CHAPTER 1

Foundations

The Spectrum of Vertical

Q: Emily is a 20-year-old who presents to you for treatment (Figure 1.1). Compare her soft tissue midface height to her soft tissue lower anterior facial height (LAFH). What do you observe?

Figure 1.1 (a, b) Emily's facial photographs. (c, d) Soft tissue midface height (vertical distance from supraorbital ridges/soft tissue Glabella to Subnasale) is compared to LAFH (vertical distance from Subnasale to soft tissue Menton). Abbreviations used in this book are defined in the Appendix.

A: Emily's soft tissue proportions are nearly ideal. Her soft tissue midface height is equal to her soft tissue LAFH. [1, 2] Also, the distance from Subnasale to Stomion is approximately one-half of the distance from Stomion to soft tissue Menton. She exhibits lip competence without either an interlabial gap (ILG) or an overclosed appearance.

Q: Next, evaluate her vertical skeletal and dental features using her cephalometric tracing (Figure 1.2). What do you observe?

A: The (percentage) skeletal LAFH is expressed as a proportion of (linear) skeletal LAFH to (linear) skeletal TAFH (total anterior facial height). TAFH is the distance Nasion–Menton. LAFH is the distance measured from Menton along a Nasion–Menton line to a point where ANS projects perpendicularly to the Nasion–Menton line. For Emily, the LAFH/TAFH ratio (expressed as a percentage) is 54%. Ideal LAFH/TAFH is 55%, and one standard deviation difference from ideal is approximately 2% [3]. So, Emily's LAFH/TAFH is normal. In terms of mandibular plane (MP) angles, her FMA is normal (26°; ideal is 25°) and SNMP is normal (30°; ideal is 32°). Her maxillary first molar root apices are located below the palatal plane, a feature generally found in patients with vertical maxillary excess. Finally, the maxillary central incisor tip extends below the upper lip by approximately 4 mm (ideal 2–4 mm) [4]. A primer on the determination of facial height and other cephalometric parameters used in this book can be found in the Appendix.

Q: A number of Emily's skeletal and dental features were described as normal, but not ideal. Should not the terms normal and ideal be synonymous? In other words, if the ideal (average) FMA is 25°, should not this exact value be considered normal and all other FMA values considered abnormal?

A: No. "*Normal" constitutes a range of values and not one specific number*. That is why we say, "within the range of normal," or WRN. In other words, an FMA of 25° is normal but so can FMAs of 26°, 27°, 28°, 24°, 23°, and 22° be considered WRN. The same can be said for LAFH/TAFH proportions, ANB values, FMIA values, and so forth. Ideal can be considered one specific value, for instance, the average value measured from a population, but normal is a range—not a number. Think of it this way, there exists a broad range of what we consider beautiful human faces, and normal human faces cover an even broader range. A discussion of "normal" cephalometric values can be found in the Appendix.

Q: Evaluate Emily's vertical dental features, as seen intraorally (Figure 1.3a). What do you observe?

A: Overall, she presents with normal overbite (10–20% vertical overlap of her mandibular incisors by her maxillary central incisors measured in centric occlusion, Figure 1.3b). Variation

Orthodontics in the Vertical Dimension: A Case-Based Review, First Edition. Thomas E. Southard, Steven D. Marshall and Laura L. Bonner.

