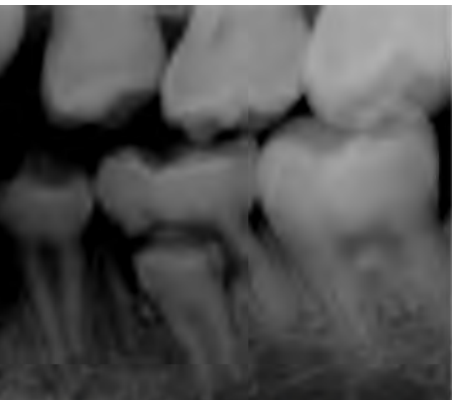
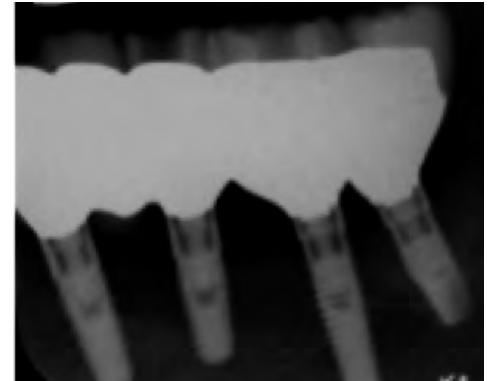
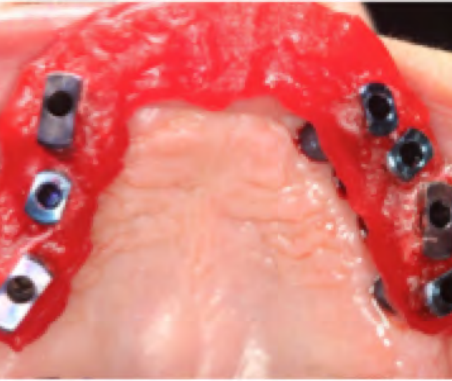
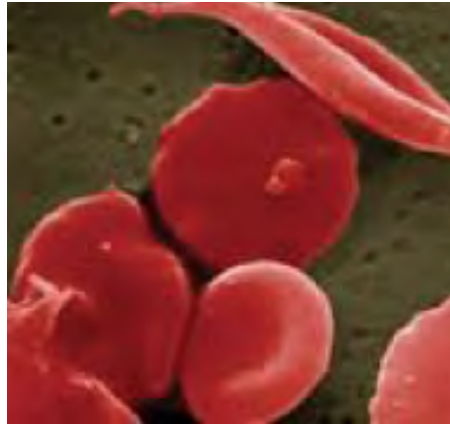


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COLUMBIA DENTAL REVIEW





COLUMBIA UNIVERSITY
College of Dental Medicine

COLUMBIA DENTAL REVIEW 2013-2015

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The Columbia Dental Review (CDR) is an annual publication of Columbia University College of Dental Medicine (CDM). This journal is intended to be a clinical publication, featuring case presentations supported by substantial reviews of the relevant literature. It is a peer-reviewed journal, edited by the students of the school. The editors are selected on the basis of demonstrated clinical scholarship.

Authors are primarily CDM students from pre-doctoral and post-doctoral programs, CDM faculty and residents, and attendings from affiliated hospitals. Peer reviewers are selected primarily from the CDM faculty. Submissions undergo a blind peer review system whereby the authors are not known by the reviewers (at least two per manuscript). Instructions for authors wishing to submit articles for future editions of the CDR can be found on the last page of this journal. Opinions expressed by the authors do not necessarily represent the policies of Columbia University College of Dental Medicine.

Editors' Note

Dear Readers,

I am delighted to welcome you to the 2013-2015 edition of the Columbia Dental Review. The College of Dental Medicine has a long history of producing excellent research, and the goal of the Review is to share some of the innovative and collaborative work that take place at our school. Thank you to our team editors for their hard work, and I hope you enjoy the issue.

Sincerely,
Alina O'Brien '17

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Comprehensive Oral Care in a Young Woman with Sickle Cell Disease and History of an Intracranial Hemorrhage

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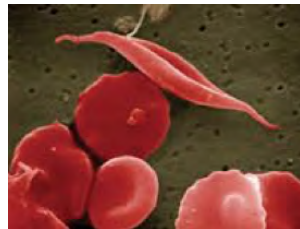
Abstract

It is estimated that 70,000 to 100,000 people in the United States have sickle cell disease (SCD), with an incidence of 1 of every 500 births in African Americans and about 1 of 36,000 births among Hispanic Americans. SCD was once considered a childhood disease, but now more than 95% of those affected survive beyond age 18, many into their forties, fifties, and beyond. SCD has been associated with a variety of oral and dental manifestations, although whether these are directly related pathogenically or due to socioeconomic factors is not always entirely clear. Planning and performing dental and oral surgical procedures in individuals with SCD presents unique challenges. The purpose of this report is to review basic facts about SCD that the dentist should know as well as specific considerations in caring for adult patients with this condition.

Introduction

Medical Considerations:

Hemoglobin (Hgb), the oxygen carrying protein of the blood, is a tetramer of 4 proteins, 2 α -globin chains and 2 β -globin chains encoded by genes on different chromosomes.¹ Patients with SCD have a mutation of the gene that codes for the β globin chains, a single nucleotide substitution that replaces a normal hydrophilic glutamic acid



Source: National Heart, Lung, and Blood Institute; National Institutes of Health; U.S. Department of Health and Human Services.

