinic is unlikely.

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Noses, tongues, and teeth

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The subject of nasal respiration is once again popular on the orthodontic lecture circuit. Renewed interest in the relationship between breathing patterns, tongue position and orthodontic management is always a welcome event, as it reminds the orthodontist that appliance forces have enemies as well as friends. In this paper, we shall not address ourselves to the importance of these factors with regard to the functional-

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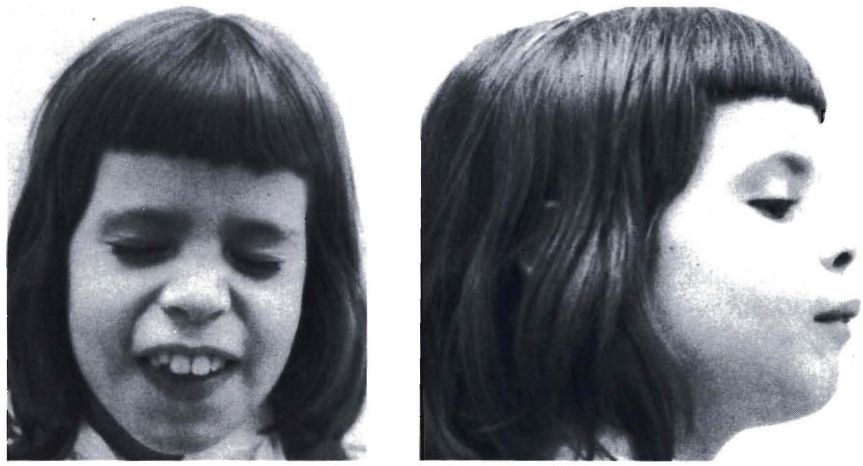
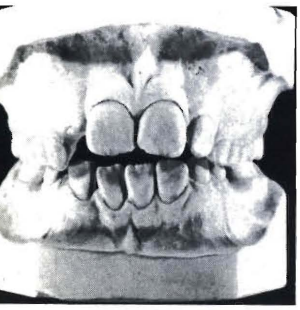
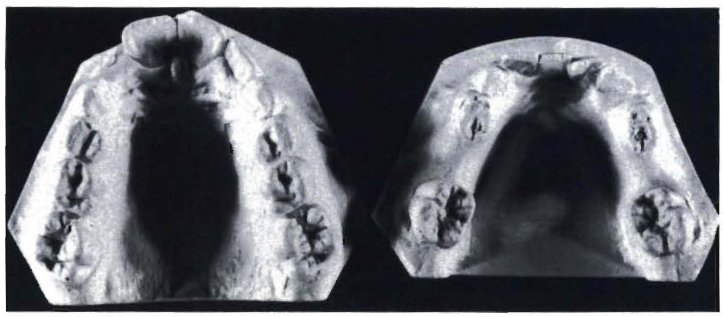


Figure 1. Records at time of first examination. Patient is seven years of age. Note open bite.





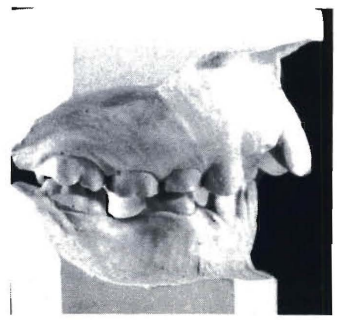
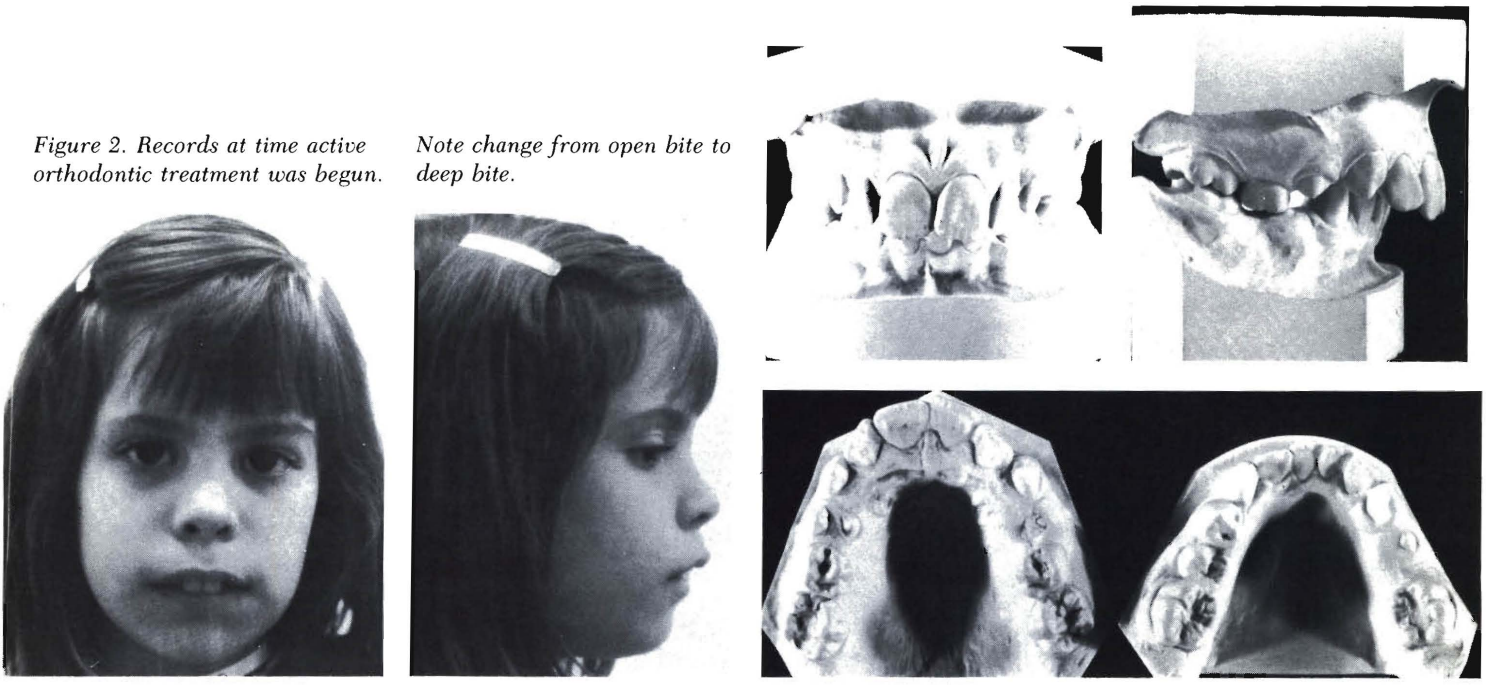


Figure 2. Records at time active orthodontic treatment was begun. Note change from open bite to deep bite.



matrix concept of growth, but rather to their effect upon arch-form and occlusal contacts. Once growth has been completed, probably no factors have a greater influence upon the maintenance of arch-form and occlusal contact than normal breathing and normal tongue posture during rest and function.[1] This concept is based upon acceptance of a cause-and-effect relationship between muscle forces, arch-form and occlusal contacts. This relationship seems much clearer to the clinician than to the researcher. For an interesting and critical review of the conflicting views on the subject of nasal breathing and dentofacial morphology, the reader is referred to a recent publication by O'Ryan *et al.*[2]

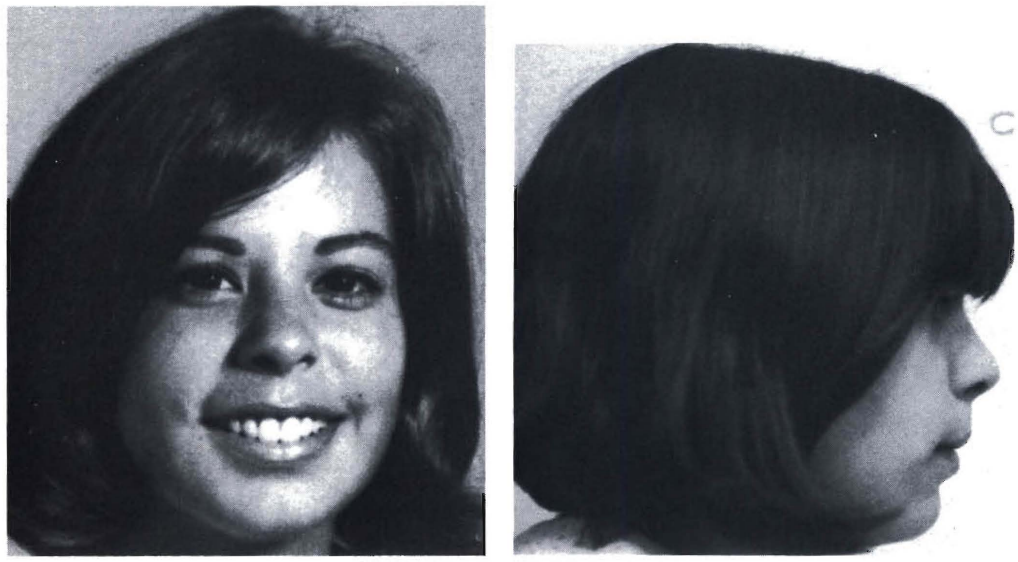
Mouthbreathing, regardless of the cause, can be counted upon to: (a) lower the rest position of the tongue in order to clear the airway, and (b) alter the net molding effect of tongue, lips and cheeks upon the position of the teeth. This may lead to a crossbite due to lack of tongue support for the maxillary arch, and spacing or flaring due to loss of the restraining force of normal lip closure. Abnormal tongue position during rest, swallowing, or speech may greatly reduce the chances of orthodontically producing and maintaining good interarch relationship in the vertical plane of space. In other words, mouthbreathing and abnormal tongue posture are major ingredients for an orthodontic treatment failure, or a compromise result at best.

The orthodontist, observing the effect upon arch form of positive changes in tongue and lip position, may experience unbridled joy—the joy that comes from watching a malocclusion improve by itself, or from the knowledge that a dramatic change in prognosis has taken place. The orthodontist, observing the effect of an unaltered abnormal tongue and lip position upon a completed orthodontic case, may experience unbridled sadness—the sadness that comes from watching a corrected malocclusion return to its original form.

The spontaneous changes that come about when normal breathing and tongue behavior are restored may occur in one or two stages, depending upon the degree of anteroposterior discrepancy for a given patient. In Class II cases with a large overjet, the first stage changes are mainly in the vertical direction. The complete molding effect of normal lip closure must wait until the orthodontic correction of the overjet takes place. What is important is that the potential for the normal molding effect has been brought about by removing whatever may have been the cause of nasal obstruction or abnormal tongue position. The prognosis for postretention stability now changes from very negative to very positive.

For the Class I patient, spontaneous changes in buccolingual, vertical and anteroposterior directions may take place simultaneously. Adjunctive mechanical therapy may or may not be necessary. If needed, it may take the form of a Hawley, or fixed appliance. They will all succeed under the stimulus of a normal tongue-lip environment.

The case presented below illustrates some of these principles. A seven-year-old female patient presented for orthodontic consultation (Figure 1). The Class II malocclusion was complicated by the factors mentioned above, namely tongue-thrust and mouthbreathing. The patient was sent for medical consultation to determine the cause of nasal obstruction. Allergies and enlarged adenoid tissue were the causative factors.[3] These were treated medically and surgically. Within a period of one year, normal breathing potential was restored. The patient was subsequently sent for tongue-thrust and speech therapy. The spontaneous changes that took place as a result of guiding treatment are shown in Figure 2. It is important to emphasize the change in prognosis



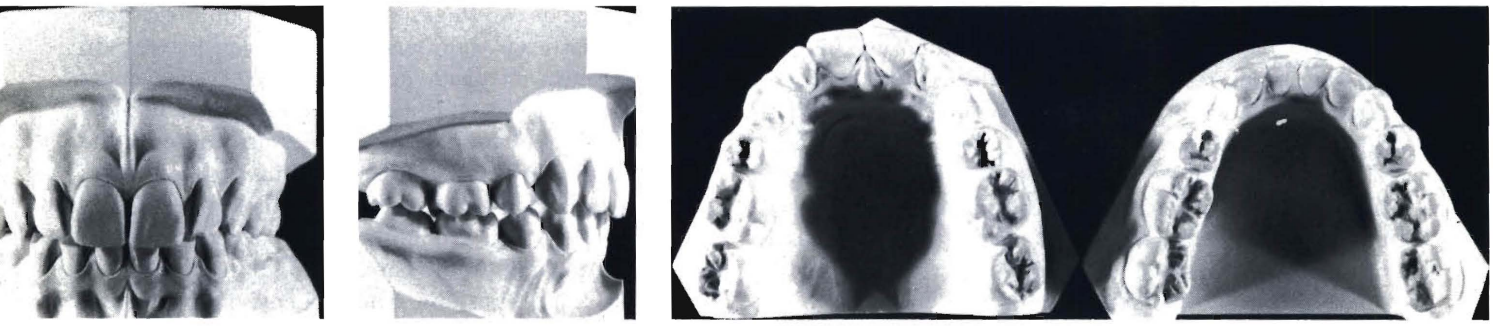


Figure 3. Final records, one year postretention. Note stability of vertical correction.

that now occurred. In the presence of an open-bite at the onset of active treatment, the orthodontist is justifiably concerned about posttreatment stability. In the presence of a deep bite, this concern is greatly reduced. At age twelve, active treatment was begun. Hygiene was very poor and in the interest of preserving tooth enamel, bands were removed early and spaces were not completely closed. In spite of this, the final records, one year postretention (Figure 3), justified our positive prognosis.