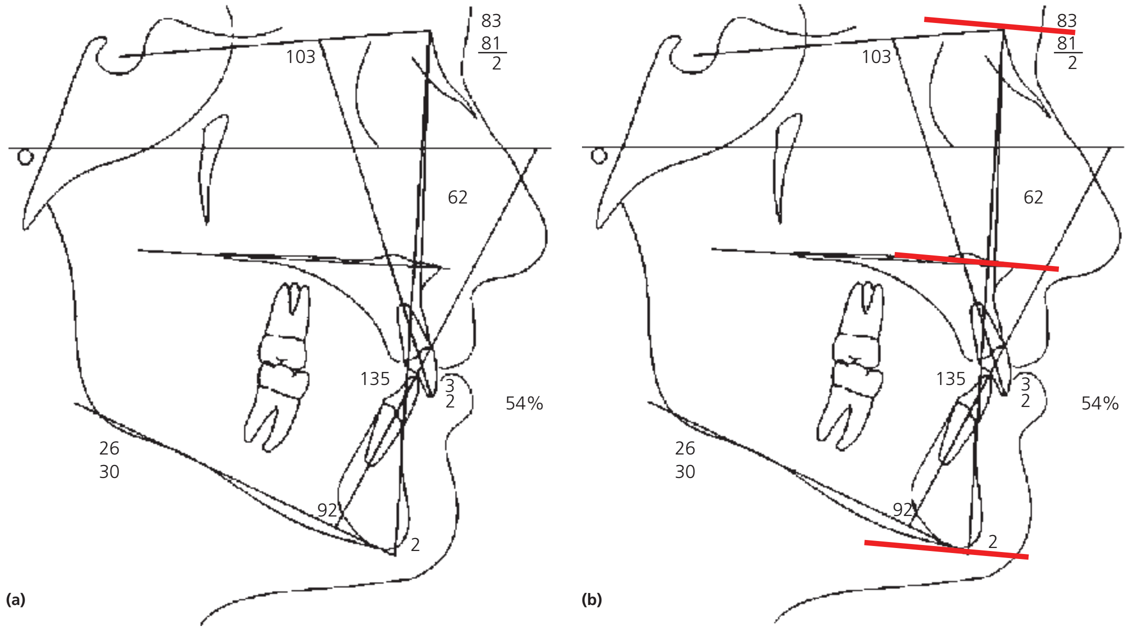


Figure 1.2 (a) Emily's lateral cephalometric tracing. (b) Skeletal TAFH is the distance Nasion–Menton. Skeletal LAFH is the distance measured from Menton along a Nasion–Menton line to a point where ANS projects perpendicularly to the Nasion–Menton line. A primer on the determination of facial height and other cephalometric measurements used in this book can be found in the Appendix.

Figure 1.3 (a) Frontal intraoral view of Emily with (b) approximately 20% central incisor overbite measured relative to mandibular central incisor crown length.

in overbite is illustrated in Figure 1.4, which ranges from excessive vertical overlap (deep OB, Figure 1.4 left) to an absence of vertical overlap (open bite, Figure 1.4 right). Emily's maxillary right lateral incisor (Figure 1.3a) exhibits inadequate vertical overlap with her mandibular right lateral incisor—resulting in dark incisal embrasures. Her maxillary central incisor gingival margins are at the same level (ideal), her right and left maxillary lateral incisor gingival margins are about even but stepped down slightly compared to the centrals (ideal), and her right canine gingival margin is stepped up slightly compared to the right lateral (ideal, central–lateral–canine gingival heights are "high–low–high"). Her left canine gingival margin is even with her left lateral gingival margin (less ideal). Finally, her midlines are coincident (ideal), and the height of the gingival papilla between the maxillary central incisors is about one-half the vertical crown length of the central incisors (ideal) [5].

Combining Emily's soft tissue (clinical), skeletal (cephalometric), and dental (intraoral) features, we conclude that she is vertically normal. If we imagine a spectrum of patients presenting with varying degrees of vertical facial development (Figure 1.5), then we can imagine plotting Emily as normal.