with a hydrophobic valine residue. The abnormal Hgb that is formed, called Hgb S, tends to polymerize when oxygen tension in the blood or the tissues is low, forming a rigid polymer inside the red blood cell (RBC) membrane. The RBCs also dehydrate, become inflexible and deformed, producing the characteristic "sickled" shape. These abnormal RBCs adhere to the endothelial cell lining of the blood vessel causing obstruction, called vaso-occlusion. The major clinical features of SCD are caused by vaso-occlusion, leading to ischemia of tissues, infarction, and injury to multiple organs, often accompanied by severe painful "crises." There is also vascular inflammation, endothelial damage, and increased RBC destruction leading to severe anemia. Only patients that are homozygous for the Hgb S gene have SCD. Patients that are heterozygous and have only one copy of the Hgb S gene have what is called sickle cell trait, a benign condition without anemia found in 8% of African Americans. Sickle cell trait confers some protection from malaria, which accounts for the high prevalence of the Hgb S gene among people of African descent, particularly in equatorial Africa

where malaria is endemic.²

Forty-nine states and the District of Columbia in the United States have mandatory newborn genetic screening for SCD, so most affected individuals born in the US will be detected at birth.² In most affected individuals, painful crises and progressive organ damage alternate with relative inactivity of the disease. Events that tend to trigger crises include infections, dehydration, stress, and extreme changes in temperature. Some of the more common complications of SCD disease include destruction of the spleen and an increased risk of infection, an enlarged heart from chronic anemia, skeletal deformities and growth disturbances, osteomyelitis and osteoporosis, and kidney disease. The acute chest syndrome is a potentially fatal condition with chest pain and lung damage that can be precipitated by infections or by surgical procedures. Cerebral vascular disease including hemorrhagic stroke affects more than 10% of people with SCD by 18 years of age.¹ SCD can also be associated with significant psychosocial problems due to frequent episodes of severe pain, hospitalizations, and physical disability.³

Effective treatments for SCD are limited, although the search for new approaches continues.⁴ Hydroxyurea is an anti-cancer agent that has been used for many years. It appears to reduce the production of Hgb S by inhibiting DNA synthesis, decreasing sickling.² During acute crises, the usual treatment is hydration and aggressive pain management; patients often require large doses of narcotics for pain control. In the past, most patients were given daily oral penicillin to reduce the chances of developing infections like pneumococcal pneumonia, the risk of which was increased because functionally they lack a spleen. More recently, with greater attention to vaccination, the number of patients receiving prophylactic penicillin is greatly reduced and often not used at all in the US in children over age 5.¹ The only curative treatment is bone marrow transplantation which is done before organ failure occurs if it is to be useful. A number of newer therapies are being investigated, including gene therapy, but these are not definitely shown to be beneficial.² The NIH summary statement available online at http://www.nhlbi.nih.gov/health/prof/blood/sickle/sc_mngt.pdf is an excellent resource for health professionals caring for patients with SCD (3).

Oral Manifestations:

The association between dental caries and SCD has been investigated in different populations. Several investigators have compared African American adults with SCD to

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controls.⁵⁻⁷ Patients with SCD tend to have a much greater prevalence of caries, but also tend to have lower social economic status, which may affect their access to care. There may also be a tendency for greater focus on their hematologic than on their dental condition, also affecting caries rates. Conversely, Fukuda and coworkers found a lower colonization rate with mutans streptococci and lower caries prevalence in pediatric SCD patients probably due to prophylactic penicillin therapy they received to prevent systemic infections.⁸ Whether this is still true now that penicillin is less widely used, at least in the United States, is not known. In summary, there are no clear data demonstrating that SCD actually predisposes to dental caries.

Therapies Under Investigation for SCD (2)
Xanthine oxidase inhibitors (byproduct of DNA metabolism)
Nitric Oxide generators (dilate blood vessels)
Statins (lower cholesterol, reduce vascular damage)
Monoclonal antibodies (anti-inflammation)
Vascular lubricants (help prevent RBCs plugging vessels)
Antifungal agents (prevent activation of leukocytes)

Similarly, there are conflicting reports regarding an association between periodontal disease and SCD.⁹⁻¹¹ While some authors have reported increased plaque index, gingivitis index, and even bone loss in patients with SCD, many others have found no significant difference between patients with SCD and controls.^{9,11} In one recent study, there was no difference in serum cytokine profile in children with periodontal inflammation regardless of whether or not they had SCD, suggesting that there was no direct immunologic relationship between SCD and periodontal inflammation.¹² Instead, like dental caries, gingivitis in patients with SCD likely results from socioeconomic factors, poor oral hygiene, and a focus on non-oral hematologic health issues.

Other oral conditions have been more directly associated with SCD. Luna and colleagues reported the prevalence of malocclusion to be 63% in preschool children with SCD and 100% in 12 to 18 year olds with SCD.¹³ The most commonly reported abnormalities are increased overjet, greater teeth angulation and incisor separation, prognathism, and diastemas. These malformations are thought to result from expansion of the bone marrow in both the maxilla and the mandible due to increased red blood cell production. Dental pulp necrosis that is unrelated to caries is another condition that has been repeatedly associated with SCD. In one recent study, pulp necrosis was 8 times more frequent in clinically intact teeth in patients with SCD as compared to

controls in the absence of trauma by two methods of pulp vitality testing.¹⁴ Sickle shaped cells are visible in tooth sections of dental pulp a few days after a sickle cell crisis. Plugging of the small vessels of the pulp chamber can lead to infarction and necrosis of tissue and even cause periapical lucencies on x-rays.² This can be associated with pain; toothaches are more common in patients with SCD than in normal controls, but pulp necrosis can also be painless.² Neuropathies have also been described that can affect any nerve but have been most frequently reported to involve the mental nerve and result in either loss of sensation or paresthesias of the jaw.¹⁶ Finally, osteomyelitis of the maxilla and mandible have been reported in patients with SCD, probably also as a result of necrosis and secondary infection.² A variety of organisms cause osteomyelitis in SCD: staphylococci and E. coli are most common in the jaw.¹¹

Table 2. Oral Manifestations of SCD	
Probably Directly Related to SCD	Probably Related to Socioeconomic Factors & Poor Oral Hygiene
Malocclusion, overjet, prognathism, diastemas	Increased Caries
Delayed eruption	Increased Periodontitis
Dental Pulp Necrosis/Toothache	
Radiographic laddering, calcifications, or lucencies	
Neuropathies (Mental Nerve)	
Osteomyelitis	

Case Report

A 26-year-old African American female presented to Columbia University Medical Center Dental Clinic for comprehensive dental care with a chief complaint of "I think I have a cavity." The patient's medical history was notable for sickle cell disease, diagnosed by screening at birth. She reported relatively mild painful crises that occur one to three times per year lasting less than a day. She has managed her pain mostly at home with intermittent use of prescription and over-the-counter analgesics; she denies chronic use of pain medications. Her last admission to the hospital for a painful vaso-occlusive crisis was in 1992. Her most serious complication of SCD occurred in 2010 when she developed a headache and was admitted to the hospital with a "brain hemorrhage." She was found to have had a subarachnoid hemorrhage, without evidence for an aneurysm. She recovered without residual neurologic deficits and has had no new CNS bleeding since that admission. There have been no other hospital admissions since 2010. She is considered to be functional without a spleen. Medications include folic acid that she takes once/day to aid in the production of new red blood cells. She takes Tylenol with codeine or ibuprofen as needed for pain.

