Thyroid administration to reduce root resorption

The following three case reports are presented to illustrate the effects a thyroid supplement can have during orthodontic treatment.

Eric L. Loberg, DMD; Christer Engström, DDS, Odont Dr

Each of these patients received .5 gr Thyroid (Proloid, Parke Davis) daily during active orthodontic treatment. The thyroid supplement was discontinued after retainers were placed. None of the patients had a history of thyroid disease and each had an uneventful dental history and normal periodontal health. One patient experienced TMD symptoms. The maxillary incisors in all three cases required intrusion and lingual root torque, movements commonly associated with root resorption. Treatment for all three patients included full bonded single bracket edgewise appliances (.018 X.025) with Lang rotation levers and cervical facebows. The patients were treated nonextraction and were finished in ideal Class I occlusions. All radiographs were taken at the same laboratory by the same technician using the same equipment.

Case JM

Patient JM presented at 15 years with an Angle Class II, division 2 with crowding, a deep overbite and a 2.5 mm anterior slide (Centric Relation - Centric Occlusion) as well as significant symptoms of TMD (muscle pain and daily headaches). Active treatment time was 1 year 3 months and the interval between the initial and final radiographs was 2 years 2 months. The amount of incisor intrusion and lingual root torqure requiredcan be seen on the maxillary superimposition. Steiner's method of superimposition was used to determine tooth movement (the original NA line and the maxilla).

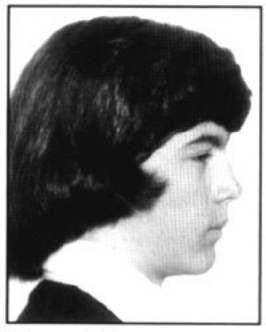


Figure 1A

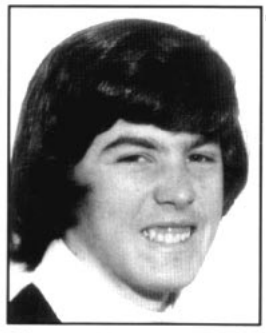


Figure 1B

Case JM
Figure 1A-B
Pretreatment facial photos.

Figure 2A-B
Posttreatment facial photos.

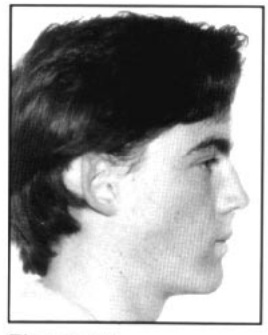


Figure 2A

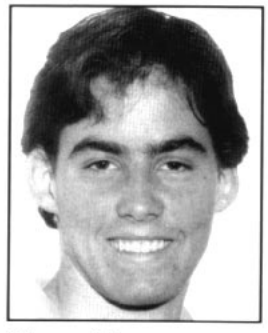


Figure 2B

Because of indications of root resorption at the apices of the maxillary incisors, .5 gr Thyroid (Proloid, Parke Davis) was prescribed during treatment. Extensive intrusion and lingual root torque were part of the treatment. However, there was no progression of the root resorption of the maxillary incisors. TMD symptoms were eliminated with treatment and JM has remained asymptomatic and pain-free.

