ORIGINAL ARTICLE

Stability of bilateral sagittal split ramus osteotomy: Rigid fixation versus transosseous wiring

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Although many improvements have been made in orthodontic surgical procedures for mandibular retrognathism, relapse continues to occur. This study was designed to compare the stability of rigid and nonrigid fixation between 2 groups of patients who had undergone mandibular advancement surgery via sagittal split ramus osteotomy. Retrospective cephalometric measurements were made on 54 randomly selected orthognathic surgical patients. The patients, 7 males and 47 females, were divided into 2 groups: 28 patients stabilized by means of rigid fixation and 26 patients fixated with interosseous wires. The age of the patients ranged from 15.3 to 49.7 years. Lateral cephalograms were used to evaluate each patient at 3 distinct intervals: 7.0 ± 2.0 days before surgery (T1), 34.4 ± 15.0 days postsurgery (T2), and 458 ± 202 days after sagittal split osteotomy (T3). Eighteen linear and angular measurements were recorded and differences between the 3 time periods were evaluated. Statistical analyses were performed to assess the differences in the 2 fixation types between and within each group at different time intervals. The following measurements showed statistically significant skeletal relapse over time, for the *P* value .0028: Co-Go, ANS-Xi-Pm, IMPA, overbite, and overjet. The remaining variables showed no statistically significant relapse. The only measurement that showed a statistically significant group difference between T1 and T2 was DC-Xi-Pm. Results of the study led to the following conclusions: there was statistically significant relapse in mandibular length, lower anterior face height, mandibular arc, lower incisor inclination, overbite, and overjet in each group, regardless of the type of fixation. The potential was greater for relapse in patients stabilized with transosseous wiring. Although multifactorial, relapse in overbite and overjet may be a combination of skeletal and dental changes. (Am J Orthod Dentofacial Orthop 2000;118:397-403)

In nongrowing individuals, orthognathic surgery makes it possible to treat skeletal and dental dysplasias that are beyond the reach of orthodontic therapy alone. Since the introduction of the sagittal split procedure by Trauner and Obwegeser, this surgical procedure has gained significant popularity and is often the procedure of choice for patients with mandibular retrognathia. The potential for skeletal relapse, however, is of major concern. Some of the factors contributing to relapse include: condylar distraction from the glenoid fossa, posterior migration in response to soft tissue and muscle pull, lack of control of the proximal segment during surgery, and inadequate fixation periods or method of fixation. Magnitude of distal segment advancement, unfavorable postsurgical growth, pre-existing temporomandibular joint (TMJ) derangement, age at operation, condylar osteolysis and remodeling, and inadequate fixation between the proximal and distal segments also contribute to skeletal and dental relapse.[1-11]

During the last 15 years, many improvements have been made in orthodontic surgical procedures for mandibular retrognathism. Initially, surgical procedures used interosseous fixation of the bone segments with stainless steel wires. Today, a technique of rigid approximation using miniplates and various configurations of screws has been developed. Both fixation methods have brought new possibilities to the treatment of dentofacial and craniofacial deformities. The position taken by advocates of rigid fixation, that it changes bone healing and eliminates relapse, raises the question of whether rigid fixation has improved the stability of common orthognathic surgical procedures.

Stability of mandibular advancement procedures has been divided into short-term and long-term relapse. Several authors[1-3,8,9,12] have noted that relapse with wire osteosynthesis occurs during and soon after maxillomandibular fixation. The adverse movement of osseous structures after surgery could be linear, rotary, or a combination of both. When it occurs within 6 to 8 weeks of surgery, it is known as early relapse and is usually due to movement at the osteotomy site.[13]

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Table I. Demographic data for rigid and wire osteosynthesis

	Rigid fixation	*Wire fixation*	*Total sample*
Males	6	1	7
Females	22	25	47
Age of patient	26.7 ± 9.1	29.1 ± 9.9	27.9 ± 9.5

Early relapse has also been seen with rigid fixation.[4-6,14-16] Like wire fixation, large advancements are strongly associated with relapse. Gassman et al,[15] noted that early relapse occurred at the osteotomy site as a result of linear and rotary changes at the junction of the proximal and distal segments. It was assumed that large advancements were associated with increasing amounts of stretch on the surrounding soft tissue envelope and a smaller amount of bone at the interface between the segments. They suggested that early relapse could be prevented with skeletal wires and a period of maxillomandibular fixation.