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She has never been treated with hydroxyurea and has not required any recent transfusions. She was up to date on all immunizations and does not take penicillin or any other antibiotic routinely to prevent infections. She is allergic to latex and penicillin. Review of systems revealed occasional shortness of breath without a diagnosis of asthma or other lung disease. Her illness has not interfered with her healthcare: she has been compliant with her prior medical and dental care. On examination, her blood pressure was 136/80 mmHg and heart rate was 80 beats/min. Her dental history was notable from prior extractions of # 1, 16, 17, & 32. Her extraoral exam was within normal limits, there was no asymmetry, swelling, lymphadenopathy, or trismus. Her intraoral exam was also within normal limits. Oral cancer screening was negative. A periodontal exam revealed mild plaque-induced gingivitis. A restorative exam revealed staining, deep fissures, and plaque entrapment on the occlusal surfaces of #2, 3, 14, 15, 19, 30, 31. She had a peg lateral tooth at # 10 that had been built up with composite.

Her radiographs are shown in Figures 1 and 2. No active carious lesions were identified.

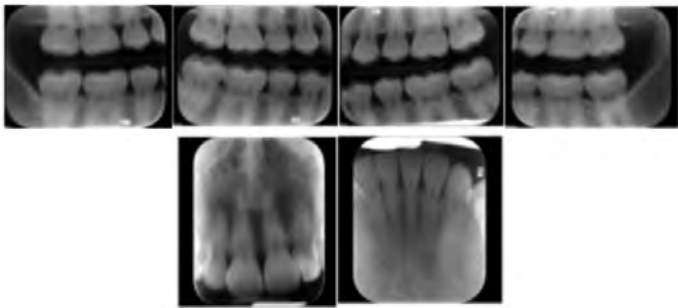


Figure 1 Molar and Premolar Bitewing and Central Incisor periapical views

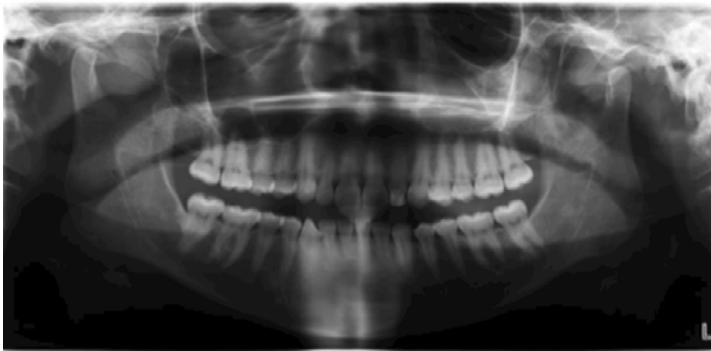


Figure 2 Panoramic radiograph

In developing a treatment plan, due to the presence of deep fissures on her molars, sealants were recommended for #2, 3, 14, 15, 19, 30. However due to financial considerations, she elected to only proceed with a cleaning and application of a sealant to #19, the stained tooth that she thought had been affected by caries.

Discussion

As more and more patients with SCD are living longer with their illness, it is increasingly likely that dentists, oral surgeons, and other oral health care providers will be providing care for adults with this disease in their practices. Unfortunately, there remains a lack of consensus regarding many of the more complex issues in managing patients with SCD during procedures. The following discussion addresses some of the more common questions the dentist is likely to face.

In general, routine dental procedures can be safely performed in the dental office between crises, even in patients with SCD. A complete medical history should be taken in every patient including a list of complications, current and prior treatment, transfusions, frequency of crises, and pain management. Because patients may have received many transfusions, their risk of blood borne infections such as hepatitis or HIV is increased and should be inquired about or tested for, if appropriate. It may be reasonable to obtain a medical consult early in the course of evaluation and treatment, particularly if more invasive or surgical procedures are contemplated.²

Although the risk of caries and periodontitis are not definitely increased in patients with SCD, because infections of any type can trigger painful sickle cell crises, they must be aggressively managed. This may include systemic antibiotics and/or rinses. Most authors agree that restorations are preferred over extractions but extractions can be considered if other approaches are likely to fail.¹⁷ Osteomyelitis is a more serious deep tissue infection that has spread to involve the bone. Treatment with antibiotics is required and surgery may be needed as well. In such cases, early consultation and/or referral to an oral surgeon seem appropriate.²

One controversial issue in the management of patients with SCD during dental procedures relates to the need for prophylactic antibiotics. As discussed above, some young patients may be taking prophylactic penicillin to prevent systemic infections even in the absence of specific procedures, although this will be less likely in adults that have received all recommended vaccinations. Currently published guidelines do not specifically recommend that antibiotics be given to patients with SCD specifically for dental procedures. Tate et al. (2006) surveyed pediatric dentistry residency program directors and pediatric hematologists regarding their use of prophylactic antibiotics for children with SCD during dental procedures.¹⁵ In general, there was a lack of consensus regarding the need for antibiotic prophylaxis for children with SCD among respondents to the survey. Responses also varied depending on the type of procedure to be performed and as

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to which antibiotic should be provided. The majority of dentists and hematologists felt that patients with heart disease or those undergoing extractions should receive prophylaxis, but most hematologists would only give penicillin, whereas amoxicillin was the drug of choice for most dentists. Those antibiotic choices held true across all responses. About half of the dentists and hematologists responded that prophylaxis was also indicated for people that were asplenic or being treated under general anesthesia; half would not give antibiotics to that same group of patients. Only a minority of respondents felt that prophylactic antibiotics were indicated for more minor procedures, but 15% of dentists and hematologist would give antibiotics to children with SCD even for tooth polishing. The problem results from a lack of data demonstrating that antibiotic prophylaxis is beneficial for patients with SCD undergoing dental procedures in the absence of definite signs of infection. Research is needed to provide clearer guidelines for the management of these patients.