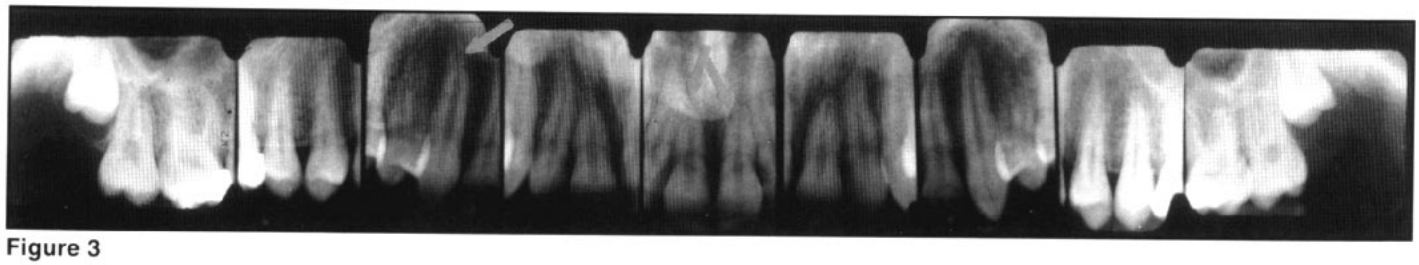


Figure 3

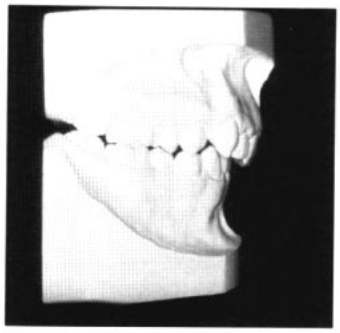


Figure 4A

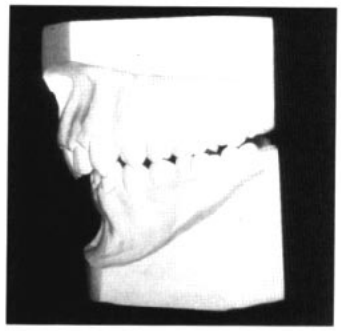


Figure 4B

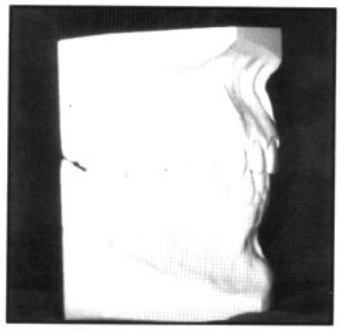


Figure 5A

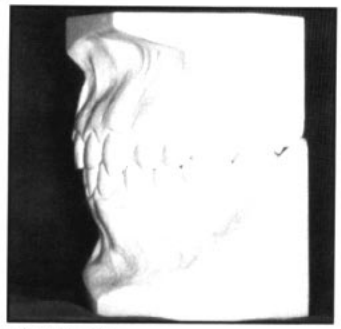


Figure 5B

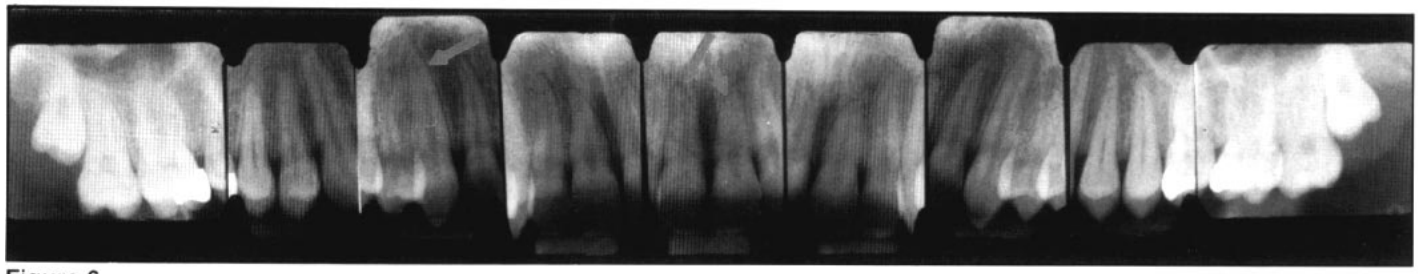


Figure 6

Case JM

Figure 3
Pretreatment periapical radiographs. Note "fuzziness" at the apices of the maxillary teeth.

Figure 4A-B
Pretreatment study casts.

Figure 5A-B
Posttreatment study casts.

Figure 6
Posttreatment periapical radiographs. No resorption of maxillary apices.

Figure 7A-B
Superimpositions show lingual root torque and incisor intrusion.