While researchers continue to search for elusive mathematical formulae to prove muscle equilibrium, the clinician is best advised to strive for normal breathing patterns and tongue positions. This will increase the chances for creating and maintaining more nearly normal arch-form and occlusal contact relations.

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Multiple localized root agenesis

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The onset of root development occurs after enamel and dentin formation has reached the presumptive cementoenamel junction. This development is guided by the enamel organ which forms Hertwig's epithelial root sheath, a developmental structure which molds the shape of the roots.[1] Since Hertwig's root sheath is formed from the enamel organ, it is quite possible that an insult to these tissues during development will cause disruption or cessation in its future morphology. This report describes a case of localized root agenesis concomitant with microdontia.

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Dr. Alexander is Associate Professor, Dept. of Children's Dentistry, School of Dental Medicine, S.U.N.Y. at Stony Brook, Stony Brook, New York 11794.

CASE HISTORY

A fifteen-year-old white male came to the School of Dental Medicine, S.U.N.Y. at Stony Brook, seeking comprehensive dental care. At the time of presentation the patient was found to be in good health. His medical

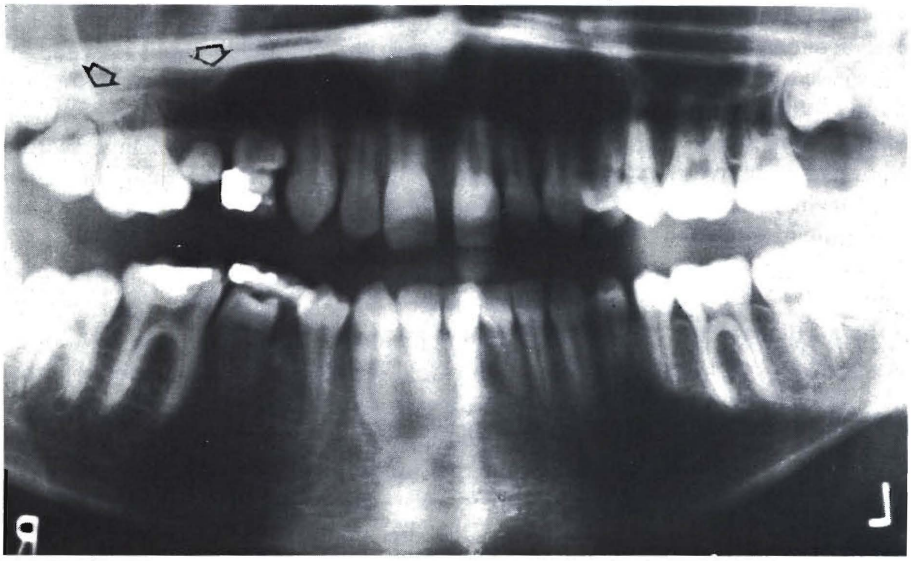

Figure 1. Panoramic radiograph illustrating root agenesis of the maxillary right second permanent molar and first and second premolars (arrows).

history was essentially within normal limits.

Regional head and neck examination was normal. Intraoral examination was essentially normal, except for an over-retained maxillary first primary molar and an over-retained mandibular right second primary molar. Multiple amalgam restorations and new and recurrent caries lesions were also present. Space loss was noted in the area of the maxillary right quadrant, due to the lack of eruption of the second premolar and to mesial drifting of the first permanent molar.

In a panoramic examination (Figure 1), the maxillary right second permanent molar, and the first and second premolars were noted to lack complete root formation. This finding was most evident in the area of the premolar teeth, where it appeared that root formation ceased shortly after crown formation (Figure 2). Additionally, the crowns of these teeth appeared to be considerably smaller than their antimeres, and were diagnosed, therefore, as being microdonts. The pulps of the premolars also appeared to be obliterated.

The immediate dental treatment consisted of restoration of all carious teeth and the extraction of the right maxillary first and second premolars and the over-retained primary teeth. A removable acrylic space maintainer with pontics was inserted until the patient was considered ready for orthodontic treatment and a fixed partial denture. The maxillary right second molar appeared to be stable in the arch, displayed minimal mobility and, therefore, was not removed. The status of this tooth, however, was noted for review at subsequent recall examinations.

DISCUSSION

Leonard, and Lerman and Gold described a condition in two patients in which localized root agenesis occurred.[2,3] A striking resemblance of this case to the two cases already reported leads us to believe that this is the third published report of idiopathic short roots. Ironically, these reports appear at approximately five- to six-year intervals—1972, 1977 and again in 1983, which suggests that their clinical appearance is infrequent, or that it is an anomaly not observed or frequently reported.

According to Finn, calcification of the maxillary premolars and second permanent molars ranges from 1.5 years to three years after birth, while root completion ranges from twelve to sixteen years of age, respectively.[4] Based upon these figures, damage to the root sheath in this patient was presumed to have occurred somewhere between the ages of three to five years.

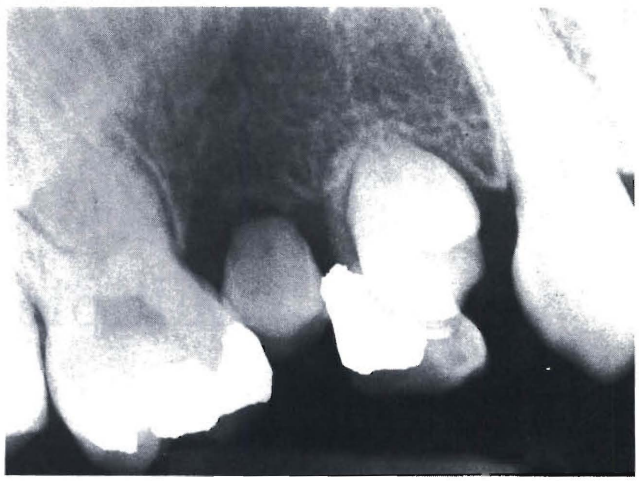

Figure 2. Periapical radiograph of the maxillary right premolar area. Note the lack of root formation and obliteration of the dental pulp. The second premolar appears to be "floating" in soft tissue.

The medical history, however, does not confirm this assumption. The over-retained primary teeth may indicate, however, that this individual was developmentally delayed and injury to these structures may have occurred later in life. The medical history does not, however, support this statement; the cause of this root anomaly, therefore, has been termed idiopathic, as described previously.[3]

Cessation of root development or total root agenesis has been associated with a variety of systemic, congenital or genetic disorders. Included here are thalassemia, Turner's syndrome, Ehlers-Danlos syndrome, scleroderma, and hypoparathyroidism.[5-8] External trauma in the form of radiation and electrical burns has been known to arrest dental development as well.[9-11]

Inheritable defects in tooth development such as dentinogenesis imperfecta have been associated with short roots.[5] Additionally, morphologic changes within the pulp chamber of those individuals have been observed. Although radiographic changes within the pulp were noted in this case, family history was negative for hereditable disorders. Histologic examination of the extracted teeth was not performed, however, to rule out any microscopic defects.

Lerman and Gold report that it is quite possible that this condition may have a familial tendency and go undetected without routine dental radiographs.[3] Other members of the family may have the same condition without being aware of it. Based upon the available information, insufficient evidence leads us to believe

that this condition is idiopathic in nature, and occurs infrequently in the general population.

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CLINICAL ERRORS AND ROOT RESORPTION

Root resorption is deleterious to the oral health of the patient for at least two reasons: discoloration or divitalization of the tooth may result and mobility and periodontal pathology can occur. Ketcham discussed the problem of root resorption as early as 1927. It was noted that appliances that encouraged bodily tooth movement caused a greater number of root resorptions than appliances that only tipped the teeth. Rudolph, in 1936, demonstrated a higher degree of root resorption in older patients and less resorption in younger patients. It is evident from studies in 1954 by Massler and Malone and in 1955 by Philips that root resorption is prevalent when teeth are bodily moved through bone. In the Massler study, 93.3 percent of all teeth examined had at least 1 mm of resorption; in the Philips study, 84.0 percent of maxillary central incisors evidenced root resorption. DeShields, in 1969, found that in a sample of nonextraction cases, 81.73 percent of maxillary central incisors underwent definite root resorption. The gender of the patient did not affect the amount or prevalence of root resorption. Since it was suspected that apical root movement could be a cause of root resorption, DeShields recommended that "unnecessary tooth movements should be avoided."

Most investigation of root resorption indicates that the more a tooth is moved mechanically (bodily or by tipping, torque, intrusion, or extrusion), the greater the chance for resorption of the roots. Since extraction of premolars requires relatively extensive tooth movements, this form of therapy for malocclusion could be expected to cause more frequent root resorption. Goldson and Henrikson found root resorption especially prevalent in maxillary central incisors that underwent root torque found severe root resorption during Stage II of the Begg technique and suggested that the resorption might be caused by movement of the incisor apices against the labial cortical plate.

Ahlin, J.H.; White, G.E. *et al*:
Maxillofacial orthopedics. Chicago:
Quintessence Books, 1984, pp 301, 302.

Development

Impacted maxillary second permanent molars

Reijo Ranta, DDS, Dr Odont

The etiology of tooth impaction includes lack of space, rotation and inclination of the tooth bud, supernumerary teeth, odontoma or other pathological entity.[1] In order to prevent impaction by physical obstruction, these are the factors that must be assessed.

The most frequently impacted teeth are the third molars, the maxillary canines and the mandibular second premolars. The other teeth are only rarely affected. Among the permanent teeth, the maxillary second molar is the least commonly involved.[1-3]

The purpose of this paper is to describe fifteen children in whom twenty-one maxillary second molars were impacted by the third molars.

MATERIALS AND FINDINGS

Over the past ten years, the author detected twenty-one maxillary permanent second molars impacted by the third molars in fifteen subjects. Of these subjects, six were examined at the Finnish Red Cross Cleft Center in Helsinki, five in the Department of Pedodontics and Orthodontics, Institute of Dentistry, University of Helsinki and four in Lohja Town Health Center. Annually at least a thousand children attend for orthodontic treatment or examination in each of these places of treatment.

Dr. Ranta is an orthodontist with the Finnish Red Cross Cleft Center and senior lecturer in the Department of Pedodontics and Orthodontics, Institute of Dentistry, University of Helsinki, Helsinki, Finland.