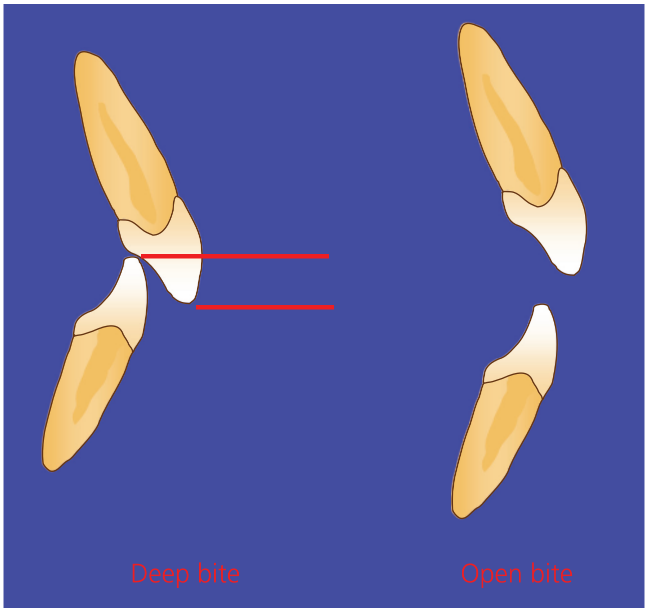


Figure 1.4 Illustration of maxillary and mandibular central incisors in the sagittal view depicting incisor overbite, ranging from deep (left) to open (right).

Q: Let's use our spectrum to compare subjects who vary considerably in vertical facial development. Three more patients present for examination (Figure 1.6, left to right Kelly, Ashley, and Alexis). Compare their soft tissue midface heights to their soft tissue LAFHs. Compare other facial features. What do you observe?

A: A stark contrast in vertical soft tissue proportions exists between Kelly, Ashley, and Alexis. Kelly's LAFH is shorter than her midface height, Ashley's LAFH is equal to her midface height, and Alexis's LAFH is longer than her midface height. Kelly and Ashley are facially symmetric, while Alexis has a slight chin deviation to her right. Kelly appears overclosed. Kelly and Ashley demonstrate lip competence with the distance from Subnasale to Stomion approximately one-half the distance from Stomion to soft tissue Menton (ideal). Alexis presents with lip incompetence (ILG), so Stomion (the midpoint of the oral fissure when the lips are closed) does not exist. Alexis presents with an ILG of 8 mm. An ILG of up to 2 mm may be considered normal [6].

Q: Evaluate and contrast their respective vertical skeletal and dental features using cephalometric tracings (Figure 1.7). What do you observe?

A: Kelly's skeletal LAFH/TAFH is 50%, more than two standard deviations less than ideal (55%) (Figure 1.7d). Ashley's LAFH/TAFH is 56%, normal (Figure 1.7e). Alexis's LAFH/TAFH is