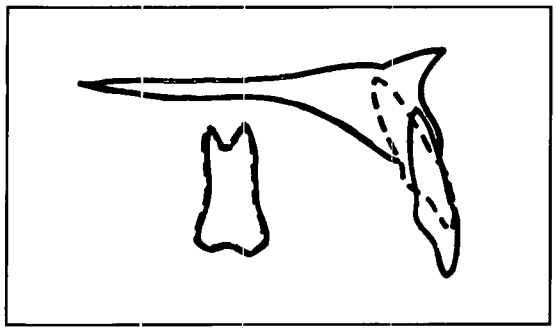


Figure 7A

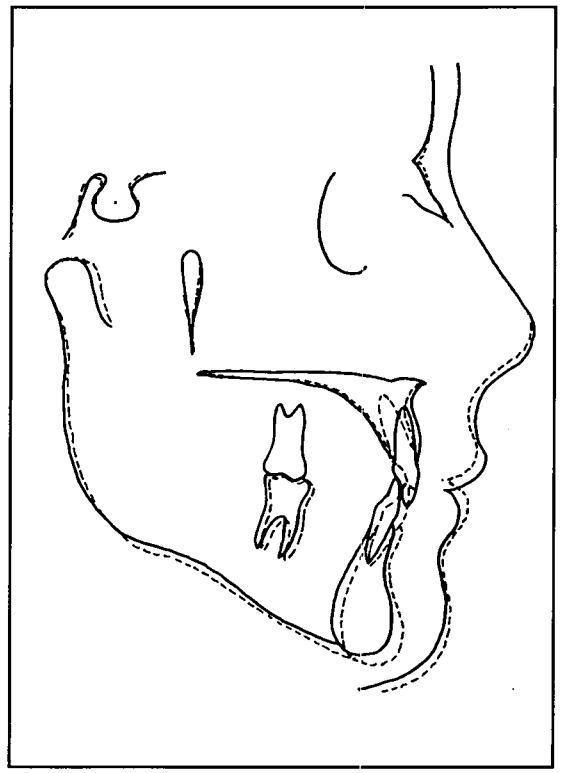
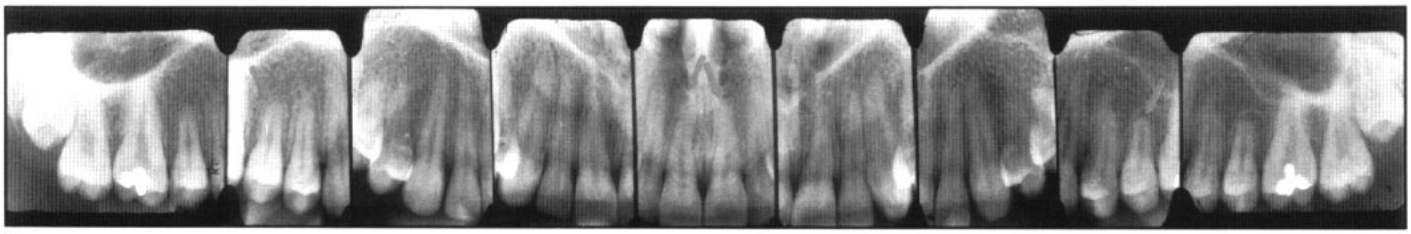


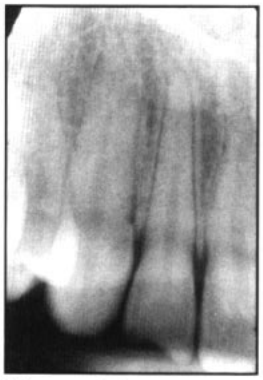
Figure 7B

Case NG

This patient presented at 16 years 6 months with an Angle Class I-II malocclusion with crowding and a deep overbite. The active treatment period was 1 year 2 months. The time between radiographs was 1 year 9 months. Initial periapical radiographs showed beginning root shortening on the maxillary incisors. The maxillary canines showed a lack of definition in the apical third and the maxillary second premolars already showed blunting and shortening of the roots. Because of the concern for continued root resorption during treatment, .5 gr Thyroid (Proloid, Parke Davis)