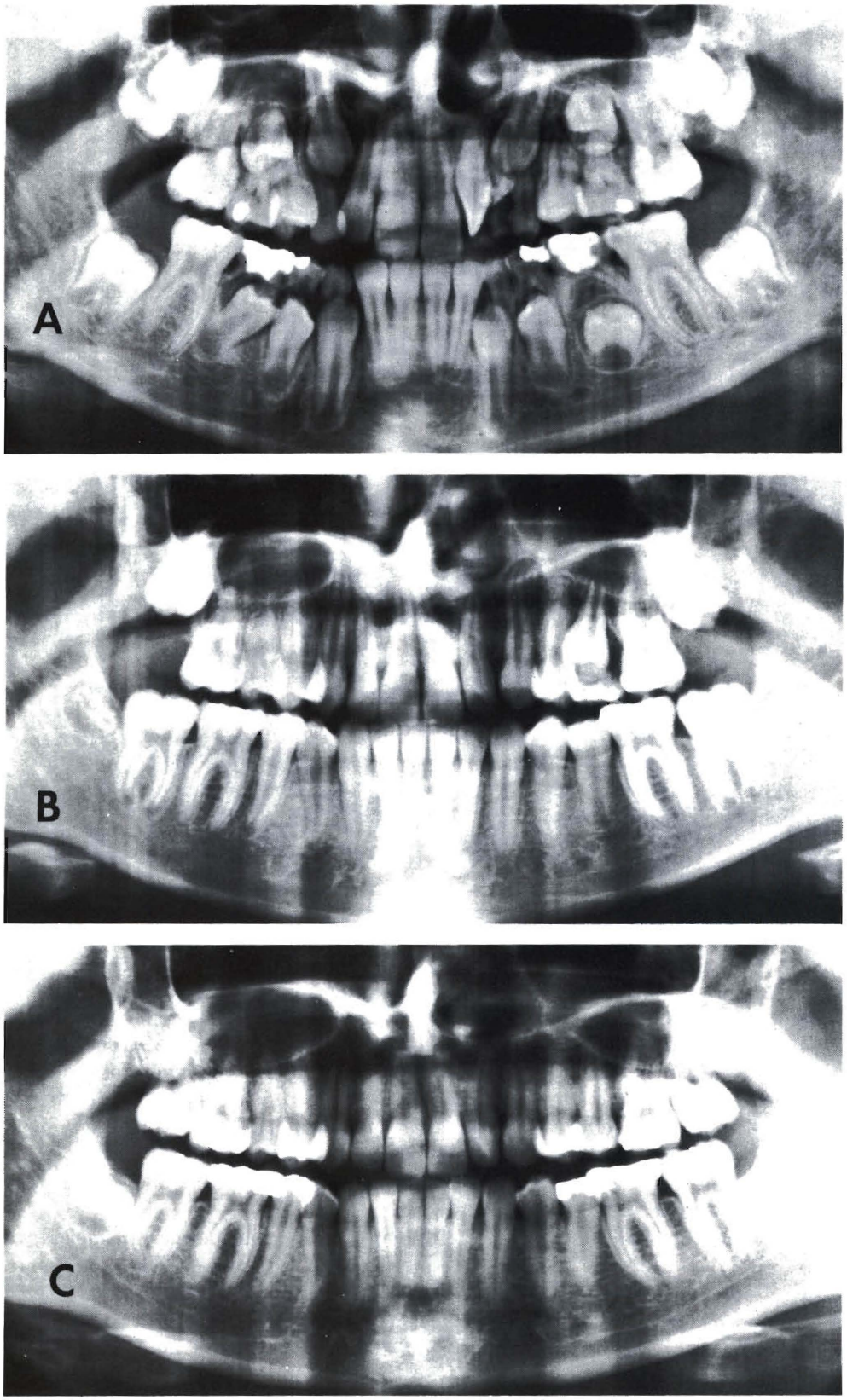

Figure 1. A, a girl with cleft lip at the age of nine years, and bilateral maxillary second molars impacted by the third molars. B, the location of the upper second molars at the age of twelve years, twelve months after removal of the upper third molars. C, spontaneously erupted second molars at the age of sixteen years.

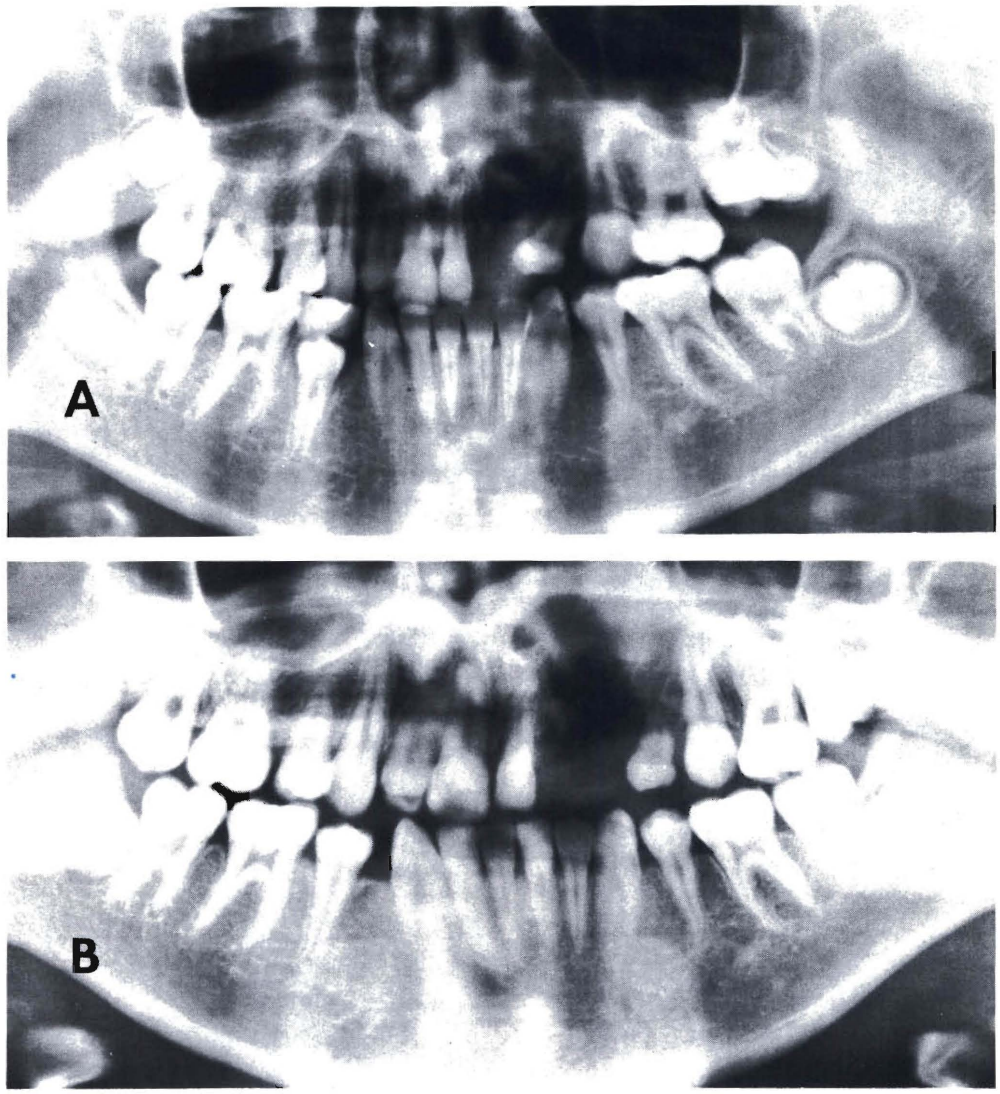

Figure 2. A, a girl with cleft lip and palate at the age of 13.5 years, and the left maxillary second molar impacted by the third molar. B, delayed eruption of the second molar followed by the accelerated eruption of the third molar at the age of fifteen years.

Among the fifteen subjects, there were six with cleft lip and/or palate and nine without anomalies. Among the eight girls and seven boys, six had bilateral impactions and nine had unilateral impactions. Of the unilateral cases, five were on the right side and four on the left side. In these subjects, other impacted teeth were observed in two cases: one had an impaction of the left permanent maxillary canine; and the second had impacted lower third molars. At the time of the diagnosis of impaction, the age-range of the subjects was from nine to eighteen years.

The third molar was generally positioned occlusally and palatally in relation to the second molar, and the crown of the third molar was inclined mesially (Figures 1, 2). The times of crown and root formation of the second and third molars were within normal limits in all subjects, and the inclination of the first and second molars in relation to the occlusal plane was normal. No gross deviations from the normal length of the upper dental arches were noted, excluding two subjects with cleft lip and palate, who had a severe maxillary hypoplasia. The upper first molars had not undergone any distal inclination during the orthodontic treatment, in these two cases.

The eruption of the second permanent molars was observed in relation to the eruption of their antimeres and of the lower permanent second molars. Only in one subject did the second molar erupt without removal of the third molar (Figure 2). Fourteen third molars were removed; in one subject, aged eighteen

years, the second molar was removed. The eruption of the second molars was observed regularly and confirmed after the removal of the fourteen third molars (Figure 1).

DISCUSSION AND CONCLUSIONS

The reason for the impaction of the twenty-one maxillary second permanent molars was the occlusal, mesial and palatal malposition and the mesial inclination of the buds of the adjoining third molars. No other reasons for impaction were observed. All fourteen second molars erupted normally after the removal of the third molars. In six of the fifteen subjects, the impaction was bilateral. These confirm the etiology to be a physical barrier, namely, the third molar. Furthermore, the fourteen third molars were removed before the beginning of root formation or of their eruption. Thus, the malposition of the buds of the third molars occurred during the formation of the dental lamina and the tooth buds. In spite of the relatively normal lengths of the maxillary dental arches, the lack of space for the buds of the third molars may be the reason for their malposition.

The recommended treatment of the maxillary second permanent molar impacted by the third molar is removal of the third molar when the patient is eleven to fourteen years of age. It is imperative that the eruption of the second molar be carefully observed and confirmed.

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IMPACTION

Sex: No predilection

Clinical Features: Impaction or retention of teeth is one of the most common developmental dental defects encountered in man. Failure of teeth to erupt may be the result of a physical barrier, unexplained loss of eruption force, or a tumor or cyst of the jaws arising from odontogenic or non-odontogenic tissues. The mandibular third molars. and maxillary canines are most frequently involved, followed by the maxillary third molars. Maxillary central incisors, mandibular canines, and premolars in both arches are also retained with some degree of frequency. Supernumerary teeth such as mesiodens and premolars often fail to erupt. Multiple impactions and supernumerary teeth are encountered in cleidocranial dysostosis, which is characterized by the aforementioned dental defect in conjunction with agenesis or hypoplasia of the clavicles and craniofacial anomalies including Wormian bones, open fontanels, hypoplastic sinuses, and other defects of both membranous and endochondral bones. Gardner's syndrome is also associated with multiple dental impactions. Impacted teeth are also seen in various forms of amelogenesis imperfecta and in syndromes associated with enamel defects. The significance of impacted teeth rests with the latent potential of the follicular tissues to undergo cystic or neoplastic transformation.

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Bilateral malposition of maxillary second premolars: report of case

Wa Sham Cheung, BA, DDS

The malposition of an unerupted permanent tooth is a fairly common dental abnormality. Excluding causes such as impaction, or displacement by a tumor or trauma, the most common cause is from ectopic eruption. Ectopic eruption of a tooth has been defined as an invasion of an abnormal alveolar position during its eruptive course.[1] It has been suggested that certain intrinsic factors may predispose the ectopic eruption of the first permanent maxillary molars.[2] The ectopic eruption of permanent incisors, canines and premolars is usually caused by extrinsic factors such as over-retained primary teeth, inadequate space in the alveolar arch due to hereditary crowding, or loss of arch length. With the exception of abnormally formed dental structures like mesiodens, supernumerary teeth, or odontomas, the idiopathic malposition of permanent tooth buds before their eruptive phase is unusual. The more frequently reported cases are the transposition of the maxillary canines and first premolars.[3-5] Platzer noted that the retention or early loss of primary teeth could be the causative factor for the transposition.[6] Mader and Konzelman cited two possibilities for the transposition abnormality.[3] It could be the result of the transposition of the anlagen of the teeth during odontogenesis, or from the migration of a tooth away from its normal path of eruption.

The present article is to report a case of bilaterally malpositioned maxillary second premolars.

Dr. Cheung is in the private practice of pedodontics in Coquitlam, B.C., Canada.

CASE REPORT

Patient D.A., eight-year-old male, North-American Indian, was presented to the dental office for routine dental examination. D.A. is a well-developed, well-nourished boy without any known contributory medical or dental history. The mother was concerned about his lingually rotated erupting maxillary permanent lateral incisor.

The panoramic radiograph revealed the bilaterally malpositioned unerupted maxillary second premolars. The image of the maxillary second premolar was superimposed on that of the first premolar on each side (Figure 1). The periapical radiograph on each side of the maxillary posterior region confirmed these findings (Figures 2,3).

The patient's dentition was in the mixed stage. The permanent maxillary right lateral incisor was rotated slightly lingually (Figure 4). No other abnormalities were noted.