A few other general guidelines have been suggested. There does not appear to be any reason to avoid local anesthetics or anesthetics containing vasoconstrictors even though vaso-occlusion is a known complication of SCD.² A recent retrospective review found that patients undergoing surgery could be successfully treated in the outpatient setting and without any special protocol.¹⁶ General recommendations included keeping the patient warm, warming all intravenous solutions prior to infusion, and maintaining good hydration and good oxygenation, which are recommended for all patients. Some authors recommend transfusing patients to a hemoglobin level of 10 mg/dl prior to surgery, although this recommendation is not based on the results of controlled

clinical trials.¹⁻² Finally, close attention to the patient's psychosocial history and family and social support network is indicated. Patients with SCD have a lifelong illness that is often punctuated with episodes of severe pain, hospitalizations, systemic complications, organ failure, and a shortened life expectancy. Not surprisingly, in some instances their illness and prior experiences with the health care system may have complicated their ability to obtain optimal care. It is important to discuss these issues with the person and their family and to take them into consideration when developing and implementing a comprehensive care plan.

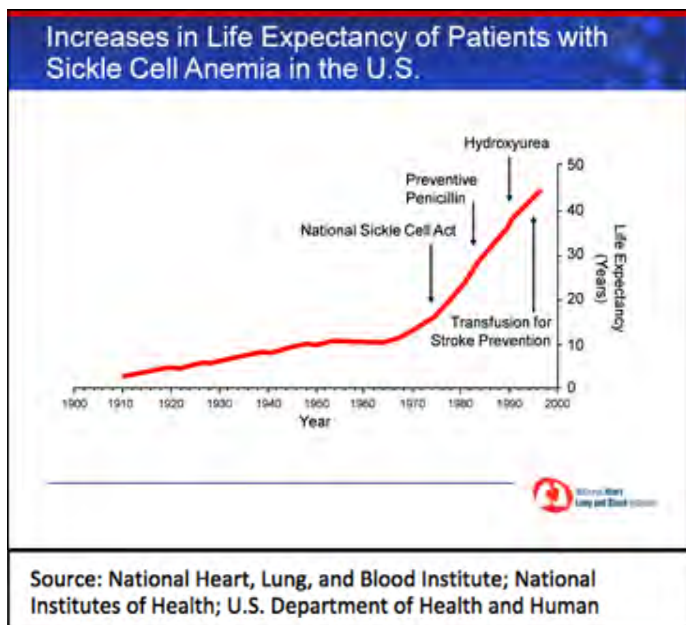
Conclusion

Sickle Cell Disease is the most common genetic hematologic disease. With modern treatment, most survive into their adult years and many dentists will care for people with SCD. Patients with SCD are at increased risk for periodontal disease, caries, malocclusion, pulp necrosis, and osteomyelitis. Despite their illness, most patients with SCD can be successfully treated in the dental office, can receive local anesthesia and can even undergo more invasive procedures, including extractions and oral surgery, as long as procedures are performed when they are stable and not during or shortly after painful crises.

Whether or not patients with SCD benefit from antibiotic prophylaxis for dental procedures is uncertain; most experts do not recommend prophylaxis for routine procedures such as cleanings.

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Gastrointestinal Disorder in a Patient with an Anterior Open Bite

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Abstract

An anterior open bite malocclusion poses challenges for both the patient and the orthodontist. An open bite prevents complete mastication of food prior to deglutination. Patients with masticatory dysfunction are more susceptible to gastrointestinal disorders.⁴ Patients seek treatment from an orthodontist to correct their open bite, in an effort to cure, or at least minimize, their gastrointestinal symptoms.

Introduction

The oral cavity is the entrance to the gastrointestinal system. Structures within the oral cavity, such as the teeth, tongue, and salivary glands, breakdown food and transport it to the stomach for further digestion. The muscles of mastication, which transfer force to the mandible and teeth, generate chewing force.³ Masticatory performance produces a high degree of variation in chewing force and strokes among the general population. "It is theorized that the insufficient breakdown of food and reduced exposure to saliva lead to inadequate pre-fermentation, impaired bolus formation, insufficient secretion of gastric juice acid and, finally, to digestive disorders."³ A recent study shows adults with class III malocclusion, which results in decreased bite force, occlusal contact, and masticatory efficacy, have more digestive complaints and gastrointestinal disorders.⁴ Likewise, an anterior open bite severely impedes biting-off function and mastication.³ Orthodontic treatment is indicated in patients with malocclusions, such as an anterior open bite, to increase masticatory efficacy and hopefully improve gastrointestinal disorders.³ Treatment of an anterior open bite requires a complete understanding of the etiology and accurate diagnoses. Etiologic factors contributing to an anterior open bite include: (1) abnormal skeletal development; (2) imbalances in the surrounding soft tissues and muscles; (3) malposition or displacement of anterior teeth; and (4) parafunctional habits.^{1,2} At the initial visit, complete diagnostic records are taken to establish a diagnosis and determine the etiology of malocclusion. These records typically include a complete medical and dental history, clinical examination, study models, intraoral and facial photographs, radiographs and cephalogram(s). Analyses of soft tissue and skeletal measurements performed on the cephalogram(s) are central to the diagnosis. Large skeletal deviations from the mean may indicate a need for surgical intervention.

Surgical correction of an anterior open bite overcomes the

restrictions posed by orthodontic treatment alone, allowing for larger corrective movements. Combined orthodontic and orthognathic surgical treatment manages the etiology of the malocclusion, and establishes a harmonious maxillary/mandibular dentoskeletal relationship through the coordination and alignment of arch forms. Surgical method selection and degree of movement are highly dependent on the nature and extent of skeletal, dental, soft tissue, and functional discrepancies. Postoperative management and orthodontic retention is essential to maintain the corrections obtained from combined surgical and orthodontic treatment.²

The following case report demonstrates the use of orthodontics and orthognathic surgery to correct an anterior open bite in an adult patient with gastrointestinal dysfunction.