Figures 8



Figures 9A

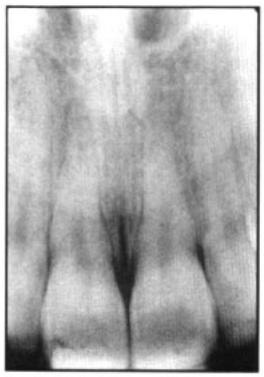


Figure 9B

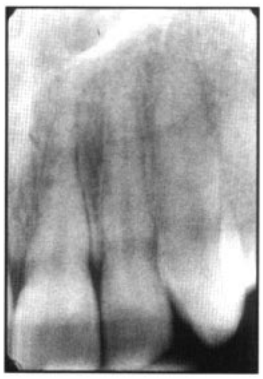


Figure 9C

Case NG

Figure 8
Periapical radiographs before treatment. Note root resorption beginning on central incisors and already shortened second premolars.

Figure 9A-C
Enlarged pretreatment periapical radiographs. Note fuzzy "moth-eaten" appearance of roots.

Figure 10
Posttreatment periapical radiographs. Central incisors look better, no continued resorption of second premolars.

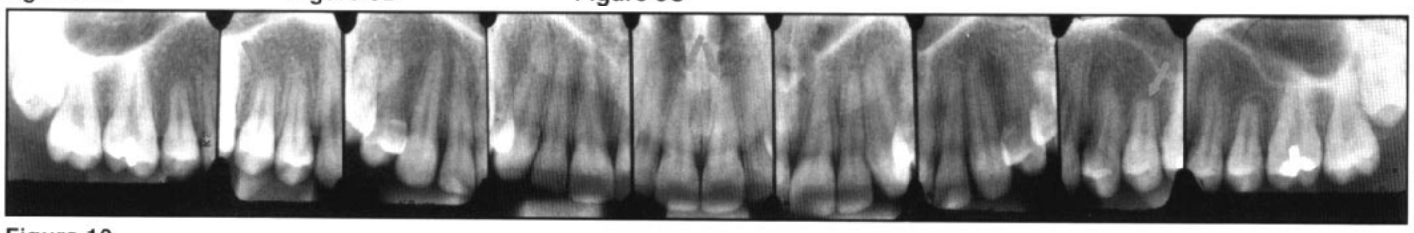


Figure 10

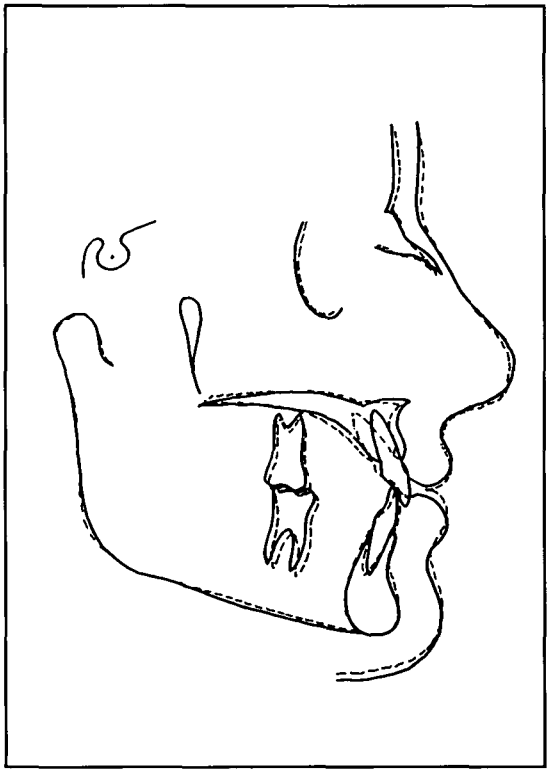


Figure 11

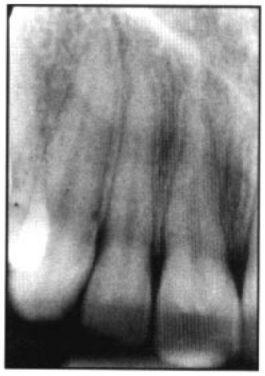


Figure 12A

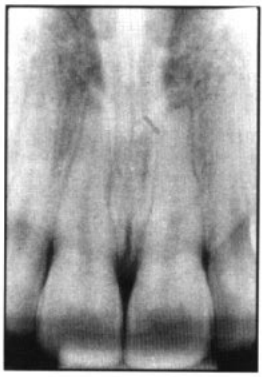


Figure 12B

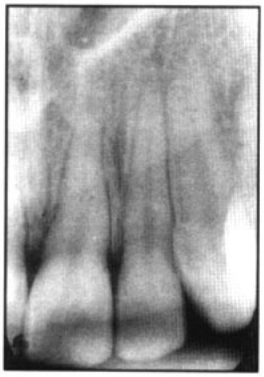


Figure 12C

Figure 11
Superimposed pretreatment and posttreatment cephalometric tracings.

Figure 12A-C
Enlarged posttreatment periapical radiographs. The roots look more defined and filled in.

was prescribed to be taken daily during treatment. The final periapical radiographs show that resorption of the premolars did not progress. Further, the roots of the maxillary central incisors seem to have an improved morphologic appearance after treatment. The apical third of the canines looks more defined and filled in.

Case KL

Patient KL presented at 9 years 2 months with an Angle Class II, division 1 malocclusion with crowding and bimaxillary constriction. Treatment was divided into two phases. During the

Case KL

Figure 13A-B
Pretreatment facial photographs.

Figure 14A-B
Pretreatment study casts.

Figure 15A-B
Enlarged periapical radiographs of KL's mother. Note root resorption of incisors.

Figure 16
Enlarged pretreatment periapical radiograph of KL Note poor root morphology.

Figure 17
Posttreatment radiograph shows long, strong roots.

Figure 18A-B
Posttreatment periapical radiographs. Note length of second premolar roots.