The panoramic radiograph of D.A.'s natural younger brother, J.A. (seven years old) revealed a congenitally missing permanent mandibular left lateral incisor (Figure 5).

Since both brothers were adopted, the dental histories of the other members of their natural family were not available.

MANAGEMENT CONSIDERATIONS

Different approaches can be taken in managing this case. Ideally, the full complement of the maxillary den-

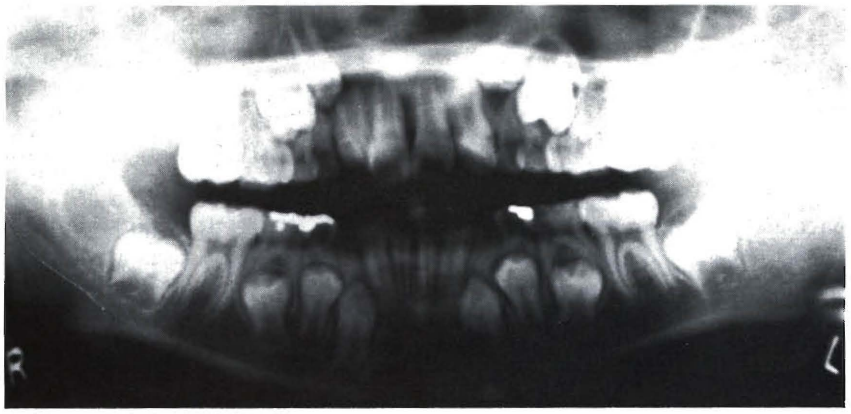

Figure 1. Panoramic radiograph of the patient showing bilateral malposition of the maxillary second premolars.

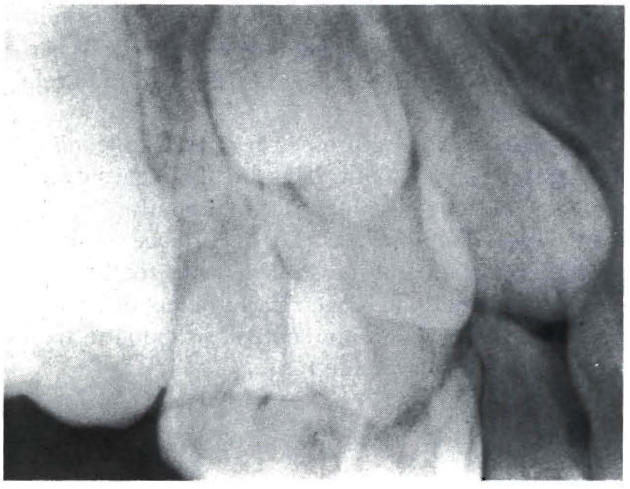

Figure 2. Maxillary right posterior periapical radiograph

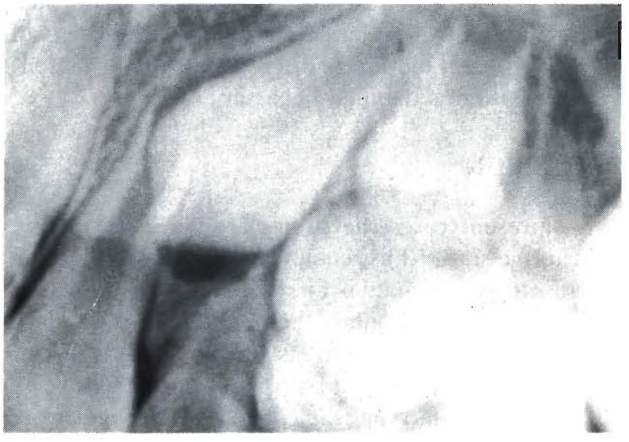

Figure 3. Maxillary left posterior periapical radiograph.

tition would be maintained, if a premolar on each side could be moved distally to the appropriate position with orthodontic treatment. Since the first and second premolars on each side appeared to be located side-by-side buccally and palatally, it may not be possible to move one premolar on each side to the ideal position without causing excessive root resorption.

An alternative is to remove surgically the more deviant-positioned premolar, on each side, as well as the maxillary second primary molars; then move the maxillary permanent molars mesially, and align the maxillary arch at the same time with a fixed orthodontic appliance.

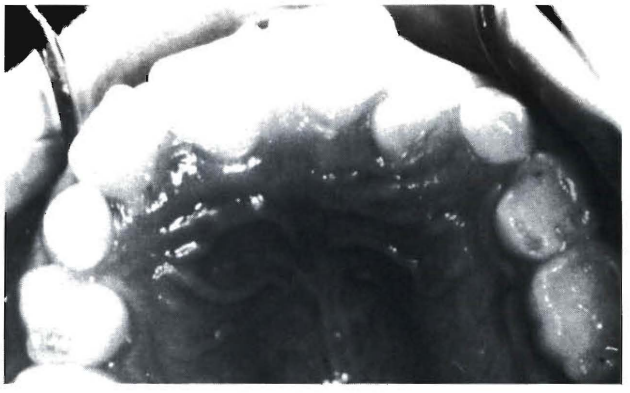

Figure 4. Intraoral view of the maxillary arch of the patient.

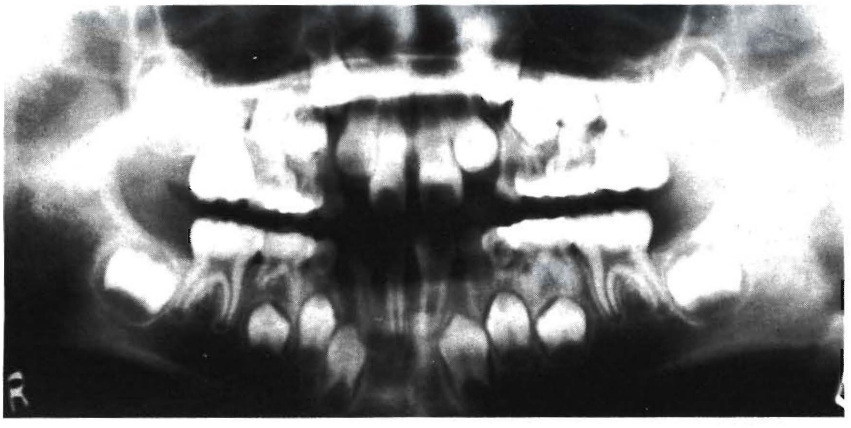

Figure 5. Panoramic radiograph of the patient's brother, showing congenitally missing mandibular left lateral incisor.

The third option is to remove just one maxillary premolar on each side, retain the maxillary second primary molars, then align the maxillary arch orthodontically. Fixed prostheses, possibly the more conservative type (the acid-etched bridges) will be constructed, when the maxillary second primary molars exfoliate naturally.

The patient is presently only eight years old. It will be at least two or three years before any attempt at management of the case will be initiated. The final treatment plan will be based largely on the severity of the arch-size and tooth-size discrepancy; but orthodontic intervention is deemed necessary.

DISCUSSION

In the present case, the premolars were still in the preeruptive stage; thus, ectopic eruption would not be a satisfactory explanation for their unusual positions. Unlike the maxillary canine, the premolar does not have a long path to follow from its developmental origin. Deviation from its normal path is inconceivable.

It has been found that the congenital absence of the maxillary second premolar is fairly common.[7] The occurrence of supernumerary teeth in the maxillary premolar area is uncommon.[8] It is possible that the patient may be missing the maxillary second premolars, congenitally and bilaterally, and also have a supernumerary maxillary first premolar on each side.

Stewart theorized that congenital absence of teeth may rise from:

- ☐ Physical obstruction or disruption of the dental lamina.
- ☐ Space limitation, especially third molars.
- ☐ Functional abnormalities of the dental epithelium.
- ☐ Failure of induction of the underlying mesenchyme.[9]

The causes of supernumerary teeth or hyperdontia are not clear. The permanent tooth germ begins formation when there is further proliferative activity within the dental lamina at a point where it joins the enamel organ of the primary tooth germ.[10] It is possible that another burst of proliferative activity starts at another point of the dental lamina at about the same time. Consequently, an extra tooth germ may develop. It has also been suggested that hyperdontia can be the result of twinning.[11] According to Tannenbaum and Alling, twinning occurs when a tooth bud cleaves completely, resulting in the formation of an extra tooth in the dental arch that is usually a mirror image of its adjacent partner. The term schizodontia has also been used to refer to this occurrence.

The patient's natural brother also has a congenitally missing permanent tooth. This highly suggested hypodontia could be hereditary in their family.

This case demonstrates the importance of radiographic examination of the succedaneous teeth, in the early mixed dentition stage. Early detection of such abnormality can allow interceptive orthodontic treatment at the most favorable time.

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Management of ectopically erupting first permanent molars

Robert D. Rust, DMD
Guthrie E. Carr, DDS

REVIEW OF THE LITERATURE

Braden refers to ectopic eruption as the abnormal mesioangular eruption path of the maxillary first molar which causes it to become impacted above the distal prominence of the crown of the second primary molar.[1] The progression of this abberant eruption causes resorption of the distobuccal root of the primary second molar, possible pulpal infection and premature loss of the primary tooth.[2]

Pulver and Young have shown an incidence of about 3 percent of this condition in the population.[3,4] Young further stated that 66 percent of the ectopically erupting molars erupted into their normal positions without treatment, while the remaining molars were trapped in the "hold position". Carr observed a more frequent occurrence in children with cleft lip and palate, noting 29 percent of the girls and 22 percent of the boys had evidence of ectopic eruption after lip and palate repair.[5]

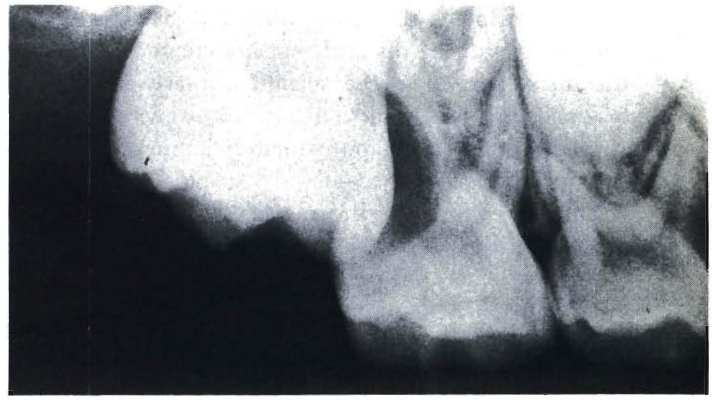
Various treatment techniques for this condition have been suggested.[1,6-8] In general, the customary treatment consisted of an auxillary spring.[9,10]

The purpose of this paper is to present a simple direct technique for tipping ectopically erupting permanent molars distally. Clinical cases by both authors have consistently provided effective treatment in numerous cases within six to eight weeks with minimal adjustments required. A representative case is reported.

CASE REPORT

A Caucasian female, five years, six months of age, presented to the graduate pedodontic clinic at the Indiana University School of Dentistry. Initial examination revealed that both right and left second primary molars showed radiographic evidence of root resorption, secondary to atypical eruption of the permanent maxillary first molars. No clinical signs of eruption of permanent molars were noted. Because of the patient's age and the eruptive stage of the permanent molars, the condition was noted, but no treatment was provided and reevaluation of the patient in six months was advised.

Figure 1. Right permanent first molar is erupting ectopically. Resorption can be noted on the right primary second molar.



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Dr. Carr is Assistant Professor, Department of Pedodontics, James Whitcomb Riley Hospital for Children, Indiana University School of Dentistry.

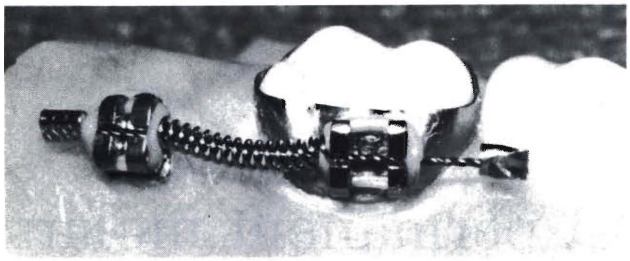

Figure 2. A model demonstrating an appliance to move the molar distally.