Case Report

The patient, a Caucasian female, 36 years of age, presented to the Columbia University Orthodontic Residency Clinic with the chief complaint, "I have an open bite and was told by my gastroenterologist that it has affected my ability to completely chew my food. I am also bothered by my crowded teeth and lisp." The patient discussed severe constipation only alleviated by laxatives and hydrocolon cleanses. Her gastroenterologist attributed the constipation to incomplete mastication of her food. As a result, the patient sought out correction of her anterior open bite and malocclusion to alleviate her gastrointestinal symptoms.

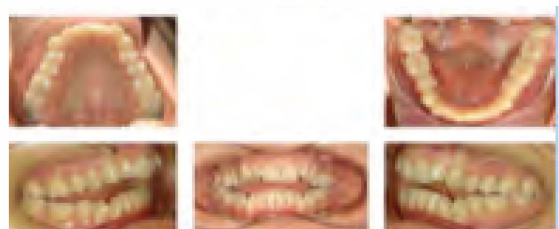


Figure 1 Initial Composite Records

Gastrointestinal Disorder in a Patient with an Anterior Open Bite

The patient reported routine dental care, previous orthodontic treatment, and denied any oral habits. At the initial visit, initial composite records were taken (Figure 1). Intraoral examination findings were noted: good oral hygiene, thin-scalloped pale pink gingiva, Class II right molar occlusion, super Class I left molar occlusion, one maxillary occlusal plane, two mandibular occlusal planes, maxillary midline coincident with the facial midline, mandibular midline 2 mm to the right of the maxillary midline, 3.5 mm overjet, and a 5 mm anterior open bite (Figure 1).

moderate crowding and a moderate Curve of Spee. As shown in Figure 3 and Table 1, the Bolton Analysis revealed slight maxillary overall and anterior tooth size excess. Space analysis confirmed crowding of 3.4 mm in the maxillary arch and 5.2 mm in the mandibular arch.

BOLTON ANALYSIS	Maxilla	Mandible	Discrepancy	Case Ratio	Ideal Ratio
Overall	95.1	85.2	-1.9	0.8951	0.9130
Anterior	47.3	35.5	-1.4	0.7495	0.7720

SPACE ANALYSIS	Available	Required	Difference
Maxilla	71.1	74.5	-3.4
Mandible	58.0	63.2	-5.2

TRANSVERSE DIMENSION	Arch Width Anterior	Arch Width Posterior	Canine Distance
Maxilla	31.5	45.6	32.7
Mandible	31.4	44.3	24.0

Tables 1a-c Bolton Analysis, Space Analysis, and Transverse Dimension

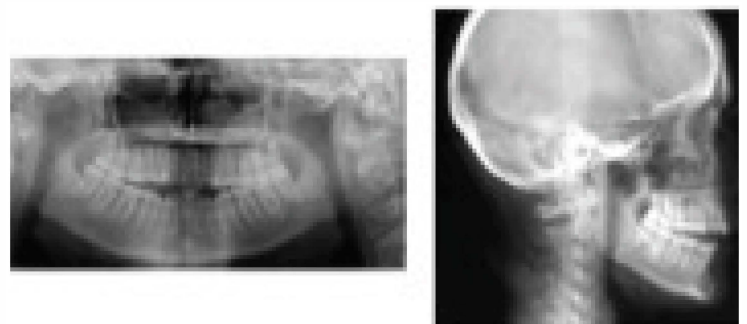
A frontal assessment revealed the patient's face to be mesofacial with an average width nose and competent lips. Her transverse fifths were equal, but she had an increased lower facial third and her chin deviated slightly to the right. Smile assessment revealed a 90% maxillary incisor display and <10% mandibular incisor display. The patient had a medium-broad smile with a flat smile arc, no gingival display, and <10% buccal corridor display. A <10% buccal corridor display indicates that there is minimal negative space between the corner of the mouth and the most posterior tooth visible during a smile. The profile assessment demonstrated a straight profile, slightly upturned nose, average chin-throat angle, average nasolabial angle (normal = 100-105 degrees), and upper and lower lip retrusion relative to the E-line (normal = lower lip on E-line and upper lip 1 mm behind E-line) (Figure 2).

The panoramic radiograph showed complete permanent dentition with fully erupted third molars and bone level, bone density, and trabeculation all within normal limits. A Columbia Analysis of the lateral cephalogram was performed and patient values were compared with mean values. Interpretation of the measurements indicated a Class II skeletal relationship, Class II denture bases, hyperdivergent denture bases producing a skeletal open bite, long lower anterior face height, retroclined maxillary incisors, increased interincisal angle, and retroclined, retruded mandibular incisors. Additional information obtained from a COGS analysis of the lateral cephalogram revealed a prominent chin, short anterior mandibular dental height, long posterior maxillary dental height, and short posterior mandibular dental height (Figure 4, Table 2).



Figures 3 Patient models

Evaluation of the patient's study models revealed a symmetric maxilla with a parabolic, tapering arch form, moderate crowding, and a mild Curve of Spee. The mandible had a symmetric, parabolic arch form with



Figures 4a-b Panoramic radiograph and cephalograph

Assessments and analyses from pictures, models, and radiographs were collected to create a problem list, establish treatment objectives, and finalize a treatment plan.

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COLUMBIA ANALYSIS				
Measurement	Value	Norm	Sig.	Interpretation
SNA (°)	83.6	81.0		
SNB (°)	78.2	78.0		
ANB (°)	5.4	3.0	*	Class II skeletal
Wits Appraisal	3.2	-1.5	*	Class II denture bases
SN - GoGn (°)	32.8	32.0		
(SN-PP) (°)	5.1	9.2		
(PP-GoGn) (°)	27.7	22.0	*	Hyperdivergent denture bases/ Skeletal open bite
V-Axis (SGn-SN)	68.5	67.0		
(S-Gn/N-Me) (%)	63.1	65.0		
(N-ANS/ANS-Me) (%)	68.2	80.0	*	Long lower anterior face height
U1 - SN (°)	88.9	103.5	**	Retroclined maxillary incisors
(U1-L1) (°)	151.5	130.0	****	Increased interincisal angle
L1 - GoGn (°)	87.1	93.0	*	Retroclined mandibular incisors
(L1-APs) (mm)	-2.0	1.0	*	Retruded mandibular incisors
L1 - NB (mm)	3.0	4.0		
Pog - NB (mm)	4.9	4.0		
Holdaway Angle (NB to H-line) (°)	6.1	8.0		
Holdaway Ratio (L1-NB:Pg-NB) (%)	0.6	1.0		

molar relationship, overjet of 3.5 mm, retroclined maxillary incisors, retroclined and retruded mandibular incisors, and multiple occlusal planes. The dental alignment concerns are the mandibular midline deviation of 2 mm to the right, moderate mandibular Curve of Spee, moderate maxillary crowding of 3.4 mm, and moderate mandibular crowding of 5.2 mm. Finally, in the transverse dimension, the soft tissue problem is the chin points to the right. These problem lists were referenced to establish the case's treatment objectives: correct the Class II skeletal open bite, achieve a Class I canine and molar relationship, achieve positive overjet and overbite, eliminate crowding in both arches, and retain the corrections (Table 3).