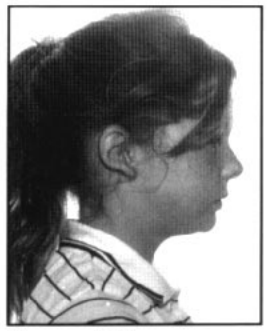


Figure 13A


Figure 13B

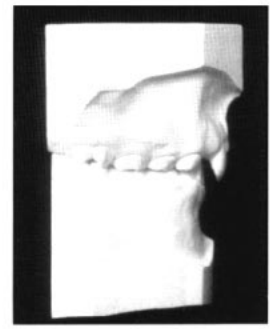


Figure 14A


Figure 14B

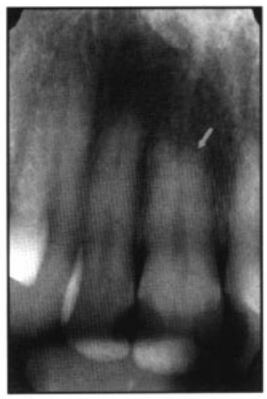


Figure 15A

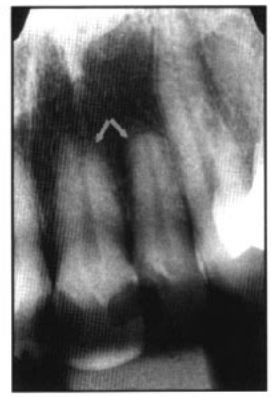


Figure 15B

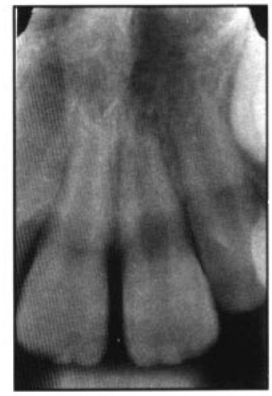


Figure 16

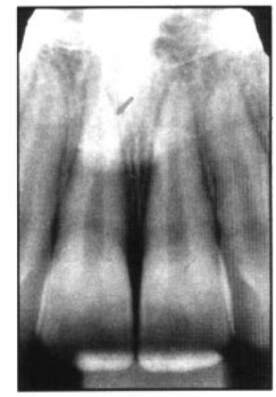


Figure 17

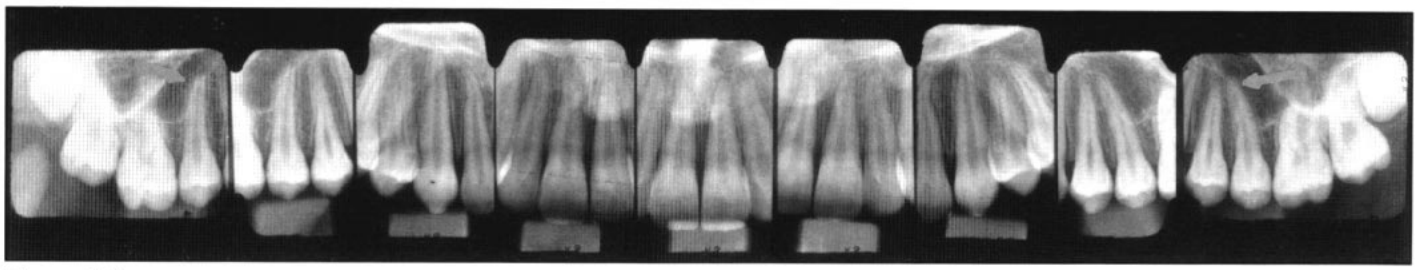


Figure 18A

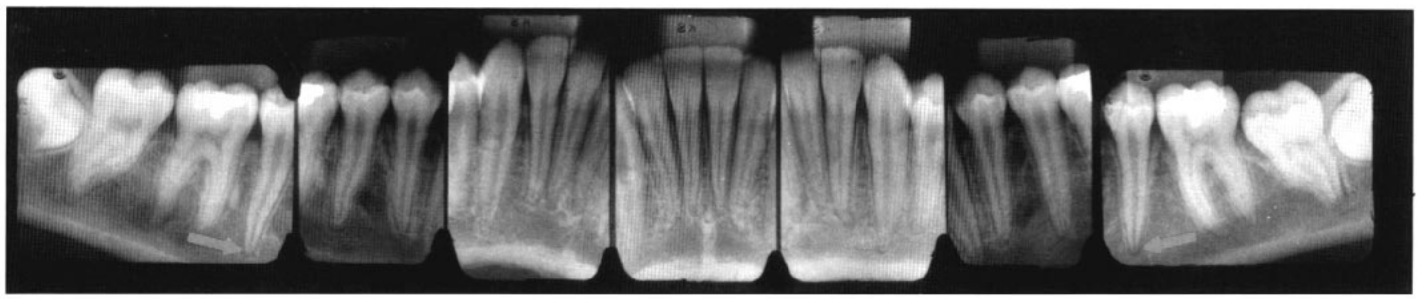


Figure 18B

first phase, the skeletal discrepancy was corrected by transverse expansion of the maxillary arch along with distal movement of the maxillary and mandibular first molars using a maxillary fixed expansion appliance (Haas type), a cervical facebow and a lower labial "E" arch. Treatment time for this first phase was 11 months. During the second phase, the teeth were aligned and the occlusion finalized with a full bonded appliance. Active treatment time for the second phase was 10 months. Because KL's mother had a history of low thyroid function and showed significant root shortening of her maxillary incisors, and because KL exhibited root morphology that indicated risk for root shortening, .5 gr Thyroid (Proloid, Parke Davis) was prescribed during active tooth movement at the second phase of treatment. The time between radiographs was 4 years 4 months. Final radiographs show significantly longer, stronger roots than expected, especially considering the severe apical resorption of the mother's incisors which would indicate a hereditary tendency for root resorption.