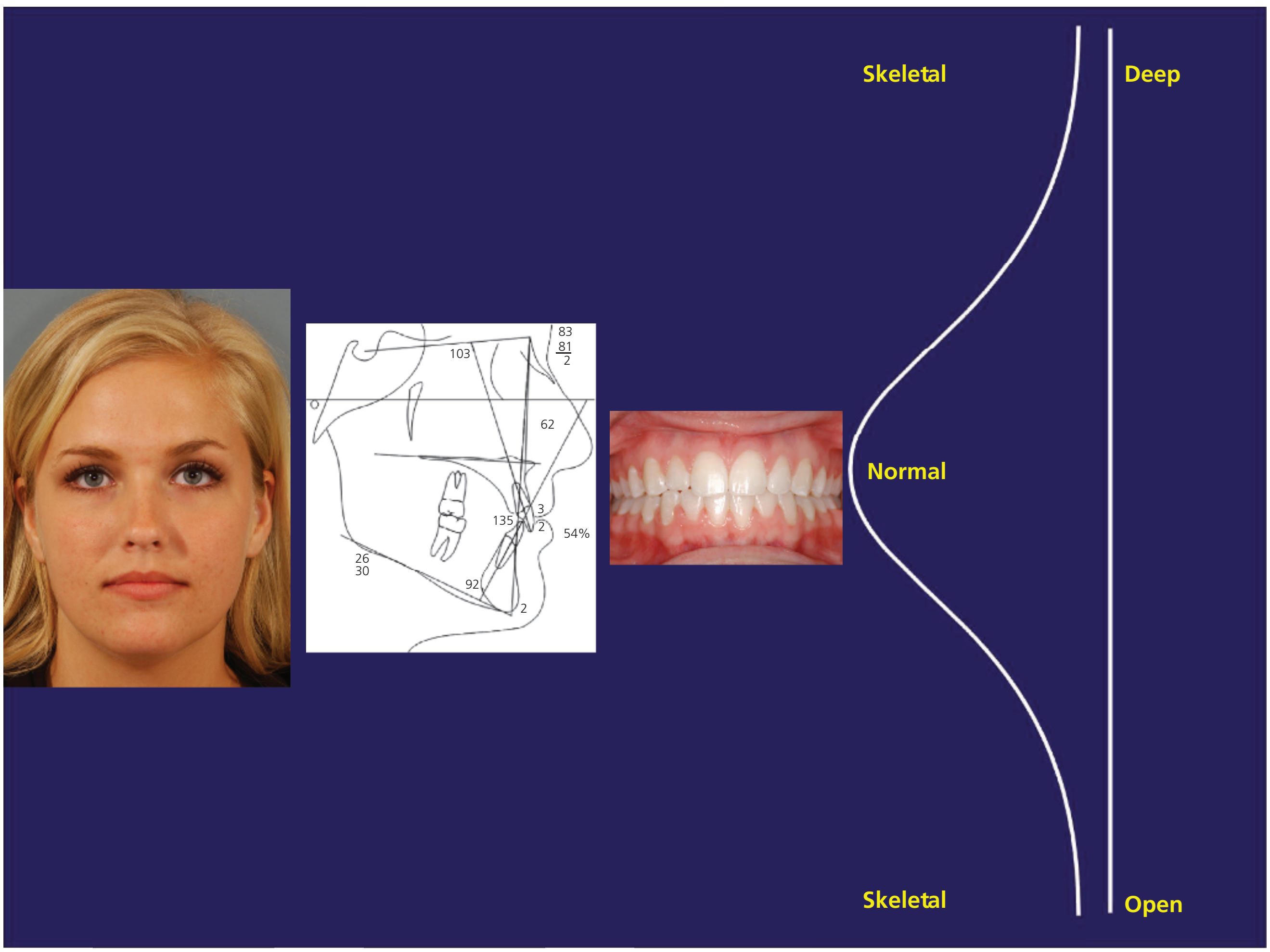


Figure 1.5 The spectrum of vertical facial development depicted on the right ranges from deficient (top, usually associated with deep bite) to excessive (bottom, usually associated with open bite). Emily is located in the center of this range and exhibits normal vertical facial development.

Figure 1.6 Variation of vertical soft tissue proportions: (a, d) Kelly, (b, e) Ashley, and (c, f) Alexis.