Both animal and clinical studies have shown that relapse can be reduced by skeletal wires.[16-20] In 1980, Schendel and Epker[16] showed that skeletal fixation prevented osseous relapse in cases of wire osteosynthesis. In a similar study, Ellis and Gallo[17] reported an insignificant 8.9% horizontal relapse of pogonion during the period of fixation when skeletal wires were combined with wire osteosynthesis. Van Sickels and Tucker[21] studied 2 groups of patients undergoing large advancements. Both groups had 3 2-mm bicortical screws placed at the osteotomy site, but 1 group had additional skeletal wires placed for 1 week. This group demonstrated significant improvement in stability in cases up to 13 mm advancement.

Progressive condylar resorption, a significant factor in long-term relapse,[22-26] not only changes the shape of the condyle with a progressive loss in condylar height, but also decreases posterior facial height. With an incidence of 2.3% to 7.7%,[22,23] it has been found to occur in both single-jaw and double -jaw procedures where the sagittal split was stabilized with superior border wires, plates, and bicortical screws. Radiographic signs of relapse usually occur between 6 and 17 months after surgery.[22,27]

Several theories on the reasons for relapse have been proposed. Kerstens et al[23] suggested that surgery stimulates a process of increased bone loading on the joint. The process is thought to be initiated by disk displacement and immobilization. Arnett and Tamborello[28] suggested that mediolateral torquing or posterior displacement of the condyle with rigid fixation might be associated with condylar resorption and late relapse. In a study by Ellis and Hinton,[29] animals that had undergone sagittal split osteotomies and were fixated with either cortical screws or wires showed evidence of resorption on the posterior surface of the condyle and the anterior surface of the postglenoid spine. Animals in the study were first identified as having posterior displacement of the condyle, then sacrificed and analyzed for resorption. Ellis and Hinton stated that alterations in condylar position may result in or induce remodeling changes within the TMJ.

The amount of advancement and the presence of pre-existing temporomandibular derangements have been shown to have an effect on condylar resorption. Scheerlinck et al[22] noted that progressive condylar resorption was 4 times greater for advancements exceeding 10 mm when compared with advancements of 5 to 10 mm. In a similar experiment, Van Sickels[18] showed that in large advancements, additional stabilization with skeletal suspension wires showed increased stabilization after 6 months. New loading forces on the deranged joint as a result of surgery have been suggested as the cause of this progressive degeneration and relapse.[30]

The aims of the present study were to compare the stability of rigid and nonrigid fixation between 2 groups of patients who had undergone mandibular advancement surgery via sagittal split ramus osteotomy. Long-term skeletal and dental changes between the 2 groups will be the primary focus of the report, although short-term relapse will also be considered.

MATERIAL AND METHODS

A retrospective cephalometric study was performed on 54 randomly selected orthognathic surgical patients, with a mean age of 27.9 ± 9.5 years, who had undergone mandibular advancement to correct a Class II skeletal discrepancy. No other surgical procedure was performed. The demographic data of the sample are presented in Table I.

All patients were treated by one orthodontist and one oral surgeon, and the surgical technique was the same for all patients. Patients with pre-existing TMJ or muscular disorders were excluded from the study. All patients were placed into ideal occlusion without the use of intermaxillary splints. Patients receiving transosseous wiring were stabilized with circumrami wires and intermaxillary fixation for an average period of 46.3 ± 10.2 days. Patients receiving rigid fixation were stabilized with bicortical screws of varying lengths, depending on the need of the patient.

Skeletal changes brought about by the surgical procedure and the stability of the changes were evaluated with lateral cephalograms taken at 7.0 ± 2.0 days before surgery (T1), 34.4 ± 15.0 days postsurgery (T2), and 458 ± 202 days after sagittal split osteotomy (T3). Linear and angular measurements were recorded and differences between the 3 time periods were evaluated. The definitions of the landmarks, planes, and angles were taken from *An Atlas of Craniofacial Growth*[31] and were chosen to represent the changes in mandibular position over the time periods studied (Figs 1 and 2).

Statistical analysis was performed to assess differences in the 2 fixation types as well as to evaluate any correlation between the presurgical craniofacial morphology and the magnitude of relapse associated with the different fixation techniques. Means and standard deviations were recorded for 18 variables along with the change in each variable for both groups at the 3 different times (T1, T2, and T3). The significance of changes over the 3 points in time was determined by a repeated measures analysis of variance. In order to control for multiple independent tests and the inflation of experiment wide alpha, the desired significance level of 0.05 was reduced by a factor of 18. Therefore, each of the 18 tests used 0.05/18 or 0.0028 as the criterion for significance. The statistical errors of the cephalometric analysis were evaluated by duplicate determinations of 10 patients. These 10 patients were selected at random among those taken at T1, T2, and T3. Errors in landmark identification and tracing were evaluated statistically, suggesting a linear error of approximately 0.1 mm and an angular error of 0.5°.