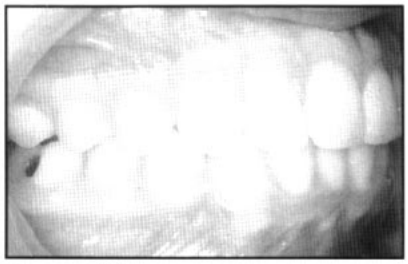


Figure 19A

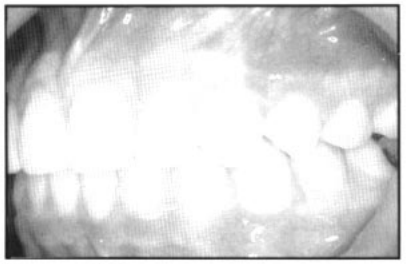


Figure 19B

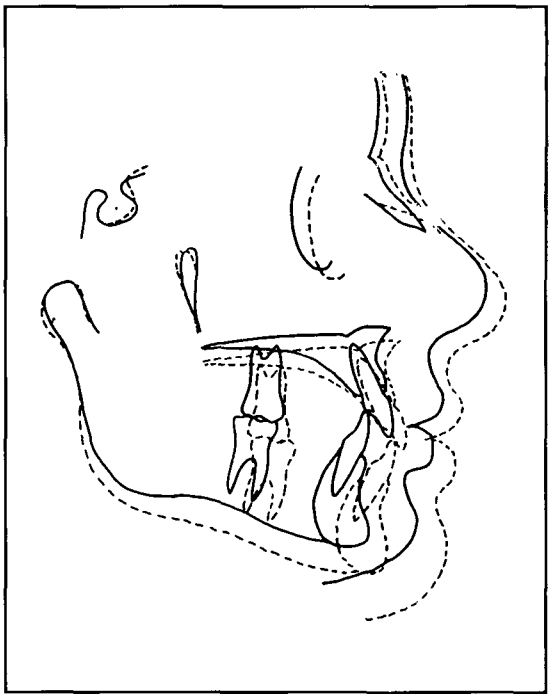


Figure 21

Conclusions

None of the patients exhibited any clinical side effects from the .5 gr Thyroid and all appeared to benefit from the thyroid supplement. All were taken off the medication at the time retainers were placed with no clinical side effects.

Acknowledgments

The authors wish to acknowledge Dr. Howard M. Lang who was the pioneer in prescribing Thyroid for root resorption.

Author Address

Dr. Eric L. Loberg
10231 Santa Monica Blvd.
Los Angeles, CA 90067

Dr. Eric Loberg is in private practice in Los Angeles and is an Assistant Professor of Orthodontics at UCLA. He is past president of the Southern California Component of the Edward H. Angle Society of Orthodontists, (1992-1993).

Dr. Christer Engström was Professor and Chairman of the Section of Orthodontics at UCLA through March 1993 and is now in clinical practice in Sweden.

Figure 20

Figure 19A-B
Posttreatment intraoral photographs.

Figure 20
Posttreatment photo.

Figure 21
Superimposed pretreatment and posttreatment cephalometric tracings.

Commentary: Thyroxine administration and its effects on root resorption

Richard L. Christiansen, DDS, MSD, PhD

Dr. Loberg's paper should be of interest to every practicing orthodontist who is concerned about the preservation of root structure during treatment. For the most part, orthodontists can be pleased with the preserved structure of the roots and alveolar bone in treated cases. There are always, however, occasions when evidence of root resorption is cause for amazement.

Klaushofer[1] reported in the *Journal of Bone and Mineral Research* in 1989 that prostaglandin plays an important role in the bone-resorbing actions of thyroid hormones. Thyroid hormones increase osteoclastic bone resorption in neonatal mouse calvaria by stimulation of prostaglandin, especially prostacyclin synthesis. Thyroid hormones also work via a separate prostaglandin-independent mechanism. Klaushofer noted that the mechanism by which thyroid hormones influence bone resorption is not clear at present, except that it certainly involves activation of osteoclasts. Viewed from

this perspective, Dr. Loberg's statement that "thyroxine administration seems to lower the frequency of root resorption" might more accurately be phrased, "Thyroxine administration seems to increase the rate of alveolar bone resorption, thus, indirectly decreasing root resorption."