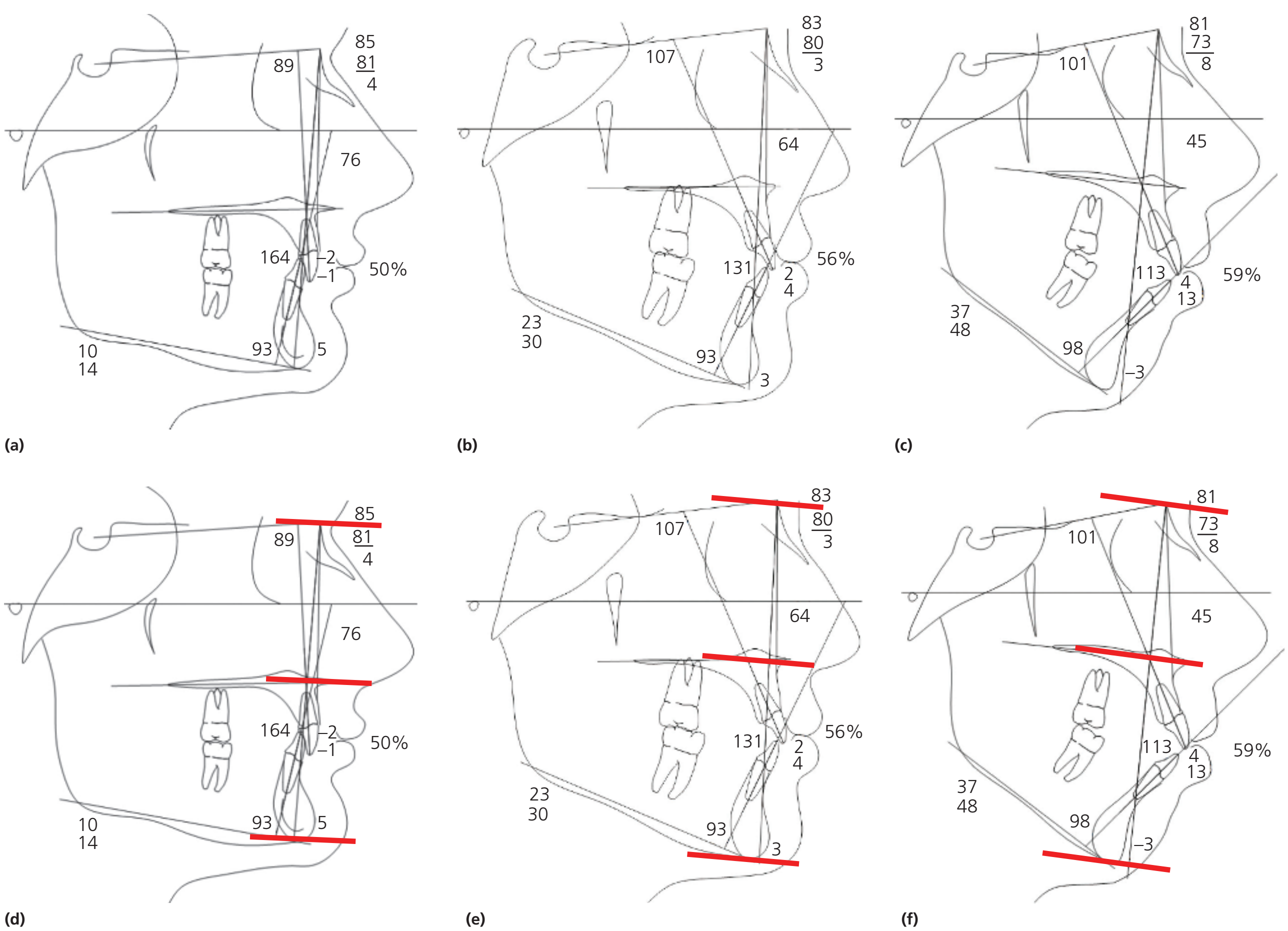


Figure 1.7 Variation of vertical skeletal and dental features: (a, d) Kelly, (b, e) Ashley, and (c, f) Alexis.

Figure 1.8 Variation of intraoral vertical dental features for the subjects shown in Figure 1.6: (a) Kelly, (b) Ashley, and (c) Alexis.

59%, two standard deviations greater than ideal (Figure 1.7f). Kelly's MPAs are flat (FMA = 10°, SNMP = 14°) compared to ideal (FMA = 25°, SNMP = 32°), and her MP, FH, and SN (Sella–Nasion) lines are roughly parallel. Ashley's MPAs are normal (FMA = 23°, SNMP = 30°). But Alexis's MPAs are steep (FMA = 37°, SNMP = 48°), and her MP, FH, and SN lines diverge significantly. Kelly and Ashley's maxillary first molar apices are located at their respective palatal planes (ideal), while Alexis's maxillary first molar apices have erupted well below her palatal plane. Kelly's maxillary central incisor tip extends below her maxillary lip by about 4 mm (ideal 2–4 mm), but this maxillary lip coverage could change if she were to separate her jaws slightly and let her maxillary lip relax. Ashley's and Alexis's maxillary incisor tip extends below their maxillary lips by 1–2 mm.

Q: Finally, evaluate their vertical dental features, as seen intraorally (Figure 1.8). What do you observe?