RESULTS

The results of the cephalometric evaluation are given in Table II. Six of the 18 values showed statistically significant skeletal relapse over time (Co-Go, ANS-Me, ANS-Xi-Pm, IMPA, overbite, and overjet); 4 of the 18 values showed skeletal relapse, which may be of some clinical significance (Co-Gn, Co-Bpt, ANB, and Ar-Go-Me). None of the variables showed significant group differences based on type of treatment.

The remaining variables (SN-Pog, SN-Go-Gn, SN-Occl plane, SN-Ar-Gn, SNB, SN-lower 1, and lower 1 to NB) showed no statistically significant relapse and showed no significant group differences based on the type of fixation method. Only one variable, DC-Xi-Pm, showed significant group differences based on type of treatment , although no statistically significant relapse was noted.

Although the sample was selected randomly, the number of females included in the study far surpassed the number of males. Statistical analysis to determine whether this gender distribution affected the results of the study was performed and showed no correlation between gender and fixation type, or gender and variability in the relapse potential.

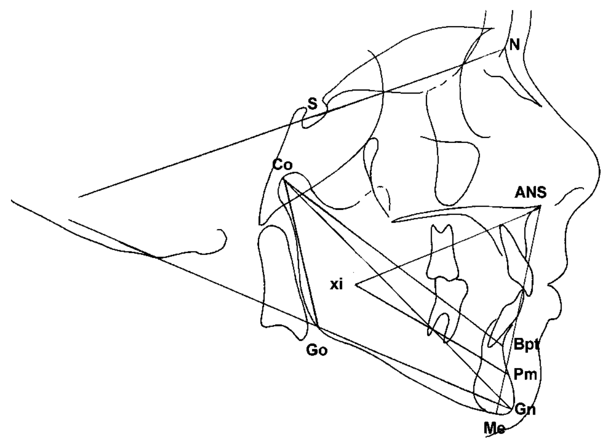

Fig 1. Cephalometric measurements used in the study.

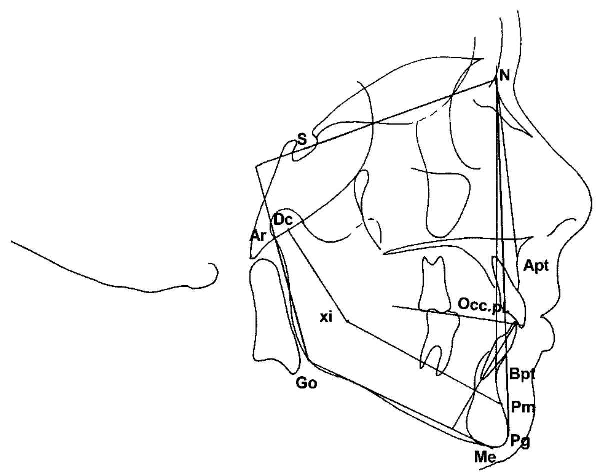

Fig 2. Cephalometric measurements used in the study.

DISCUSSION

For the majority of the values analyzed in this study, skeletal relapse was found to be an infrequent sequela of surgical correction. In contrast to previous studies, the results indicate that the extent and direction of skeletal change were predictable for most of the variables, with some individual variations in response to surgical treatment. The relapse seen in Co-Go, ANS-Me, ANS-Xi-Pm, IMPA, overbite, and overjet is statistically significant and noteworthy. Each variable will be discussed individually.

Changes of tooth position in relation to skeletal postsurgical relapse have been mentioned in numerous studies. Compensatory changes in the dentition are said to occur as the skeletal equilibrium is being reestablished resulting in such changes as remodeling of Apt

Table II. Skeletal relapse in patients treated with rigid and wire osteosynthesis