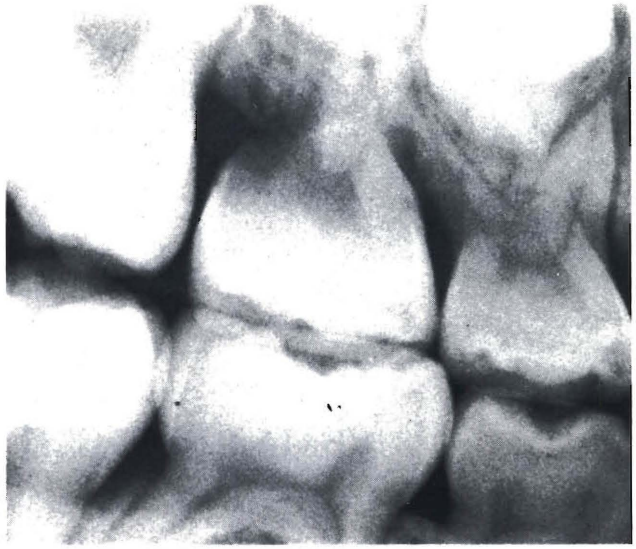

Figure 3. Six-month posttreatment radiograph. The condition is corrected.

At the recall appointment, the maxillary left permanent first molar had corrected spontaneously and continued to erupt normally. The maxillary right permanent first molar, however, showed a continued mesial migration and an increased resorption of the distobuccal root of the primary second molar (Figure 1). The tip of the distobuccal cusp was visible in the oral cavity. Treatment was instituted at this time.

A band was adapted to the second primary molar with a standard edgewise bracket on the buccal surface. A lower incisor edgewise bracket was then bonded to the exposed distobuccal cusp. A .0175 twist orthodontic wire with a .010 × .030 open coil spring was cut to fit from the mesial wing of the permanent molar bracket to the mesial wing of the primary molar bracket. The orthodontic wire was adjusted to provide 3 to 4 mm of excess length, to allow for tipping the permanent molar distally. Arch-wire stops were placed on the mesial and distal ends of the wire to resist impingement of the wire into the buccal vestibule and to prevent the wire from sliding out of the brackets. The spring was then placed with the coil compressed between the two brackets and secured with orthodontic elastic modules (Figure 2).

The patient was seen in three weeks for observation, when it was noted that the permanent molar was tipped significantly to the distal. It has not cleared, however, the distal portion of the second primary molar. The original wire was removed and a new wire and coil spring assembly with approximately 2 mm of compression were inserted.

Three weeks later, the mesial portion of the permanent molar had successfully cleared the distal prominence of the second primary molar and the appliance was removed. A six-month, posttreatment examination showed the permanent molar in a good occlusal relationship (Figure 3).

DISCUSSION

The appliance used in this case report has several advantages. A direct technique is used, which eliminates the need for impressions or laboratory procedures. Thus, a second insertion appointment is also unnecessary. Since the bracket is bonded to the buccal surface, distortion of the appliance by opposing occlusion is unlikely. The wire and coil spring can be easily removed for convenient adjustment. Undesirable rotations of the treated permanent molars have not been observed with the appliance.

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Incidence of enamel hypoplasia in primary canines

Gary R. Badger, BS, DDS, MS

Rarely has an enamel defect been noted in isolation. Most defects occur in multiples, as part of an inherited enamel hypoplasia. Enamel defects do occur regularly, however, in the primary dentition, on the facial surfaces of the primary canines at the junction of the gingival and middle thirds. Primary canines begin to calcify at approximately five months *in utero*. Enamel formation is completed at approximately nine months of age.[1] At birth, approximately one-third of the enamel is formed; completion of enamel formation will occur in the next nine months. The junction of the middle and gingival thirds should calcify, therefore, around four and one-half months of age, assuming a steady calcification rate. Metabolic disturbances at this time may result in enamel defects. These defects may occur both in the maxillary and in the mandibular teeth. This study was undertaken to determine the incidence of such defect.

METHODS AND MATERIALS

Fifty-five children between the ages of one year, six months and eleven years, six months were selected at random. There were thirty females (average age, 5.75 years) and twenty-five males (average age, 5.5 years). Each child was examined using a dental mirror and explorer, in a typical setting. Teeth determined to have the hypoplastic defect were characterized by:

- ☐ No caries.
- ☐ No restoration at that location.
- ☐ A defect occurring on the labial surface of primary canine at the junction of the middle and gingival thirds (Figure).

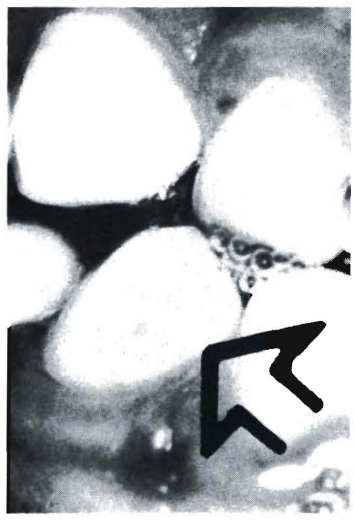

Figure. Hypoplastic lesion on right mandibular primary canine (arrow).

Table 1 ☐ Incidence of enamel hypoplasia in primary canines.

Canines affected	Females (30) percent	Males (25) percent
2 maxillary and 2 lower	8	6.7
1 maxillary	16	13.3
1 lower mandibular	4	10.0
2 maxillary and 1 mandibular	0	3.3
2 mandibular and 1 maxillary	4	3.3
2 maxillary only	0	3.3
2 mandibular only	8	6.6
1 maxillary and 1 mandibular	4	0.0
	44	46.5

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Table 2 ☐ Location of canine affected; 22 percent of all teeth examined had defects.

	Females (120) percent	Males (100) percent
Right maxillary	22.7	25.9
Right mandibular	22.7	22.2
Left maxillary	27.3	22.2
Left mandibular	27.3	29.7

RESULTS

Results showed that 22 percent of all teeth examined had a hypoplastic area. Forty-four to 46 percent of patients examined had at least one tooth involved. Table 1 shows the percentage of all patients having each combination of defects. Table 2 demonstrates what percentage of primary canines was affected in all patients.

DISCUSSION

The occurrence of hypoplastic defects on 22 percent of all primary canines examined suggests a frequent disturbance of the matrix formation at about 4.5 months of age. Equal occurrence in both male and female indicates no difference due to sex.

CONCLUSION

Twenty-two percent of all primary canines investigated in this random study showed evidence of a hypoplastic defect at the junction of the gingival and middle thirds. Further investigation as to the etiology is indicated.

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ENAMEL HYPOPLASIA

Sex: No predilection

Clinical Features: Damage to ameloblasts during odontogenesis, if severe, will result in defective enamel formation. A variety of infectious, nutritional, chemical, and traumatic factors play a role. (Because of their unique characters, congenital syphilis and dental fluorosis are considered separately.) Perhaps the most common form of enamel hypoplasia is seen in isolated permanent teeth, whereby caries with periapical spread of infection or trauma to a deciduous tooth results in damage to ameloblasts forming the crown of the subjacent developing permanent tooth. The affected tooth, so-called Turner's tooth, is yellow or brown, and the enamel surface is pitted or chalky. Occasionally, many teeth show pitting or rough, poorly calcified enamel matrices. In these instances, one of the childhood infectious diseases associated with prolonged elevated temperature may have resulted in ameloblastic injury, in which case only that portion of the teeth under development at the time of infection manifests hypoplastic changes. Enamel hypoplasia with a similar distribution as that seen with febrile childhood infections may occur in rickets, congenital hypoparathyroidism, and birth injuries.

Treatment: When the pulp chambers have receded to an appropriate level, permanent restorations may be fabricated. In the interim, resin restorative materials may be periodically placed to achieve a desirable esthetic appearance.

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Case reports

Nisentil-induced respiratory arrest: report of case

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Paul J. Schwartz, DMD
C. Richard Bennett, DDS, PhD

Historically, management of the uncooperative pediatric dental patient with sedative medications has been associated with a small incidence of adverse reactions. Recent trends in pedodontics have been toward pharmacologic management of young patients who are either physicially or emotionally incapable of cooperation, through the subcutaneous administration of narcotic analgesics. The use of alphaprodine (Nisentil) has been advocated to decrease apprehension and provide a more relaxed patient, thus minimizing the psychic trauma frequently associated with extensive dental treatment in young patients.

Alphaprodine is a synthetic narcotic with a chemical structure and pharmacologic activity similar to meperidine (Demerol). It is particularly valuable in pediatric dentistry because of its rapid onset and short duration of action, following intraoral, submucosal injection. The central nervous system activity of alphaprodine is usually manifest within ten minutes of submucosal administration. The duration of its central effects are reportedly between one and two hours (onset time and duration are variable, and dose related). The major adverse reaction associated with alphaprodine, as with all narcotics, is the possibility of respiratory arrest.[1]

Following is a case report in which this potentially

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fatal reaction was encountered and subsequently treated without ill effects.

CASE REPORT

A well-developed three-year-old black male child weighing 18.2 kg presented to the Graduate Pedodontic Clinic for dental care. Initial work-up revealed the need for multiple dental restorations. Because the child appeared to pose significant behavioral/management difficulties, subcutaneous alphaprodine sedation was selected. Ten mg alphaprodine (0.55 mg/kg) plus 0.2 mg atropine were injected from a 1 cc tuberculin syringe into the mucobuccal fold of the posterior maxillary arch on the side opposite to the one in which a local anesthetic was to be administered. Dental therapy, however, was provided with moderate management difficulties, apparently because the degree of sedation and cooperation was clinically insufficient. The child was given another appointment.

At the second appointment, a submucosal injection of 14.0 mg alphaprodine (0.77 mg/kg) plus 0.2 mg atropine into the mucobuccal fold of the posterior left maxillary arch was made. As with the previous appointment, the patient's vital signs were monitored, utilizing a precordial stethoscope and a pediatric blood pressure cuff. Within five minutes the patient was quiet and accepted the injection of half a standard dental carpule of 2 percent lidocaine with 1:100,000 epinephrine. No physical restraint was required. A rubber dam was applied and the first preparation begun.

At ten minutes following administration of the alphaprodine, the patient was responsive to verbal stimuli, although a decrease in respiration rate was observed by the anesthesiologist. Tidal volume appeared unchanged as judged by palpation of the child's chest during respiration. Similarly, other vital signs remained stable. Within one minute, the airway became partially obstructed, as noted by the appearance of tracheal "tugging" and audible snoring sounds. The problem was easily corrected by repositioning the patient's head into an extended position, in order to move the mandible forward and thus lift the base of the tongue away from the posterior pharyngeal wall to provide an open airway. The patient, however, became progressively unresponsive to verbal and tactile stimuli, necessitating discontinuation of the dental procedure.

The rubber dam was immediately removed and a triple airway maneuver performed by placing both index fingers on the posterior borders of the ramus, and thrusting the mandible forward to insure an optimal airway and thus permit evaluation of spontaneous respiration. Since respirations were no longer evident, an Ambu-bag with full face mask was immediately applied to provide positive pressure ventilation in order to overcome the narcotic-induced apnea. One hundred percent oxygen was connected to the Ambu-bag assembly, to supplement the inspired concentration of oxygen. Simultaneously, naloxone hydrochloride (Narcan) 0.2 mg, a specific narcotic antagonist was administered by deep IM injection into the anterior thigh. Heart rate and blood pressure remained stable throughout the apneic period.

At this point, it must be emphasized that mechanical support of airway patency and ventilation was instituted as the first emergency measure. The administration of a narcotic antagonist must not take place until these measures have been successfully instituted. Under no circumstances should the administration of such an agent be used in lieu of mechanical respiratory support.

Within five minutes of the administration of the naloxone, respiratory depression was completely reversed and positive pressure ventilation was discontinued. Although the child quickly regained consciousness, he remained in a suitably sedated state for continuation of dental treatment. The dentist, however, elected to place a temporary restoration and continue treatment at another appointment. The child was retained for observation for an additional sixty minutes, and then dismissed in an alert and oriented state with vital signs stable and within normal limits.

DISCUSSION

This case is typical of narcotic overdose. Extreme somnolence with intermittent airway obstruction progressing to frank respiratory depression characterized by decreased respiratory rate and/or tidal volume is a classic presentation.[1,2] The sequence of events is not unpredictable when narcotics are administered to excess. Conversely, they are to be expected, when doses reach either relative or absolute overdose levels. Recognition of a predictable sequence of events, combined with effective monitoring and use of emergency equipment, can easily prevent such a predictable reaction from becoming a tragedy.