A: Kelly presents with 100% OB (Figure 1.8a, 100% overlap of her mandibular incisors by her maxillary incisors). Her maxillary incisors are "stepped down" relative to her maxillary posterior teeth. Ashley has an ideal OB of 10–20% (Figure 1.8b). Alexis has a 2–3 mm anterior open bite with maxillary incisors stepped up relative to the maxillary posterior teeth and mandibular incisors stepped down relative to the mandibular posterior teeth (Figure 1.8c). In terms of gingival levels, all Kelly's maxillary incisors have gingival margins at the same level (due to central incisor overeruption). Ashley's central incisor gingival margins are slightly uneven, but she demonstrates reasonably normal high–low–high gingival margin relationships proceeding from maxillary central incisors to canines. Alexis's maxillary central incisor gingival margins are even, and she demonstrates a reasonable high–low–high gingival margin relationship proceeding from central incisors to canines. Coincidence of maxillary and mandibular midlines cannot be ascertained in Kelly's frontal view, Ashley's midlines are coincident, and Alexis's midlines appear to be reasonably coincident. Kelly demonstrates a short gingival papilla between her supraerupted maxillary central incisor crowns. Ashley's and Alexis's maxillary incisor gingival papilla heights are approximately ideal (one-half of their respective central incisor crown lengths).

Q: Based on soft tissue (clinical), skeletal (cephalometric), and dental (intraoral) features, where would you place Kelly, Ashley, and Alexis on the spectrum of vertical facial development?

A: Kelly's features place her at one end of the vertical spectrum—skeletal deep bite. Alexis has features placing her at the other end of the vertical spectrum—skeletal open bite. Ashley is normal, similar to Emily in Figure 1.1. Kelly and Alexis are plotted with Emily along the vertical spectrum in Figure 1.9.

Q: Let's complete our vertical spectrum. Three additional patients present for examination (Figure 1.10, Cassie (a), Grace (b), and Adair (c)). Compare their soft tissue midface heights to their soft tissue LAFHs. Compare other facial features. What do you observe?

A: All three are reasonably similar and normal. Cassie, Grace, and Adair have LAFHs equal to their midface heights (ideal). All three are symmetric and demonstrate lip competence, do not appear overclosed, and present with Subnasale to Stomion heights approximately one-half their Stomion to soft tissue Menton heights.

Q: Evaluate and contrast their respective vertical skeletal and dental features using cephalometric tracings (Figure 1.11). What do you observe?

A: In terms of vertical skeletal relationships, all three are quite similar and WRN. Cassie's LAFH/TAFH is ideal (55%), Grace's is normal (54%), and Adair's is normal (56%). All three have MPAs that range widely but are still WRN: Cassie's FMA is 27° and her SNMP is 35°, Grace's FMA is 20° and her SNMP is 30°, and Adair's FMA is 27° and his SNMP is 38°. Cassie's and Grace's maxillary first molar apices are erupted away from the palatal plane, while Adair's maxillary first molar apices are level with his palatal plane (ideal). Finally, Cassie's maxillary central incisor tip extends below the upper lip by about 2–4 mm (ideal), Grace's maxillary incisor tip extends below the upper lip by about 2 mm (normal), and Adair's maxillary incisor tip is about at the level of the upper lip (less than normal).

Q: Evaluate their vertical dental features, as viewed intraorally (Figure 1.12).

A: This is where significant differences exist between the three patients. Cassie has a 50–60% OB with overerupted maxillary central incisors, Grace has an OB WRN (10–20%) with stepped up maxillary incisors, and Adair has an open bite of 1–2 mm with stepped up maxillary incisors. In terms of gingival levels, Cassie's central incisor gingival margins are uneven—her right maxillary central incisor gingival margin is low due to its supraeruption. All four of Grace's maxillary incisor gingival margins are approximately even and sit above her maxillary canine gingival margins. Adair's central incisor gingival margins are approximately even and are situated much higher than either his lateral incisor or his canine gingival margins. Cassie, Grace, and Adair did not exhibit ideal high–low–high gingival margin relationships (proceeding from maxillary central incisors to maxillary canines). All three have approximate coincidence of maxillary and mandibular midlines. Cassie demonstrates a short gingival papilla height between her supraerupted maxillary central incisor crowns; Grace's and Adair's gingival papilla heights between the central incisors are ideal (about one-half of her and his central incisor crown lengths).