Cephalometric value	*Type of fixation*	*Mean (SD) value at T1 (presurgical)*	*Mean (SD) value at T2 (immediate postsurgical)*	*Mean (SD) value at T3 (long-term follow-up)*	*Mean change (SD)*	*P value*
Co-Gn (mm)	Rigid	120.79 (8.70)	125.54 (8.43)	124.91 (8.92)	–0.442 (1.819)	.005
	Wiring	116.15 (5.04)	120.79 (5.41)	119.96 (5.65)	–0.587 (1.870)	
Co-Go (mm)	Rigid	62.35 (6.75)	62.61 (6.14)	61.70 (6.95)	–0.644 (1.891)	.000*
	Wiring	58.28 (5.53)	58.20 (5.35)	57.00 (5.17)	–0.851 (1.843)	
Co-B pt (mm)	Rigid	106.25 (6.94)	111.55 (7.02)	110.86 (7.60)	–0.492 (2.020)	.007
	Wiring	102.16 (5.16)	107.85 (5.36)	106.89 (5.84)	–0.683 (2.346)	
ANS-Me (mm)	Rigid	68.64 (6.36)	70.92 (6.28)	70.00 (6.39)	–0.652 (1.427)	.000*
	Wiring	66.21 (6.26)	68.53 (5.77)	67.87 (5.69)	–0.468 (1.172)	
ANS-Xi-Pm (degrees)	Rigid	42.38 (4.70)	43.49 (4.21)	43.13 (4.51)	–0.258 (1.660)	.002**
	Wiring	41.69 (4.81)	44.43 (4.04)	43.42 (4.40)	–0.713 (1.485)	
DC-Xi-Pm (degrees)	Rigid	140.60 (5.04)	143.46 (5.67)***	143.61 (5.04)	0.106 (2.654)	.49
	Wiring	140.81 (5.80)	146.36 (5.34)	146.79 (5.56)	0.305 (3.451)	
SN-Pog (degrees)	Rigid	77.27 (3.67)	79.27 (3.61)	79.00 (3.80)	–0.189 (1.236)	.695
	Wiring	77.03 (3.11)	78.85 (3.13)	78.99 (3.61)	0.095 (1.250)	
SN-Go-Gn (degrees)	Rigid	31.10 (6.46)	32.79 (5.87)	33.20 (6.46)	0.290 (1.871)	.091
	Wiring	31.99 (5.87)	34.31 (5.64)	34.75 (6.08)	0.313 (1.758)	
Sn-Occl (degrees)	Rigid	16.32 (4.57)	16.54 (4.26)	16.50 (4.31)	–0.025 (1.138)	.164
	Wiring	15.83 (4.67)	15.99 (4.27)	16.63 (4.59)	0.454 (1.941)	
SN-Ar-Go (degrees)	Rigid	91.54 (4.87)	90.12 (5.16)	90.16 (5.40)	0.030 (2.114)	.383
	Wiring	92.41 (5.29)	88.60 (5.48)	88.08 (5.25)	–0.367 (1.839)	
SNB (degrees)	Rigid	75.46 (3.10)	77.88 (3.02)	77.61 (3.39)	–0.189 (1.364)	.554
	Wiring	75.08 (3.42)	77.37 (3.54)	77.44 (3.67)	0.054 (0.935)	
ANB (degrees)	Rigid	5.02 (2.04)	2.63 (1.65)	3.00 (1.95)	0.265 (0.909)	.004
	Wiring	5.79 (1.49)	3.44 (1.55)	3.73 (1.49)	0.203 (0.667)	
SN-L1 (degrees)	Rigid	49.96 (6.91)	50.14 (5.29)	50.46 (5.41)	0.227 (2.760)	.055
	Wiring	49.92 (7.68)	49.13 (6.80)	50.33 (7.01)	0.851 (2.955)	
L1-NB (degrees)	Rigid	25.36 (6.30)	27.77 (5.02)	27.05 (4.47)	–0.518 (2.367)	.056
	Wiring	25.01 (7.27)	28.14 (6.20)	27.47 (6.02)	–0.479 (2.914)	
IMPA[1] (degrees)	Rigid	97.76 (7.07)	97.09 (6.66)	96.16 (5.92)	–0.657 (2.364)	.000*
	Wiring	97.84 (8.51)	96.76 (7.61)	94.96 (8.04)	–1.270 (2.850)	
Ar-Go-Me (degrees)	Rigid	118.77 (6.89)	122.43 (7.17)	123.00 (6.95)	0.404 (2.530)	.028
	Wiring	119.62 (8.23)	125.00 (7.13)	126.64 (6.83)	1.159 (4.458)	
Overbite (mm)	Rigid	3.32 (2.08)	1.61 (0.91)	2.20 (1.14)	0.417 (0.794)	.000*
	Wiring	4.00 (2.19)	1.53 (0.74)	2.27 (1.31)	0.522 (1.067)	
Overjet (mm)	Rigid	6.16 (2.11)	1.48 (0.50)	2.09 (0.89)	0.429 (0.750)	.000*
	Wiring	5.77 (2.18)	1.37 (0.44)	1.79 (0.68)	0.299 (0.578)	

Standard deviation reported in parenthesis.
*Statistically significant relapse, no difference based on type of fixation, $P = .000$.
**Statistically significant relapse, no difference based on type of fixation, $P = .002$.
***Group differences based on type of fixation, $P = .002$.