Prompt recognition of subtle changes in clinical signs of a sedated child requires diligent observation, or depression of reflexes and loss of consciousness may go unnoticed. Goodson and Moore emphasized the importance of adequate monitoring in order to prevent

or detect adverse reactions to premedications. They reported changes in respiratory functions to be the first changes noted in all cases reviewed. Due to the variation in onset of action following submucosal injection of alphaprodine, they further warned of the possibility of a delayed overdose reaction taking place, when this route of administration is employed.

Continual patient observation must take place once treatment has begun, particularly for the presence of consciousness. At no time may the patient be permitted to lapse into an unresponsive state. With the production of unconsciousness, upper airway obstruction is invariably produced. If the obstruction continues, hypoxia and hypercarbia (the combination is called asphyxia) will rapidly ensue. If the adverse reaction is not adequately managed by this point, the risk of circulatory collapse, cardiac arrest and death is present. The early recognition and prompt intervention described in the case presented were undoubtedly responsible for its uneventful outcome. Note that the use of only a pediatric blood pressure cuff, a precordial stethoscope and continuous observation of the patient provided all the essential monitoring required. Although additional medications, intravenous administration supplies, EKG monitoring, etc. were readily available, minimal office emergency equipment used in conjunction with an adequate level of knowledge was sufficient to manage this reaction.

The duration of respiratory depression following such a reaction is dependent on the initial dose of alphaprodine and the susceptibility of the patient to the drug. Respiratory depression may exceed the duration of action of naloxone. Thus following the administration of the narcotic antagonist, continued monitoring is essential, in order to identify promptly a second bout of respiratory depression. A repeat injection of the reversing agent may be necessary.

A variety of clinicians have repeatedly stated that no universally accepted standard dosages for alphaprodine exist for children.[2,3] Higher basal metabolic rates, various environmental factors and even the differing nature of the dental procedures have all been suggested as factors to be considered, when selecting the proper dose. For pediatric dentistry, the manufacturer of alphaprodine recommends 0.3-0.6 mg/kg by submucosal route only.[1] Tobias, Lipschultz and Album reported an effective dose range between 0.55-1.0 mg/kg, mean 0.78 mg/kg, with no adverse reactions, when simultaneously injected with a mean dose of 0.33 mg/kg promethazine (an antihistamine, included to potentiate the narcotic effects while minimizing nausea and vomiting).[3] In the same article they proposed a mean dosage of 0.95 mg/kg administered with promethazine to be the most efficacious.

Creedon advocated a dose of 0.5 mg/lb (1.1 mg/kg) without promethazine for children under six years of age.[4] Corbett utilized a single injection of 6 mg alphaprodine plus 1.25 mg promethazine for all the patients in his study, independent of body weight.[5] Still other variations have been reported.[6] It should be noted that promethazine as well as other psychosedatives and hypnotics, not only potentiate the therapeutic effects of alpha-prodine but also potentiate the central respiratory depressant effect.[2] All of the regimens, regardless of proposed doses or combinations, share the potential of producing an adverse reaction characterized primarily by respiratory system dysfunction.

A case has been presented to demonstrate the expected sequence of adverse events, following the excessive administration of alphaprodine to a pediatric dental patient. Special emphasis has been placed on the importance of monitoring, in the recognition and management of respiratory depression. The judicious administration of alphaprodine combined with appropriate monitoring of the state of consciousness, as well as of the function of the cardiovascular and respiratory systems, affords a safe and efficient means of providing pediatric sedation.

Prompt attention to maintenance of a patent airway coupled with mechanical support of ventilation could avert disaster in almost all cases of narcotic overdose.

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Congenital micrognathia and microglossia: An experimental approach to treatment

Robert A. Boraz, DDS
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Micrognathia is the unusual smallness of the lower jaw, with recession of the chin.[1] There is a significant amount of knowledge concerning micrognathia in the literature. This problem has been identified as a major factor in a number of syndromes.[2] The etiology of micrognathia may be developmental, congenital or acquired. Treatment of this entity originally consists of maintaining the patient's airway and facilitating normal eating. Corrective measures may be taken later, and usually involve surgical intervention.[3-8]

Microglossia signifies unusual smallness of the tongue. Very little is known and very few cases have been reported of severe microglossia.[9] The cause is uncertain and the treatment is not defined. The tongue is derived from the first, second, and third branchial arches. The body and apex of the tongue originate in three prominences on the inner aspect of the mandibular or first branchial arch. The base of the tongue develops from a prominence formed by the union of the bases of the second and third branchial arches.[10] Some disturbance in this mechanism can be assumed, therefore, to be responsible.

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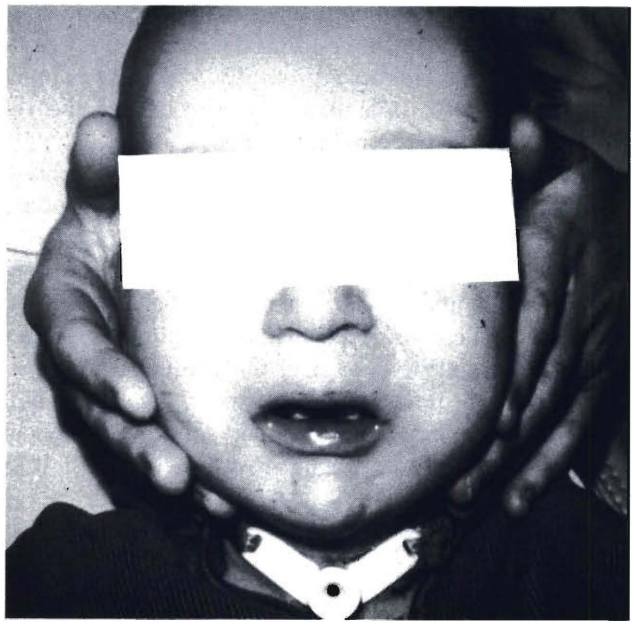
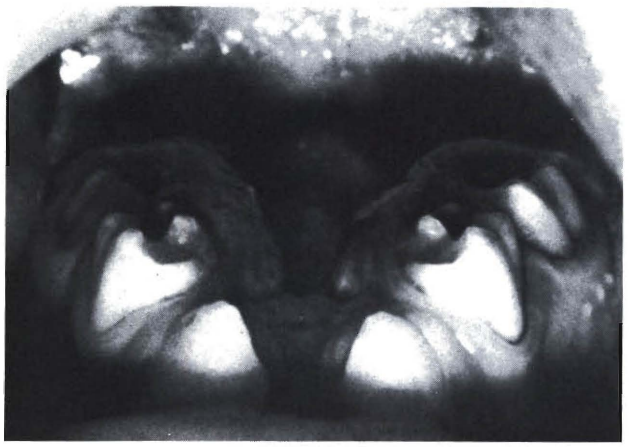
CASE PRESENTATION

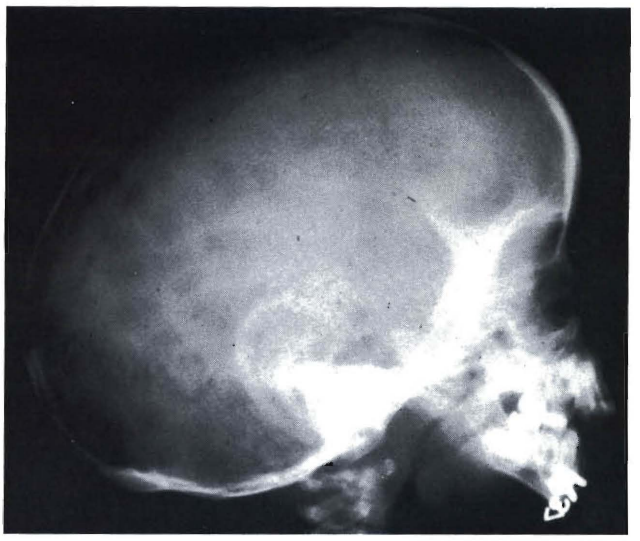
D. K. was born in Cheyenne, Wyoming, September 30, 1978. He weighed six pounds, one-quarter ounce, was nineteen and a half inches long, and had a head circumference of 34 cm. At birth, it was immediately apparent that he had an abnormally shaped head, suggestive of premature synostosis, micrognathia, a severely hypoplastic tongue and a small hypopharynx.

1. Photograph of patient demonstrating abnormal head shape, micrognathia, and tracheostomy.





2. Intraoral view of micrognathia and microglossia.



3. Cephalometric radiograph revealing hypoplastic mandible.

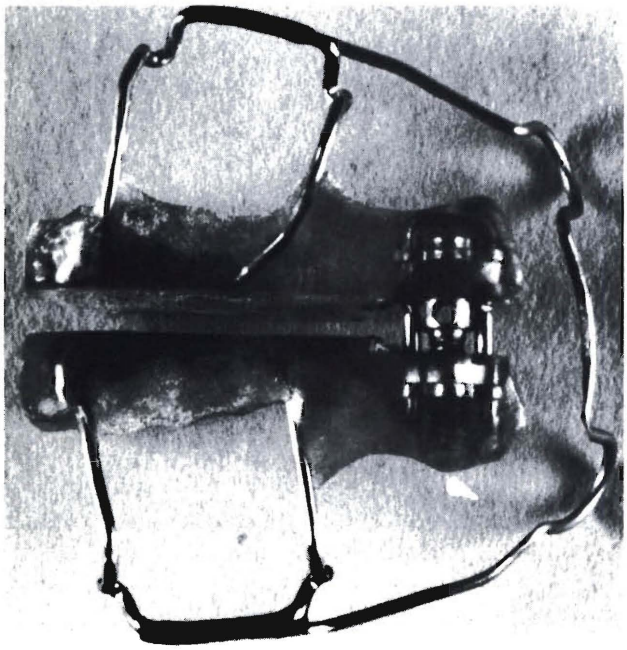
D. K. was a fullterm baby. He was the second child of a healthy twenty-one-year-old mother and a twenty-three-year-old father. The sibling was a normal female, three years older than D.K. The mother had no prenatal care, took no medications during pregnancy, drank no alcohol, and smoked one pack of cigarettes per day. Delivery was by caesarean section.

D.K. remained in the hospital for three weeks. He was then transferred to the University of Colorado Medical Center in Denver, because of difficulty in breathing. He remained at the Center through age eight months. At age four and a half months, a tracheostomy was performed to facilitate oxygenation. At age ten months, he underwent a sagittal suture release with plastic inserts. Geneticists were not able to determine a syndrome or cause for the multiple anomalies. A chromosome analysis was within normal limits. The patient returned to Cheyenne, Wyoming and remained hospitalized until age one year.

Shortly after this time, D.K.'s parents separated and the mother and child moved back to the mother's home in Parsons, Kansas. D.K. was enrolled in a preschool for the developmentally disabled. He was admitted to the University of Kansas Medical Center in Kansas City at age two years, four months, and underwent a thorough evaluation, under general anesthesia. The findings included severe micrognathia, a small, pedunculated mass for a tongue with papillae on the surface, a very small epiglottis, bilateral otitis media, and an inability to visualize the larynx. Neurologic and audiologic evaluations later revealed no abnormalities. A CT scan of the head was also normal.

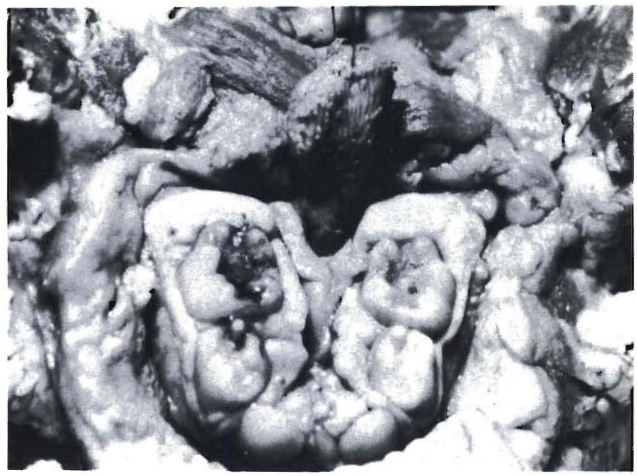
The dental evaluation revealed a greatly underdeveloped lower portion of the face and retruded chin. In crying, the muscles of the floor of the mouth were noticeable in their movement. The tongue was not functional and consisted of a rudimentary structure resembling a bud of a tongue. The maxillary arch appeared symmetrical, though small, with a thickening of the soft tissue covering the midpalatine suture. The bicanine diameter was 21.0 mm. The maxillary molars were slightly rotated, but a normal spacing pattern for all maxillary teeth was present. In the mandibular arch, there was extreme constriction of the alveolar process. The bicanine diameter was 3.5 mm and the bimolar diameter was 7.0 mm. The mandibular central and lateral incisors were missing. The bimaxillary relationship was one of extreme mandibular retraction with buccoversion of all maxillary teeth. Radiographs revealed the absence of all mandibular incisor teeth, primary and permanent. There was a general hypoplasia of the mandible and marginal alveolar atrophy. The gingival tissues were enlarged around most teeth, but especially pronounced on the lingual surfaces of the mandibular molar teeth. The oral hygiene was adequate and no evidence of dental caries was observed.