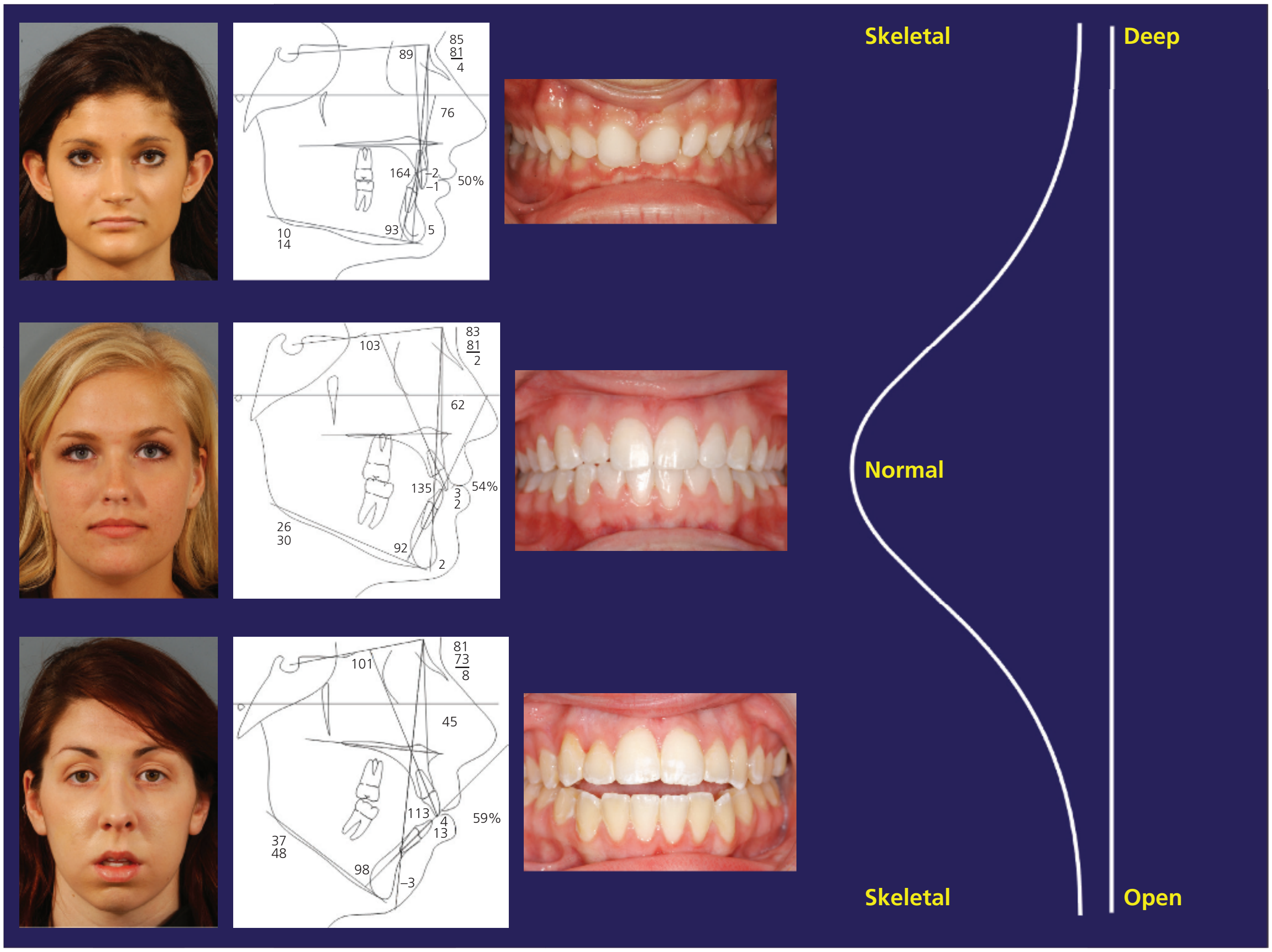


Figure 1.9 Kelly, Emily, and Alexis plotted on the spectrum of vertical facial development.

Figure 1.10 Variation of vertical soft tissue proportions: (a, d) Cassie, (b, e) Grace, and (c, f) Adair.