Problem List		Treatment Objectives
Vertical Dimension	Soft Tissue	•Correct Class II skeletal open bite
	Skeletal	
Anteroposterior Dimension	Skeletal	•Establish Class I canine and molar relationship
	Dental	
	Dental	•Achieve positive overjet and overbite
	Alignment:	
Transverse Dimension	Soft Tissue	•Eliminate crowding
	Skeletal	

Table 3 Problem list and treatment objectives

COGS ANALYSIS				
Measurement	Value	Norm	Sig.	Interpretation
CRANIAL BASE				
Ar-PTM (HP) (mm)	29.8	32.8	*	Short posterior cranial base
PTM-N (HP) (mm)	50.2	50.9		
HORIZONTAL (SKELETAL)				
N-A-Pg (°)	5.7	2.6		
N-A (HP) (mm)	2.0	-2.0	*	Protrusive anterior maxilla
N-B (HP) (mm)	-5.9	-6.9		
N-Pg (HP) (mm)	-1.6	-6.5	*	Prominent chin
VERTICAL (SKELETAL/DENTAL)				
N-ANS (Perp HP) (mm)	48.4	50.0		
ANS-Me (Perp HP)(mm)	79.0	80.6	**	Long anterior lower third facial height
PNS-N (Perp HP) (mm)	51.2	50.6		
Mand Plane - HP (°)	26.6	24.2		
U1 -NF (Perp NF) (mm)	28.7	27.5		
L1 -MP (Perp MP) (mm)	33.1	40.8	****	Short anterior mandibular dental height
U6 -NF (Perp NF) (mm)	24.3	23.0	*	Long posterior maxillary dental height
L6 -MP (Perp MP) (mm)	26.7	32.1	**	Short posterior mandibular dental height
MAXILLA, MANDIBLE				
PNS-ANS (HP) (mm)	49.5	52.6		
Ar-Go (mm)	46.4	46.9		
Go-Pg (mm)	78.8	74.3		
B-Pg (MP) (mm)	8.5	7.2		
Ar-Go-Gn (°)	118.8	122.0		
DENTAL				
OP-HP (°)	5.8	7.1		
A-B (OP) (mm)	-3.1	0.4	*	Class II
U1-NF (°)	93.9	112.5	***	Retroclined maxillary incisors
L1-GoGn (°)	87.1	95.5	*	Retroclined mandibular incisor

Tables 2a-b Columbia Analysis and COGS Analysis on the pre-surgical cephalograph

In the vertical dimension, the soft tissue problem is an increased lower facial height; the skeletal issues are hyperdivergent dental bases, skeletal open bite, and long anterior lower third facial height; the dental concerns are an anterior open bite of 5 mm and a lateral open bite from 6 to 6. The anteroposterior dimension skeletal problems are class II relationship, protrusive anterior maxilla, and prominent bony chin. The dental issues are class II right

Treatment to improve the patient's soft tissue, skeletal, and dental discrepancies was accomplished via pre-surgical orthodontics, orthognatic surgery, and post-surgical orthodontics. The pre-surgical orthodontic treatment sequence began with third molar extractions to minimize surgical interference. Orthodontic treatment was performed using a straightwire appliance system. Ceramic brackets were bonded to the teeth and a series of wires were used to level and align the dentition, alleviate crowding in both arches, and coordinate the upper and lower arches. Arch wires were built up to 19x25 SS with crimpable hooks for surgery.

The surgical plan included a two-jaw surgery. The surgical

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team performed a one-piece LeFort 1 osteotomy. This included 5 mm of posterior impaction and 2 mm of maxillary advancement to the maxilla. An intraoral vertical ramus osteotomy was performed to asymmetrically advance and rotate the mandible 1 mm to the left and allow for autorotation of the mandibular complex. Posterior impaction of the maxilla, followed by autorotation of the mandible, served to close the anterior open bite and achieve several millimeters of overbite. The maxilla was advanced slightly to compensate for autorotation of the mandible and establish a Class I molar and canine occlusion bilaterally, with proper overjet. In addition, the mandible was rotated asymmetrically to correct midline discrepancy and achieve a proper occlusal relationship. These movements also served to improve the soft tissue profile.

Minor orthodontics was required post-surgically, after healing. Pre-surgical orthodontics alleviated the crowding in both dental arches; however, it produced flaring of the mandibular incisors. During the surgical correction, the dentition was placed into Class I molar and canine relationships. Minimal overjet was present due to mandibular incisor flaring. Lower IPR was used to reduce flaring and increase overjet while maintaining molar and canine relationship achieved during surgery. Settling and detailing of the occlusion was performed. The patient is currently completing this phase of treatment. Post-orthodontic retention will include a lower fixed retainer, an upper Hawley retainer, and a positioner.



Figure 5 Superimposition of initial, pre-surgical, and post-surgical cephalogram tracings to demonstrate skeletal and dental changes

Discussion

The treatment plan addressed the patient's specific diagnoses with pre-surgical orthodontics, orthognathic surgery, and post-surgical orthodontics. The changes achieved from pre-surgical orthodontics and surgical treatment can be observed in the cephalograph tracings and superimpositions in Figure 5.