In accordance with Moss' Functional Matrix Theory of Growth, it was hypothesized that the extremely small mandible was at least in part due to the lack of pressure exerted by the rudimentary tongue. A plan was derived to attempt to stimulate the lateral growth of the mandible by exerting pressure with a modified mandibular Schwarz appliance. This appliance is similar to the jackscrew appliance utilized in rapid palatal expansion. This appliance, however, was to be utilized to exert lateral



4. Dental appliance used to stimulate lateral mandibular growth.

pressure and stimulate bone growth, not split the mental symphysis. If successful in obtaining lateral growth of the mandible, a mandibular advancement procedure would be performed at approximately age nine years. Alginate impressions were taken of the maxillary and mandibular arches. A modified mandibular Schwarz appliance was fabricated utilizing a custom made expansion screw, to treat the constriction of the mandibular alveolar arch. The patient was taken to the operating room and, under general anesthesia, three circummandibular wires were inserted to retain the appliance. The mother was trained to turn the screw on the appliance once a week. Due to her inability to comply, an orthodontist in the patient's home town agreed to adjust the appliance. Use of the appliance was discontinued approximately eight months later, when it was determined that maximum utilization of the appliance had occurred. The bicanine diameter was 5.7 mm, an increase of 2.2 mm; and the bimolar diameter was 9.0 mm, an increase of 2.0 mm. After a short respite, the intention was to fabricate a new appliance to continue the process. The patient, however, died before this was accomplished. D.K. took a nap in his preschool and never woke up. An autopsy revealed



5. Mandibular and alveolar arch at autopsy.

that the cause of death was a mucous clot completely obstructing the airway.

The case described utilized an experimental approach for treatment of an unusual combination of craniofacial anomalies. The hypothesis that the dental appliance fabricated in conjunction with the Functional Matrix Theory of growth could successfully expand the mandible in a lateral dimension was never fully tested. The initial results, however, were positive and merit further clinical evaluation in similar cases.

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A status report on diet, nutrition and cancer

Nutrition

T. Colin Campbell, PhD

CURRENT POSITIONS ON CANCER

Although investigations of a link between diet, nutrition and cancer can be traced back at least half a century, it is only within the last decade or two that a large number of such studies have been initiated in earnest. One of the first major conferences on this subject was held in 1975; since then, a large number of conference proceedings and reviews have been published.[1-3]

A particularly influential review was that of Doll and Peto.[4] These authors were commissioned by the Office of Technology Assessment of the U.S. Congress to evaluate the epidemiological evidence in support of a relationship of environment and cancer. They identified several types of environmental factors and concluded that on the basis of the literature they reviewed, diet could account for 10 percent-70 percent of cancer and, as a result, possibly be the most important environmental cause of human cancer. However, such a broad range in percentages indicated considerable uncertainty within the research community and/or the inability to estimate accurately the proportion of cancers caused by dietary practice. Other significant environmental causes included tobacco exposure (25 percent-40 percent), occupation (2 percent-8 percent) and alcohol (2 percent-4 percent).

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Shortly following the completion of the review by Doll and Peto, the U.S. National Academy of Sciences (NAS) was contracted by the U.S. National Cancer Institute to conduct an in-depth review of the research literature on diet, nutrition and cancer. An expert scientific panel was convened in 1980 and included experts in diverse disciplines, such as biochemistry, microbiology, embryology, epidemiology, experimental oncology, internal medicine, microbial genetics, molecular biology, molecular genetics, nutrition, nutrition education, public health and toxicology.

The first of its two reports was nearly 500 pages in length and summarized the panel's findings on the contributions of individual dietary constituents to cancer causation.[5] Rather than trying to estimate a specific proportion of cancers caused by diet, the panel concluded that "cancers of most major sites are influenced by dietary patterns."

The panel believed that any estimates of specific proportions of cancer caused by dietary practice could be misleading. The fact that the disease is multifactorial suggests there would be great difficulty in determining how much the clinical onset of cancer might be delayed or prevented by dietary practice, as opposed to the manipulation of other exposure conditions. Furthermore, the extent to which dietary, as well as nondietary, risk factors would operate in different individuals could also be highly variable.

On the basis of the evidence reviewed, the panel also found that it was possible "to formulate interim dietary guidelines that are both consistent with good nutritional practices and likely to reduce the risk of cancer." Although priorities were not assigned to the six interim guidelines, the two that will likely have the most impact in the marketplace were the recommendations 1) to reduce dietary fat intake from the current 40 percent-45 percent to 30 percent of caloric intake and 2) to emphasize "the importance of including fruits, vegetables and whole grain cereal products in the daily diet."

These recommendations generally conform with the U.S. Dietary Guidelines for Americans, which were previously published jointly by the U.S. Department of Agriculture and the U.S. Department of Health, Education and Welfare (now the Department of Health and Human Services).[6] The earlier recommendations were largely based on studies showing a relationship between dietary practice and risk of cardiovascular diseases. Since the release of the NAS report on diet, nutrition and cancer, similar dietary guidelines have been or soon will be published by the American Institute for Cancer Research, the American Cancer Society and the U.S. National Cancer Institute (NCI).[7-9]

CURRENT RESEARCH ACTIVITIES

Parelleling the explosive interest in this topic on the part of the public information agencies is a similar increase in research activity within the scientific community. The U.S. National Cancer Institute is now funding more than twenty human intervention trials to test the effectiveness of various nutrients in reducing cancer risk.[10]

In addition, there has been a sharp increase in the past five years in the number of investigator-initiated research projects now funded by NCI. The nutrients currently being studied by NCI include vitamins A, C and E and the trace mineral selenium. These intervention programs are organized so that appropriate population groups are enlisted and a careful analysis of the efficacy, dosage form and amount are undertaken before the study is begun.

It is too early to speculate on what type of data these studies will produce. One could speculate that, although a statistically significant positive effect may be produced, only a limited number of individuals particularly deficient in the study nutrient would respond. The implications of a negative result would also have to be evaluated with considerable caution since single nutrient effects may only comprise a small proportion of the total dietary effect and, as a consequence, such negative results should not discredit the general dietary theory, which considers multiple factors.

A new nonprofit organization, the American Institute for Cancer Research, was recently organized for the purpose of funding research and public information projects on diet, nutrition and cancer interrelationships. The American Cancer Society has embarked on a major survey of putative relationships between lifestyle factors and cancer incidence.

Another program has been initiated at Cornell University, Ithaca, NY, to study the effect of multiple dietary risk factors in the causation of selected cancers in the People's Republic of China with major funding being provided by the U.S. National Cancer Institute, the China Center for Preventive Medicine and the U.S. Food and Drug Administration. This study includes 130 survey sites in China and will attempt to delineate not only the contributions to cancer risk of individual dietary components but also the significant interactions that may occur between such components. Previous nutrition literature would suggest that such interac-

tions could be extremely numerous and substantial in their effects on cancer causation.

It is hoped by many that this activity and interest within the research and consumer communities will bring results within the coming decade that will enable the American public to make the appropriate modifications in their dietary practice to reduce cancer risk.

A PERSPECTIVE ON EVALUATION OF LITERATURE

Most reviews catalog and evaluate mechanisms that might account for the effects of various dietary substituents. In spite of numerous hypotheses of mechanisms, it is unlikely that a common mechanism will be found for all substituents or even for individual substituents. Probably the more relevant question for this review is whether the evidence supporting a causative effect of dietary substituents or food on cancer risk is strong enough to warrant the development of guidelines.

The strength of that evidence may be evaluated by examining the consistency of results within various human studies, within diverse animal studies, and between human and animal studies. From that perspective, there is considerably more strength of evidence within this literature than is initially apparent. For example, let us consider only the relationship between dietary fat intake and colon cancer risk within various epidemiological studies (i.e., do not consider vitamin A effect for sake of this example). Statistically significant results from such studies may exhibit either a positive relationship (as fat intake increases, cancer risk increases), an inverse relationship or no relationship. Those are the only three possibilities.

With this strategy, the multiple studies so far reported clearly show that as fat intake increases, colon cancer risk increases. Many of these studies show a positive relationship, some report no relationship and virtually none report a negative relationship. Furthermore, when that strategy is judged against a) the knowledge that epidemiological methodology is crude and will underestimate a real relationship and b) the knowledge that most of the studies reporting no relationship were undertaken in the more homogeneous population groups, the strength of the evidence between dietary fat intake and colon cancer risk becomes even more impressive.

A strategy of this type gives a new perspective to the evaluation of the nutrition and cancer literature. More consistency of data suddenly appears, as opposed to the inconsistency that would obviously exist if some studies were to show a positive relationship, some no relationship and some a negative relationship. When the literature is evaluated in this way for each nutrient, cancer risk increases with higher intake of dietary fat and protein and lower intakes of vitamin C, vitamin A (as beta carotene), vitamin E and dietary fiber.[5] This conclusion does not imply that we should expect every study or every population group to exhibit these effects, only that when there is an effect, it will be highly probable that the direction of the effect will be as indicated.

The most significant implication of these data is that a compilation of these nutrient effects strongly suggests that a diet enriched in plant products reduces cancer risk. A diet emphasizing fruits, vegetables and whole grain products, as recommended by the NAS report, would provide the proper intake for each of these nutrients. This recommendation to increase the consumption of plant products is in accord with similar recommendations on diet and other chronic diseases, such as cardiovascular disease and diabetes. Future research should be addressed to the dissection of mechanisms as well as to a more definitive understanding of the relationship between cancer risk and various levels of nutrient intake.

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Stress and nutrition

Deborah Kipp, PhD, RD

Stress is a well-known and frequently used, but ill-defined, term. It has different meanings for different people, depending on life experiences and health history. An interaction between nutrition and stress to preserve health is known, although much work needs to be conducted before a thorough understanding of the relationship is gained.

Changes in metabolism and requirements for calories and protein have been thoroughly investigated during the past decade and clearly defined with illness and injury.[1-4] Unfortunately, our current knowledge of vitamin and mineral requirements with these types of physical stress is inadequate to establish recommended levels for recovery.

Changes in the levels of circulating hormones have been demonstrated with emotional stress.[5,6] Knowledge, however, of the influence on metabolism and nutrient requirements is lacking.

In addition to physical and emotional stress, chemical stressors, such as tobacco, drugs and caffeine, may have an impact on nutritional requirements. Chemical stress factors will not be addressed at this time because they are beyond the scope of this paper.

STRESS

Primary communication networks within the body to coordinate and integrate metabolism and the maintenance of homeostasis include the nervous, hormonal and vascular systems.[7] The nervous system is the communication center, assessing the body's status relative to the environment (e.g., pain, thirst) and relays information for adjustment to these changes. The endocrine system rapidly adapts to maintain homeostasis of blood substrates, such as glucose, and regulates synthesis and degradation of substrates to meet energy needs. The vascular system delivers oxygen, transfers chemical messengers and eliminates toxins and metabolic end products.

Stress, as described by Selye, is the "nonspecific response of the body to any demand made upon it."[8] The nonspecific response refers to a generalized adaptation that must occur with stress to maintain physiologic equilibrium.

In addition to the nonspecific response, there are also unique, specific responses to various forms of stress. Depending on the stress, integrated changes in the nervous, endocrine and vascular systems occur to varying magnitudes and durations.[1-4] These changes, in turn, will influence the metabolism and, consequently, the requirements of nutrients.

The physiological response to a stress is dependent on the type, magnitude and number of stresses imposed, genetic predisposition, frequency of exposure, age and other conditioning factors.[8] Consequently, two individuals respond differently to the same stress.