Dr. Loberg suggests that thyroxine administration should be considered for some patients, especially those who begin to show root resorption or who have low thyroid function. It may be premature to consider human use of thyroxine to slow or prevent root resorption. In 1992, Stepan and Limanova[2] reported on 58 patients who had undergone thyroidectomies due to cancer and subsequently received chronic doses of thyroxine. While both bone formation and bone resorption biomechanical indices rose, vertebral density was reduced. This was accentuated in the older female patients. Another report in 1992 in the *Journal of Bone and Mineral Research*[3] found that male rats given large doses of thyroxin developed a reduced cortical density in femoral bones. The cortical bone was unchanged. In a variety of clinical reports, 20 studies noted reduced bone density while two studies reported no density changes. In one of these studies,[4] 35 white male patients with a variety of thyroid disorders including Grave's Disease, received long-term thyroid hormone replacement with no significant differences in bone mineral content among any subgroups. In the second study,[5] 49 patients receiving thyroid hormone showed no evidence of lower bone mineral density than a control group. Publication of this article, however, stimulated numerous letters to the editor disagreeing with the conclusion of no thyroxine effect on bone density.

A final point which should be made relates to collagen in the bone. In studies reported in 1991, Harvey, et al.,[6] measured bone-collagen-associated compounds in the urinary excretion of 19 patients with untreated overactive thyroid glands and 40 women (20 postmenopausal) on T4 replacement therapy. The majority of the patients showed bone collagen breakdown. This may represent an interesting problem for orthodontic treatment because of the high collagen levels in the periodontal membrane and the increasing frequency of adults seeking orthodontic treatment.

In reference to new research directions, researchers in a 1991 report[7] suggested gallium nitrate inhibits bone resorption in neonatal mice calvaria. Gallium nitrate incorporates into hydroxyapatite crystal, which produces an enlarged crystal that is more resistant to cell-mediated resorption. If we could selectively incorporate the gallium nitrate-hydroxyapatite crystal into cementum or dentin, the concern for orthodontic-induced root resorption might be reduced. Other new potent biphosphonates—300 to be precise—are under investigation to inhibit bone resorption. One compound inhibited stimulation of bone resorption for 2 weeks and inhibited normal non-stimulated bone resorption for at least 7 days.[8]

In summary, the use of thyroxine may open new vistas for orthodontics at the molecular/hormonal level. Practical applications for day-to-day practice may be years away; however, the impact is potentially most remarkable.

R.L. Christiansen is a Professor in the Department of Orthodontics and Pediatric Dentistry, School of Dentistry, University of Michigan in Ann Arbor.

References

1. Klaushofer K, et al. Bone-resorbing activity of thyroid hormones is related to prostaglandin production in cultured neonatal mouse clavaria. J Bone Mineral Res 1989;4:305-312.
2. Stepan JJ, Limanova Q. Biochemical assessment of bone loss in patients on long-term thyroid hormone treatment. Bone and Mineral 1992; 17:377-388.
3. Ongphiphadhannakul B, et al. Excessive L-thyroxine therapy decreases femoral bone mineral densities in the male rat: Effect of hypogonadism and calcitonin. J Bone Mineral Res 1992;7:1227-1231.
4. Franklyn JA, et al. Long-term thyroxine treatment and bone mineral density. Lancet 1992;340:9-13.
5. Toh SH, Brown PH. Bone mineral content in hypothyroid male patients with hormone replacement: A three-year study. J Bone Mineral Res 1990;5:463-467.
6. Harvey RD, et al. Measurement of bone collagen degradation in hyperthyroidism and during thyroxine replacement therapy using pyridinium cross-links as specific urinary markers. J Clin Endocrinol Metabolism 1991;72:1189-1194.
7. Lakotos P, et al. Gallium nitrate inhibits bone resorption and collagen synthesis in neonatal mouse calvariae. J Bone Mineral Res 1991;6:1121-1126.
8. Muhlbauer RC. BM 21.0955, a potent new biphosphonate to inhibit bone resorption. J Bone Mineral Res 1991;6:1003-1011.
9. Newman WG, et al. Etiologic factor in external root resorption. Am J Orthod 1975;67:522-539.