Pre-surgical orthodontics resulted in flaring of the incisors,

extrusion of the lower molars, and slight counterclockwise rotation of the mandible. The degree of flaring of the mandibular incisors is indicated by the Li-GoGn angle. Pre-surgical orthodontics moved the mandibular incisors from retroclined to a proclined and flared position, increasing the measurement from 87 to 109. The ideal measurement is 92. The excess flare resulted because 5.3 mm of crowding was alleviated without premolar extraction. To bring the Li-GoGn angle closer to 92, and to increase overjet, lower IPR was performed post-surgically. Another acceptable treatment option to alleviate the mandibular crowding of 5.3 mm is bilateral first premolar extraction. This option would provide better incisor angle position, but would finish with molar occlusion in Class III. In addition to Class III molar relationship, this option was not selected due to soft tissue considerations. The patient presented with a collapsed profile and lip position. Extraction of two mandibular premolars would exacerbate these soft tissue problems. Therefore, non-extraction treatment was chosen to improve lip position and avoid Class III molar occlusion.

The surgery produced the following skeletal changes: posterior impaction of the maxilla, counterclockwise rotation of the mandible, and slight changes to the mandibular body and ramus length. A Columbia analysis and COGS analysis of the post-surgical cephalogram confirmed many of these changes (Table 4).

The skeletal changes allowed for correction of the Class II skeletal open bite. Achieving a Class I molar and canine relationship by closing the anterior open bite and establishing appropriate interdental and interarch alignment, corrected the malocclusion. Interpreting research cited in the introduction, the improvement in occlusion can lead to improved mastication, either curing, or at least alleviating, the patient's gastrointestinal symptoms. However, improvement in chewing function after orthodontic and/or orthognathic intervention is controversial in the literature. Several studies have reported improvement in masticatory efficacy after treatment. However, other studies found that improvement in mastication took substantial time and never reached the level of untreated patients with normal occlusion. This time may be an adaptation period, in which the patient is adjusting to the new occlusion produced from orthodontic and/or orthognathic treatment.³

Different studies illustrate a controversy in the effectiveness of treating malocclusions to alleviate gastrointestinal disorders.³ Recent discussion with the patient revealed a self-reported improvement in her masticatory efficacy. She expressed an increased ability to completely chew her food following orthodontic treatment and orthognathic surgery. Additionally, she indicated diminished gastrointestinal problems, although not confirmed by her gastroenterologist. The patient said her constipation completely subsided, and that she no longer uses drugs or therapy to pass her bowels.

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The patient is pleased with her orthodontic and orthognathic treatment, reporting that it addressed her concerns and complaints.

malocclusion alleviated the gastrointestinal dysfunction. Although this case was successful, other case reports and studies have displayed controversial results. There is a need for more research on this topic to determine if orthodontic treatment to correct malocclusions can help to alleviate, or possibly cure, gastrointestinal disorders.

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4. Togawa R, Ohmure H, Sakaguchi K, Takada H, Oikawa K, Nagata J, Yamamoto T, Tsubouchi H, Miyawaki S (2009) Gastroesophageal reflux symptoms in adults with skeletal Class III malocclusion examined by questionnaires. *Am J Orthod Dentofacial Orthop* 136(1): 10.e1-10.e6.

COLUMBIA ANALYSIS				
Measurement	Value	Initial	Sig.	Interpretation
SNA (°)	81.8	83.6		
SNB (°)	79.9	78.2		
ANB (°)	1.8	5.4		
Wits Appraisal	-3.1	3.1		
SN - GoGn (°)	31.0	32.8		
(SN-PP) (°)	5.1	5.1		
(PP-GoGn) (°)	21.7	27.7		
Y-Axis (SGn-SN)	68.5	68.5		
(S-Go/N-Me) (%)	63.1	63.1		
(N-ANS/ANS-Me) (%)	75.3	68.2		
U1 - SN (°)	101.6	88.9		
(U1-L1) (°)	117.2	151.3	**	Decreased interincisal angle
L1 - GoGn (°)	109.0	87.1	**	Proclined mandibular incisors
(L1-APo) (mm)	4.3	-2.0	*	Protruded maxillary incisors
L1 - NB (mm)	8.5	3.0	**	Protruded mandibular incisors
Pog - NB (mm)	4.9	4.9		
Holladay Angle (NB to H-line) (°)	12.6	6.1	*	Increased facial convexity
Holladay Ratio (L1-NB:Pg-NB) (%)	0.6	0.6		

COGS ANALYSIS				
Measurement	Value	Initial	Sig.	Interpretation
CRANIAL BASE				
Ar-PTM (HP) (mm)	30.2	29.8	*	Short posterior cranial base
PTM-N (HP) (mm)	51.1	50.2		
HORIZONTAL (SKELETAL)				
N-A-Pg (°)	1.7	5.7		
N-A (HP) (mm)	-0.5	2.0		
N-B (HP) (mm)	-6.7	-5.9		
N-Pg (HP) (mm)	-2.5	-1.6		
VERTICAL (SKELETAL/DENTAL)				
N-ANS (Perp HP) (mm)	49.2	48.4		
ANS-Me (Perp HP)(mm)	64.0	70.0		
PNS-N (Perp HP) (mm)	47.6	51.2	*	Short posterior middle third facial height
Mand Plane - HP (°)	25.4	26.6		
U1 -NF (Perp NF) (mm)	29.6	28.7	*	Long anterior maxillary dental height
L1 -MP (Perp MF) (mm)	34.2	33.1	***	Short anterior mandibular dental height
U6 -NF (Perp NF) (mm)	24.5	24.3	*	Long posterior maxillary dental height
L6 -MP (Perp MF) (mm)	27.7	26.7	**	Short posterior mandibular dental height
MAXILLA, MANDIBLE				
PNS-ANS (HP) (mm)	49.8	49.5		
Ar- Go (mm)	41.3	36.4	**	Decreased mandibular ramal length
Go-Pg (mm)	82.3	78.8	*	Increased mandibular body length
B-Pg (MP) (mm)	9.2	8.5	*	Prominent chin
Ar-Go-Gn (°)	115.1	118.8	*	Decreased gonial angle
DENTAL				
OP- HP (°)	10.9	5.8	*	Steepened occlusal plane
A-B (OP) (mm)	3.1	-3.1		
U1-NF (°)	111.2	93.9		
L1-GoGn (°)	109.0	87.1	**	Proclined mandibular incisor

Tables 4a-b Columbia Analysis and COGS Analysis on the post-surgical cephalograph

Conclusion

Improvement in the patient's masticatory efficacy and gastrointestinal problems indicates the orthodontic and orthognathic treatment were beneficial. This report illustrates a case in which correction of a patient's

Indications for Splinting Implant Restorations: A Clinical Report

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Abstract

While the concept of splinting weak dentition is well documented and practiced, splinting of dental implant-supported prostheses is controversial in modern dentistry. Some research suggests that splinting implant restorations may be advantageous under certain circumstances, such as for short or narrow implants, crown-to-implant ratios greater than 1:1, and angled implants. This article reports a case of a patient who presented to the College of Dental Medicine with narrow implants of varying angulations in the position of teeth #18, 19, 20, and 22. This case details the subsequent restoration of implants using a splinted rigid FPD. The purpose of sharing this case is to explore the indications and management of splinting implant restorations.