and Bpt, proclination or retroclination of the anterior dentition, and "settling" of the occlusion.[5,16,30,32-57] In this sample, a number of values showed both a combination of skeletal and dental relapse: the mandibular incisors showed a statistically significant retroclination (decreased IMPA), anterior face height decreased as measured by ANS-Me, the divergence of the oral cavity decreased as measured by ANS-Xi-Pm, and overbite increased significantly. This suggests that reorientation of the segments at the osteotomy site occurred without skeletal relapse, and the changes seen are more dental in nature. One could speculate that this "settling of the occlusion" would result in the observed changes. The precise final positioning of the teeth is determined by the contacts between the teeth themselves, in both the same and the opposing arches, which guide the teeth into a functioning gnathologic mechanism.[52] If any incline of any tooth interferes with the centric relation closure arc, the mandible would most likely shift to avoid the offending incline, thus shunting the mandible away from centric and increasing the chance for relapse, both skeletal and dental in nature.[44-47] The observed changes may be a response to compensate for a change in skeletal base relationships.[57-61] Changes in incisor position from postsurgery to deband have not been well documented in the literature. Most studies tend to ignore the amount of tooth movement necessary to complete orthodontic care. Before 1986, dental changes in patients having rigid fixation for mandibular advancement were not reported. From that time to the present, only vague references and opinions have been offered when analyzing postsurgical dental responses.[34,42,46,53] Recently, the clearest example of a correlation between occlusal changes and postsurgical stability was presented by Pike et al.[54,55] Their findings closely match those of this study and suggest that further investigation into this area is warranted.

A relapse toward the pretreatment overjet is a common finding in mandibular advancement surgery. Some of the contributors of relapse include: condylar distraction from the glenoid fossa, posterior migration in response to soft tissue and muscle pull, lack of control of the proximal segment during surgery, inadequate fixation periods, and method of fixation. Magnitude of distal segment advancement, unfavorable postsurgical growth, pre-existing TMJ derangement, age at operation, condylar osteolysis and remodeling, and inadequate fixation between the proximal and distal segments also contribute to skeletal and dental relapse.[1-11] As mentioned previously, part of the relapse tendency may be related to dental compensations as muscular and skeletal balance are slowly obtained in the patient. It should be noted that the observed relapse in overjet, although statistically significant, is clinically inconsequential. A change of 0.30 mm with respect to 1.37 mm from T2 to T3 in the wire fixation group, for example, may represent 17% relapse and hence be called statistically significant, but when compared with the original presurgical record of 5.77 mm, this is only 5% relapse. Most surgeons, orthodontists, and patients would be quite satisfied with such a small return to the presurgical condition.

Significant relapse was observed for Co-Go in both the rigid and transosseous wiring group. Although tracing error is a possibility, progressive condylar resorption or remodeling of the glenoid fossa is a frequent sequela of mandibular advancement surgery. Ellis and Hinton[29] observed Condylar remodeling subsequent to posterior positioning of the condyle in cases of rigidly fixated adult *Macaca mulatta* monkeys. After mandibular advancement, changes in the cartilage thickness in the posterior condyle and resorption of the posterior aspect of the condyle and anterior surface of the postglenoid spine were noted. The investigators concluded “that alterations in condylar position may induce remodeling changes within the TMJ.” Condylar remodeling after sagittal split osteotomy has been observed in human beings as well, and compression of the condyle against the articular fossa has been considered the most frequent and likely cause.[43,48,56] In our study, condylar and glenoid fossa remodeling could have resulted in statistically significant changes in the length of Co-Go. Interestingly, the Co-Gn and Co-Bpt showed no significant relapse potential. The apparent loss in vertical height of the posterior mandible (Co-Go) without horizontal loss as measured by Co-Bpt and Co-Gn would suggest changes primarily at the gonial angle, with minimal change or remodeling in the condylar head of the mandible. Repositioning of the mandible will also affect the mechanical advantage of the jaw musculature by changing the relationship of the bite positions to the condyle. Mandibular advancement will decrease the mechanical advantage of the adductors by increasing the length of the load arm. This repositioning of the skeleton-jaw relationship can induce remodeling changes in the gonial angle, reducing the effective posterior face height.[62,63] It is not known whether the remodeling changes seen at this time will be significant in the future and further investigation would be required.