It is generally assumed that a well-nourished individual is better able to cope with stress than a poorly-nourished individual. Whether this would be directly applicable to all forms of stress is not known.

Dubos suggested that almost any form of stress may

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upset nutritional balance.[9] If an individual is marginally deficient in a nutrient, the stress may exacerbate the condition. Undernutrition is in itself a stress and the body undergoes adaptations to maintain equilibrium and adequate functional status when only a limited supply of nutrients is available. When additional stress is then superimposed on the already adapted state, the individual no longer has the same reserve capacity to adapt to the stress.

NUTRITION AND PHYSICAL STRESS

Metabolic responses during injury and fever include increased stimulation of the nervous system and elevated secretion of many hormones, e.g., catecholamines and glucocorticoids. Since these physiologic changes influence nutrient metabolism, several characteristic nutrition-related consequences can occur. These include hypermetabolism, change in the production and utilization of glucose for energy, decreased protein economy and the loss of body weight and lean body mass.[1]

CALORIE AND PROTEIN NEEDS

An assessment of the change in metabolic rate in individuals with burns, trauma, surgery, fever, etc., is frequently obtained with gas exchange calorimetry. Hypermetabolism, measured as an increase in oxygen consumption above basal, reflects an increase in catecholamine-mediated activity in the body and a greater need for calories than in the pre-illness or injury state. For example, as the result of elective surgery, metabolic rate may increase by 5 percent to 10 percent and with severe trauma or infection, metabolic rate may increase by up to 55 percent.[1] Fever, by itself, causes an increase of up to 13 percent in metabolic rate for each degree centigrade of increase in body temperature.[3]

With an increase in plasma catecholamine and glucocorticoid levels, there is increased liver production of glucose but decreased peripheral uptake of the glucose. This results in a tendency toward hyperglycemia. These dynamics of glucose metabolism generally relate to the severity of the illness or injury.[1,2,4]

The hormonal changes accompanying physical stress also increase protein degradation, with amino acids becoming the primary energy source. The consequence of this is seen as an increase in the excretion of urine nitrogen. In addition, greater nitrogen losses would result from diarrhea and wound drainage. There is a close correlation between the magnitude of increase in the rate of nitrogen excretion and metabolic rate.[4]

Thus, protein and calorie needs are greater with illness and injury. For normal individuals, a calorie-to-nitrogen ratio of up to 350 calories for each 1 g nitrogen ingested will assure adequate calories to spare protein from being preferentially used for energy. With injury, the optimal ratio is between 100:1 to 200:1.[1] Both caloric and nitrogen needs are increased until the repair process is completed, with needs decreasing as the hormonal levels return to normal.

VITAMINS AND MINERALS

There are inadequate data available from which to establish increased requirements for vitamins and minerals. If there were a greater need for any vitamin or mineral with injury, it could be the result of inadequate absorption or utilization, or increased metabolism or excretion of the nutrient.[10] For example, decreases in intracellular ions, such as potassium, result from the loss of lean body mass.

Deficiencies in vitamins or minerals could be precipitated by inadequate ingestion coupled with greater requirements. Deficiencies in vitamin C and zinc are associated with poor wound healing.[11,12] The requirements for thiamin and riboflavin, which are based on caloric intake, would increase as the need for energy increased.[12] It is not known whether an additional increment of intake above this level is needed.

FOOD INTAKE

Unfortunately, food intake often tends to be inadequate to meet nutrient requirements during the acute phases of physical stress. This results from depressed intake, nausea and vomiting or food being withheld for surgery or diagnostic tests. Increased nutrient needs combined with decreased food intake can impair and prolong the recovery process. Pre-existing malnutrition or development of the malnourished state because nutrient needs are not met is a confounding factor in the recovery process that is associated with delayed wound healing, impaired immune function and increased morbidity and mortality.[1,4]

TEMPERATURE STRESS

Other physical stresses, such as changes in ambient temperature, also may influence nutrient requirements. Humans generally control heat loss and gain by

vasomotor regulation of skin blood flow and sweat rate without major alteration in the metabolic rate.[1] However, when the environmental temperature drops below about 20°C, the sympathetic nervous system is stimulated.[2] This accelerates heat production to maintain body temperature. The amount of heat produced in the body is regulated according to the needs of the body, as influenced by the environmental temperature. Non-shivering mechanisms are involved first; then, as the environmental temperature falls, shivering will occur.

Heat production causes a rise in basal metabolic rate and, therefore, an increase in caloric needs. There are no definitive studies demonstrating greater need for protein, vitamins or minerals with cold stress. On the other hand, decreased adaptation to cold, as measured by the ability of the body to regulate body temperature, has been reported with deficiencies of some nutrients, such as iron.[13]

There is not general agreement as to whether a hot environment also alters metabolic rate and, thus, energy needs.[14] Nevertheless, if the body's capacity to dissipate heat through sweating is exceeded, the body core temperature will rise. This will elevate metabolic rate, as seen with fever. In addition, profuse sweating results in water, sodium and nitrogen losses. Of less magnitude are losses in iron, calcium and magnesium.

NUTRITION AND EMOTIONAL STRESS

Since most hormones affect metabolism, stress could theoretically alter nutrient requirements when hormonal levels are altered.[7] The difficulty in conducting carefully controlled clinical studies to document this, however, can be well-appreciated. Quantifying and comparing the impact of stresses of life is problematic. One approach to quantification has been to identify the correlations of life's stresses with development of illness, with the impact of the stressful event measured by the readjustment required when the event occurs.[15] The contribution of a stressful life or "Type A" behavior on the risk of developing coronary heart disease (CHD) has been recognized.[16,17] Type A individuals tend to have an accelerated pace of life, competitive drive and severe sense of time urgency.[17] The role of stress in contributing to or aggravating gastrointestinal disorders is commonly accepted, although hereditary factors undoubtedly play a part.

The evidence to indicate an increased need for nutrients during stress is not well-established. Most evidence of increased nutrient needs or a "protective" role of nutrients with stress are anecdotal. There are, however, scattered reports of controlled observations regarding this issue.

Scrimshaw *et al*, reported greater variation in the excretion of urine nitrogen in college students during exams than during the pre-exam period.[2] This increase was not considered to be of sufficient magnitude or duration to warrant an increase in the Recommended Dietary Allowances (RDA) for protein.[12]

The nutrient most often associated with emotional stress is vitamin C. Stress-responsive tissues, such as the pituitary and adrenal glands, contain the highest concentrations of this vitamin in the body and adrenal vitamin C is decreased in response to stress.[11] Unfortunately, there has been only a single report of increased excretion of vitamin C in a subject participating in a metabolic study, when two fellow prisoners who were also metabolic study subjects escaped during the study.[18] This report, however, involved only one subject, was made as a secondary observation, and did not control other factors which may also have influenced the alteration in metabolism of the vitamin that was observed.

Compared to the inexact testimonials frequently given by the lay person, the existing scientific evidence does not support the claims of beneficial effects of consuming greater than the RDA level of nutrients to protect against stress. It is assumed that the RDA level for the nutrients includes an adequate margin of safety to accommodate possible transient changes in nutrient requirements that might occur with the stress of life.[12]

Eating habits may change. Some people eat less during periods of stress, while others tend to eat more. The result of this could either be overnutrition or undernutrition, obviously depending on the change in eating pattern. Consuming a well-balanced meal, even during periods of emotional stress, is crucial for health.

SUMMARY

Physical stresses of illness and injury result in clear increases in caloric and protein needs and perhaps vitamin and mineral requirements that remain elevated until the repair process is complete. Extremes in temperature have less effect on nutrient needs. Greater nutrient needs with emotional stresses have not been clearly documented, although changes in eating behaviors have been noted. Malnutrition, either pre-existing or developing as a result of the stress, is in itself a stress to the body. Thus, malnutrition may interfere with the process of adaptation to stress and impair the recovery process.

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DENTAL TREATMENT DURING AND POST PREGNANCY

Most dental procedures can be carried out with safety during pregnancy. Elective procedures should be provided but clinical sessions should not be unduly long. However, where there is reason to avoid certain procedures such as taking radiographs, and when a patient is unsure whether or not she is pregnant, these procedures should be deferred until the patient's condition has been confirmed.

Established programmes of preventive dentistry should continue throughout pregnancy and a plaque removal, prophylaxis and topical fluoride treatment should be performed regularly, for preference during the first and third trimester of pregnancy.

Where no regular preventive dentistry programmes have been commenced they should be started during pregnancy and continued after the confinement.

Where community water supplies are not fluoridated fluoride supplements should be prescribed. A daily supplement of 1 mg of fluoride ion is recommended from the fourth month of pregnancy.

Sedation employing nitrous oxide and oxygen, or intravenous diazepam should be avoided wherever possible. Radiographic examinations should be avoided wherever possible.

Soon after the birth of the baby the mother should have a thorough dental examination which may include a radiographic examination. A prophylaxis, plaque removal and topical fluoride application should be performed.

While the baby is being breast fed it should have a fluoride supplement (0.25 mg fluoride daily). When dietary milk is reconstituted or diluted with fluoridated water, a fluoride supplement should *not* be given.

Therapeutic substances taken by a mother who is breast feeding appear in milk at concentrations no greater than those of the mother's serum. The baby may receive trace amounts of these substances.

Where fluoridation does not exist fluoride supplements should be employed, consistent with National Health and Medical Research Council recommendations (82nd Session, 1976).

0-1 years: 0.25 mg fluoride ion daily
1-2 years: 0.5 mg fluoride ion daily
2 years and upwards: 1.0 mg fluoride daily

Mothers should be counselled against the use of sucrose containing vitamin C fortified syrups and other foods with high sucrose contents for their infants.

—Guidelines for dental treatment: dentistry and pregnancy. Statement from National Health and Medical Research Council.
Australian Dental J, 29:265–266, August, 1984.

Abstracts

Lowe, O. and Lindemann, R.: Assessment of the autistic patients dental needs and ability to undergo dental examination. J Dent Child, 52:29–35, January-February, 1985.
The purposes of this study were to determine the number of autistic patients who would allow a dental examination, to evaluate their dental needs, and to determine whether utilization of certain behavior management tools could be used successfully on the autistic patient, to achieve an initial oral examination and clinically acceptable bite-wing radiographs.

Forty patients were selected to participate: twenty autistic and twenty nonautistic age-matched controls. Ages ranged between three and thirty years. All patients were seen on an out-patient basis, histories were taken, the patients were examined clinically, their oral hygiene and plaque indexes were scored, and bite-wing radiographs were attempted.

The initial clinical examination was achieved in 50 percent of the autistic patients. The autistic patients had lower oral hygiene levels than the controls, but comparable caries indexes. Successful management of these patients for clinical examination and bite-wing radiography included reinforcing positive behaviors; utilizing the technique of tell, show and do; and using negative reinforcement when necessary.

Autistic patients, Behavior management

Enger, D.J. and Mourino, A.P.: A survey of 200 pediatric dental general anesthesia cases. J Dent Child, 52:36–42, January-February, 1985.
A total of 200 patients ranging in age from one to fifty-two years, from two different hospitals, received dental treatment under general anesthesia, during a five-year period. Patients were hospitalized for extensive dental decay, management problems, or because they were medically compromised. There was no significance between the choice of anesthetics, length of procedure, traumatic intubation and postoperative complications. The majority of problems which occurred postoperatively were nausea with vomiting, fever and sore throat in preschool children (one to five years old). Both hospitals reported significance in that more patients returned for a six month recall who lived within versus outside the city limits.

Anesthesia, general; Dental treatment

Ranta, R.: Impacted maxillary second permanent molars. J Dent Child, 52:48–51, January-February, 1985.
Over the past ten years, fifteen subjects with twenty-one maxillary permanent second molars impacted by the third molars were detected among orthodontic patients. Bilateral impaction was noted in six and unilateral in nine subjects. The impactions were distributed equally on the right and left sides and equally for both sexes. In fourteen cases, the third molar was removed between the ages of eleven and fourteen years. The eruption of the second molars was observed periodically and confirmed. In the remaining cases, six will be extracted later; one was observed without removal of the third molar, although eruption of the second molar was delayed, and in one case, at the age of eighteen years, the second molar was removed instead of the third molar. The recommended treatment is removal of the third molar early enough to allow a spontaneous eruption of the second molar.

Impaction, Second permanent molars