Introduction

In modern dentistry, the concept of splinting weak dentitions together to support each other is a well-studied topic that is also commonly practiced. However, as we are relatively new to implant dentistry in comparison to treating natural teeth, several in vitro studies have reported conflicting results for splinting implant units in regard to minimizing the stress transfer to the restoration and supporting bone.¹⁻⁴

Initially, the concept of splinting implants originated from splinting teeth, where the assumption was that joined units improve the resistance to forces and alter the center of rotation.³ However, some argue that a concept that works on natural dentition cannot be transferred directly to implant dentistry due to differences in mechanics.⁴ Glantz et al documented unexpectedly high functional bending moments on implants on maximum biting and chewing in a conventional cross-arch splinted restoration. Vigolo and Zaccaria⁵ evaluated 144 splinted and non-splinted implants in 32 patients. The authors found no difference in marginal bone loss between the two designs.

However, splinting of implants may be indicated for short or narrow implants, crown-to-implant ratios greater than 1:1, angled implants, high loading forces, and immediate function.⁶⁻⁷

Case Report

A 90-year-old female patient was referred to Senior Clinic by external Periodontist for evaluation of restorative needs. The patient's medical history revealed that patient had been diagnosed with osteoporosis and received biannual

subcutaneous injections of Prolia® (Denosumab). Teeth #18 and #19 presented as implant retained restorations. A review of the dental history indicated that #18 and #19 implants were placed in January 2010, the implants were restored as splinted #18 and #19 likely due to angulation. In addition, further review revealed that implant fixtures for teeth #20 and #22 as well as "AlloOss" bone grafts were performed in July and September 2013, respectively. At this point, the amount of bone loss around implant fixture #20 was reviewed and diagnosed as 'restorable' by periodontists. If deemed 'non-restorable,' then the implant fixture would have to be re-implanted, or another restoration option presented to the patient.

Clinical and radiographic examinations revealed 4 narrow Nobel Select implants of varying angulations in the positions of teeth #18, 19, 20, and 22. Tooth #19 had an open margin at the interface between the implant and PFM cylinder with a possible fractured screw (Figures 1, 2).

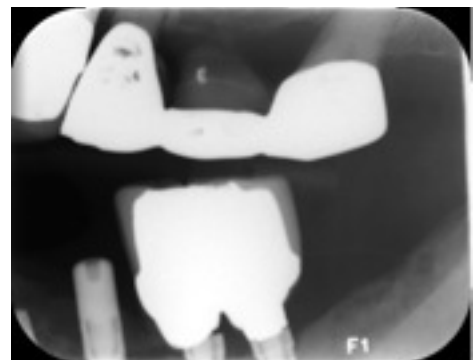


Figure 1 Patient radiograph reveals #19 with an open margin at interface between implant and PFM cylinder



Figure 2 #20 implant fixture with bone loss

Indications for Splinting Implant Restorations: A Clinical Report

Clinical Procedure

After gathering of preliminary data, clinical, and radiographic examinations, it was noted that 1) the fixtures were narrow, 2) the crown-to-implant ratio was greater than 1:1, and 3) the angulation of the implants was not ideal. After consultation with periodontists and prosthodontists, the decision was made to utilize all four implants and splint them to fabricate a 5-unit FPD (#18-19-20-X-22). It was felt that this treatment option would distribute forces more evenly than single tooth supported restorations. A screw-retained design was chosen for accessibility.

An open tray impression was taken of #18, 19, 20, and 22 impression copings (Figure 3). A framework was made from noble metal. Upon try-in, the framework had to be sectioned between #18 and #19, and #19 and #20-X-21 for passive sitting (Figure 4). The framework was soldered using GC pattern resin (Figure 5).



Figure 3 Open tray impression copings

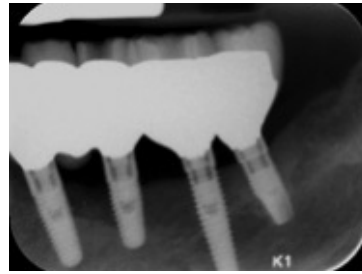


Figure 4 Framework sectioned between #18 and #19, and #19 and #20-X-21 for passive sitting



Figure 5 Framework was soldered using GC pattern resin

A glazed and finished PFM FPD was torqued to 35 N/cm. Screw holes were covered with nylon tape and composite. Occlusion was adjusted to ensure MI, even distribution of occlusion on FPD, and composite was out of occlusion (Figures 6, 7).



Figures 6, 7 Radiograph and clinical photograph of FPD #18-19-20-X-22 torqued in

A precise occlusal adjustment was made prior to delivery and torque, to minimize occlusal interference and to maximize correct force distribution. Group function was verified for laterotrusion, while anterior-guided posterior disclusion was verified during protrusion. (Figures 8, 9).



Figure 8 Group-function verified during laterotrusion



Figure 9 Anterior-guided posterior disclusion verified during protrusive movement

Implant fixtures, especially the ones with exposed surfaces, are under care of a periodontist, to try to establish and maintain soft-tissue attachment.

Discussion

A 90-year-old patient came to our clinic after treatment by a dentist outside the College of Dental Medicine. Her treatment plan was largely dictated by her pre-existing implant fixtures. It was decided that a splinted restoration would serve the patient better than single-unit implants given the non-ideal crown-to-root ratio and the size of the implants. Splinting of all teeth does pose a challenge in maintenance for most patients, as it is easier to maintain oral hygiene in single fixture restorations.⁸ Another advantage of non-splinted implants is the elimination of