Many authors[36-39,45,48,49,53,55,56] have noted that interfragment instability results in early relapse. Van Sickels and Richardson[4] demonstrated a markedly reduced horizontal movement during the first 6 weeks postoperatively at point B and pogonion in patients where rigid internal screw fixation was used. DeClerq et al[30] reported that in intermaxillary fixation patients, the ramus tended to incline immediately after surgery with gonion moving forward due to greater rotation of the proximal segment. They also found that patients fixated with screws maintained a presurgical gonial position better than those fixated with wires. Epker and Wessberg[3] stated that in intermaxillary fixation patients most skeletal relapse occurs during and immediately after the release of intermaxillary fixation. Linear and rotational changes occurring at the junction of the proximal and distal segments due to increasing amounts of soft tissue stretch and varying amounts of bony interface often cause a change in mandibular length or a change in gonial angle. They concluded that dental stabilization alone without control of the proximal segment of the mandible results in the greatest likelihood of skeletal relapse after surgical advancement. Prolonged skeletal stabilization with control of the proximal segment of the mandible is suggested as the only practical method currently available for ensuring maximum stability.[3,18]

In this study, there was excellent interfragment and gonial angle stability as measured by DC-Xi-Pm and Ar-Go-Me. However, as noted in previous studies, there was a greater potential for relapse in those patients stabilized with transosseous wiring. A greater tendency toward opening of the gonial angle was noted, and there was also a significant group difference in the orientation of the proximal and distal segment as measured by DC-Xi-Pm. Although statistically insignificant in this study, it may be of clinical significance in patients with steep mandibular plane angles and vertical clockwise growth potentials. This plasticity at the osteotomy site may not only contribute to relapse but may also contribute to altering the spatial relationships of the muscle insertions. According to a biomechanical model of jaw function,[26] the area of osteogenesis between proximal and distal segments is susceptible to bending moments of force during functional loading.

Muscle activity during the healing period of fixation produces a force couple with a rotary movement in the area of least resistance in the mandible, the sagittal osteotomy site.[56] Finn et al[64] used a 2-dimensional mathematical model and predicted an 11% decrease in mechanical advantage for both temporalis and masseter muscles after mandibular advancement surgery because of lengthening of the moment arm. They also noted that if the proximal segment rotated superiorly after sagittal split osteotomy, the insertions of the jaw adductors would be significantly altered, which in turn might alter jaw biomechanics or increase the prevalence of TMD symptoms.[2,4,64]

CONCLUSIONS

Results of the study led to the following conclusions:

1. Statistically significant relapse was seen in both groups regardless of the type of fixation, in the following measurements: Co-Go, ANS-Me, ANS-Xi-PM, IMPA, overbite, and overjet.
2. There was a statistically significant group difference between T1 and T2 in DC-Xi-Pm measurement, indicating an opening of this angle in the wire fixation group. No statistically significant relapse was observed long-term (T2-T3).
3. The relapse seen in both groups appears to be multifactorial in nature. The relapse in overbite and overjet may be a combination of skeletal and dental relapse.

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REFERENCES

1. Putnam GD, Bowman JPB, Tuinzing DB. Stability of the osteotomy site following bilateral sagittal split osteotomy: screw fixation vs IMF. Br J Oral Maxillofac Surg 1983;31:213-6.
2. Worms FW, Speidel TM, Bevis RR, Waite DE. Post-treatment stability and esthetics of orthognathic surgery. Angle Orthod 1980;50:251–73.
3. Epker BN, Wessberg GA. Mechanisms of early skeletal relapse following surgical advancement of the mandible. Br J Oral Maxillofac Surg 1982;20:175-82.
4. Van Sickels JE, Richardson DA. Stability of orthognathic surgery: a review of rigid fixation. Br J Oral Maxillofac Surg 1996;34:279-85.
5. Van Sickels JE, Larsen JA, Thrash WJ. Relapse after rigid fixation of mandibular advancement. J Oral Maxillofac Surg 1986; 44:698-702.
6. Van Sickels JE, Flanary CM. Stability associated with mandibular advancement. J Oral Maxillofac Surg 1985;43:338-41.
7. Throckmorton GS, Finn RA, William HB. Biomechanics of differences in lower facial height. Am J Orthod Dentofacial Orthop 1980;77:410-20.
8. Ayoub AF, Stirrups DR, Moos KF. Stability of sagittal split advancement osteotomy: single versus double jaw surgery. Int J Adult Orthod Orthognath Surg 1995;10:181-92.
9. Douma E, Kuftinec MM, Moshiri F. A comparative study of stability after mandibular advancement surgery. Am J Orthod Dentofacial Orthop 1991;100:141-55.
10. Will LS, West RA. Factors influencing the stability of the sagittal split osteotomy for mandibular advancement. J Oral Maxillofac Surg 1989;42:813-8.
11. Uhde MD, Sadowsky C, BeGole EA. Long-term stability of dental relationships after orthodontic treatment. Angle Orthod 1983;53:240-52.
12. Mommaerts MY, Hadjianghelou O. Positional changes after mandibular advancement by sagittal split osteotomies and wire osteosynthesis: do combined orthodontics and the Dal Pont modification of the buccal osteotomy contribute to long-term stability? J Craniomaxillofac Surg 1990;18:93-106.
13. Ellis E, Carlton DS. Neuromuscular adaptation after orthognathic surgery. Oral Maxillofac Surg Clin N Am 1990;2:811-30.
14. Kirkpatrick TB, Woods MG, Swift JQ, Markowitz MR. Skeletal stability following mandibular advancement and rigid fixation. J Oral Maxillofac Surg 1987;45:572-6.
15. Gassman CJ, Van Sickels JE, Thrash WJ. Causes, location, and timing of relapse following rigid fixation after mandibular advancement. J Oral Maxillofac Surg 1990;48:450-4.
16. Schendel SA, Epker BN. Results after mandibular advancement surgery: an analysis of 87 cases. J Oral Surg 1980;38:265-82.
17. Ellis E, Gallo JW. Relapse following mandibular advancement with dental plus skeletal maxillomandibular fixation. J Oral Surg 1986;44:509-15.
18. Van Sickels JE. A comparative study if bicortical screws and suspension wires versus bicortical screws in large mandibular advancement. J Oral Maxillofac Surg 1991;49:1293-6.
19. Mayo KH, Ellis E. Stability of the mandible after advancement and use of dental plus skeletal maxillomandibular fixation: an experimental investigation into *Macaca mulatta*. J Oral Maxillofac Surg 1987;45:243-50.
20. Ellis E, Reynolds S, Carlton DS. Stability of the mandible following advancement: a comparison of three postsurgical fixation techniques. Am J Orthod Dentofacial Orthop 1988;94:38-42.
21. Van Sickels JE, Tucker MR. Management of delayed union and nonunion in maxillary osteotomies. J Oral Maxillofac Surg 1990;48:1039-44.
22. Scheerlinck JPO, Stoelinga PJW, Bliijdorp PA. Sagittal split advancement osteotomies stabilized with miniplates. Int J Oral Maxillofac Surg 1994;23:127-31.
23. Kerstens HCJ, Tuinzing DB, Golding RB, Van der Kwast WAM.

Condylar atrophy and osteoarthritis after bimaxillary surgery. Oral Surg Oral Med Oral Pathol 1990;69:274-9
24. Phillips RM, Bell WH. Atrophy of mandibular condyles after sagittal split osteotomy. J Oral Surg 1987;36:45-9.
25. Crawford JG, Stoelinga PJW, Blijdorp PA, Brouns JJA. Stability after reoperation for progressive condylar resorption. J Oral Maxillofac Surg 1994;52:460-6.
26. Sesenna E, Raffaini M. Bilateral condylar atrophy after combined osteotomy for correction of mandibular retrusion. J Maxillofac Surg 1985;13:263-7.
27. Moore KE, Stoelinga PJW, Gooris PJJ. The contributing role of condylar resorption to skeletal relapse following mandibular advancement surgery. J Maxillofacial Surg 1991;49:448-60.
28. Arnett GW, Tamborello JA. Progressive Class II development: female idiopathic condylar resorption. Oral Maxillofac Surg Clin N Am 1990;2:699-705.
29. Ellis E, Hinton RJ. Histologic examination of the TMJ after mandibular advancement with and without rigid fixation: an experimental investigation into the adult *Macaca mulatta*. J Oral Surg 1991;49:1316-27.
30. De Clercq CA, Neyt LF, Mommaerts MY, Abeloos JV, De Mot BM. Condylar resorption in orthognathic surgery: a retrospective study. Int J Adult Orthod Oral Surg 1994;9:233-40.
31. Riolo ML, Moyers RE, McNamara JA, Hunter WS. An atlas of craniofacial growth. Center for Human Growth and Development, University of Michigan. 1974.
32. Douma E, Kuftinec MM, Moshiri F. A comparative study of stability after mandibular advancement surgery. Am J Orthod Dentofac Orthop 1991;100:141-55.
33. Kirkpatrick TB, Woods MG, Swift JQ, Markowitz NR. Skeletal stability following mandibular advancement and rigid fixation. J Oral Maxillofac Surg 1987;45:572-6.
34. Lake SL, McNeill RW, Little RM, West RA. Surgical mandibular advancement: a cephalometric analysis of treatment response. Am J Orthod 1981;80:376-94.
35. Reitzik M. Skeletal and dental changes after surgical correction of mandibular prognathism. J Oral Surg 1980;38:109-16.
36. Rubenstein LK, Strauss RA, Isaacson RJ, Lindauer SJ. Quantitation of rotational movements associated with surgical mandibular advancement. Angle Orthod 1991;61:167-73.
37. Kobayashi T, Watanabe I, Veda K, Nakajima T. Stability of the mandible after sagittal ramus osteotomy for correction of prognathism. J Oral Maxillofac Surg 1968;44:693-7.
38. Phillips C, Turvey TA, McMillian A. Surgical orthodontic correction of mandibular deficiency by sagittal osteotomy: clinical and cephalometric analysis of 1 year data. Am J Orthod Dentofac Orthop 1989;96:501-5.
39. Will LA, West RA. Factors influencing the stability of the sagittal split osteotomy for mandibular advancement. J Oral Maxillofac Surg 1989;47:813-8.
40. Astrand P, Ridell A. Positional changes of the mandible and the upper and lower anterior teeth after oblique sliding osteotomy of the mandibular ramii. Scand J Plas Reconst Surg 1973;7:120-9.
41. Barer PG, Wallen TR, McNeill RW, Reitzik M. Stability of mandibular advancement osteotomy using rigid internal fixation. Am J Orthod Dentofacial Orthop 1987;92:403-11.
42. Goz G, Joos V, Schilli W. Position of anterior teeth following surgical correction of prognathism. J Maxillofac Surg 1984;12:33-5.
43. Komori E, Aigase K, Sugisaki M, Tanabe H. Cause of early skeletal relapse after mandibular setback. Am J Orthod Dentofacial Orthop 1989;95:29-36.
44. Poulton DR, Ware WH, Baumrind S, Crane D. Surgical mandibular advancement studied with computer-aided cephalometrics. Am J Orthod 1979;76:121-35.
45. Thomas PM, Tucker MR, Prewitt JR, Proffit WR. Early skeletal and dental changes following mandibular advancement and rigid internal fixation. Int J Adult Orthod Oral Surg 1986;1:171-8.
46. Turvey TA, Phillips C, Zaytoun HS, Proffit WR. Simultaneous superior repositioning of the maxilla and mandibular advancement. Am J Orthod Dentofacial Orthop 1988;94:372-83.
47. Wisth P, Isaksen TS. Changes in the vertical position of the anterior teeth after surgical correction of mandibular protrusion. Am J Orthod 1980;77:174-83.
48. Cottrell DA, Suguimoto RM, Wolford LM, Sachdeva R, Ingrid YG. Condylar change after upward and forward rotation of the maxillomandibular complex. Am J Orthod Dentofacial Orthop 1997;111:156-62.
49. Ghafari J, Brin I, Kelley MB. Mandibular rotation and lower face height indicators. Angle Orthod 1989;1:31-6.
50. Isaacson RJ, Kopytov OS, Bevis RR, Waite DE. Movement of the proximal and distal segments after mandibular ramus osteotomies. J Oral Surg 1978;36:263-8.
51. Wade DB. Surgical-orthodontic stability in retrognathic patients: an implant study. Angle Orthod 1988;1:71-95.
52. Rekow ED, Worms FW, Erdman AG, Speidel TM. Treatment-induced errors in occlusion following orthognathic surgery. Am J Orthod Dentofacial Orthop 1985;88:425-32.
53. Harsha BC, Terry BC. Stabilization of LeFort I osteotomies utilizing small bone plates. Int J Adult Orthognathic Oral Surg 1986;1:69-77.
54. Pike JB, Sundheim RA. Skeletal and dental responses to orthognathic surgical treatment Angle Orthod 1997;6:447-54.
55. Caskey RT, Turpin DL, Bloomquist DS. Stability of mandibular lengthening using bicortical screw fixation. Am J Orthod Dentofacial Orthop 1989;96:320-6.
56. Lake SL, McNeill RW, Little RM, West RA. Surgical mandibular advancement. Am J Orthod Dentofacial Orthop 1981;80:376-94.
57. Durbin DS, Sadowsky C. Changes in tooth contacts following orthodontic treatment. Am J Orthod Dentofacial Orthop 1986; 90;375-82.
58. Kaplan H. Logic of modern retention procedures. Am J Orthod Dentofacial Orthop 1988;93:325-40.
59. Moyers RE. Moyers symposium. Ann Arbor:Center for Human Growth and Development, University of Michigan, Ann Arbor, Michigan; Feb 1984.
60. McLaughlin RP. Commentary on occlusion. Angle Orthod 1989;3:181-6.
61. McMillen LB. Border movements of the human mandible. J Prosthet Dent 1972;27:524–32.
62. Schendel S, Wolford L, Epker B. Mandibular deficiency syndrome, III. J Oral Surg 1978;45:364–77.
63. Will LA. Condylar position following mandibular advancement [MSD Thesis]. Seattle: University of Washington; 1982.
64. Finn RA, Throckmorton GS, Bell WH, Legan LH. Biomechanical considerations in the surgical correction of mandibular deficiency. J Oral Surg 1980;38:257-64.