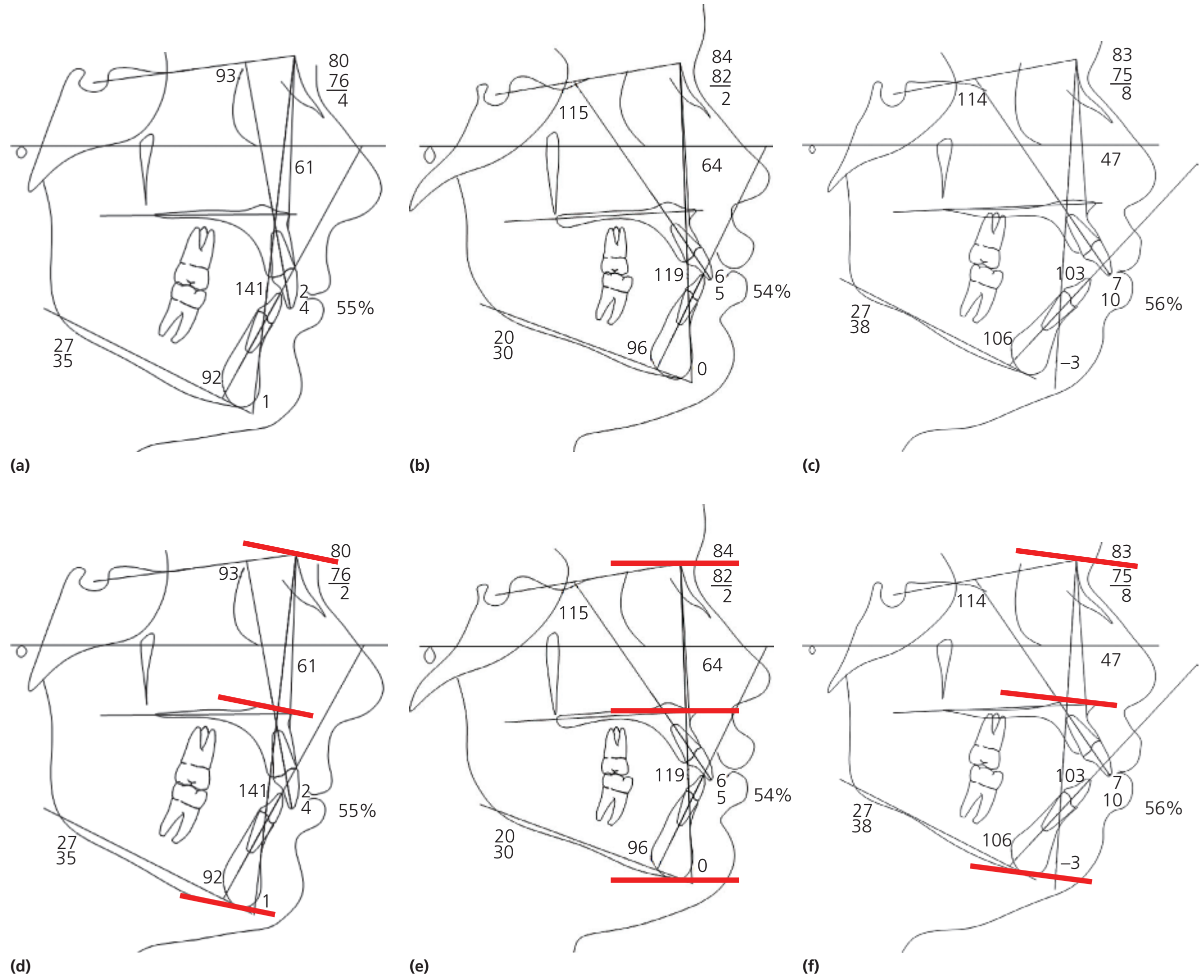


Figure 1.11 Variation of vertical skeletal and dental features: (a, d) Cassie, (b, e) Grace, and (c, f) Adair.

Figure 1.12 Variation of intraoral vertical dental features for the subjects shown in Figure 1.10: (a) Cassie, (b) Grace, and (c) Adair.

Q: If Cassie, Grace, and Adair are normal in terms of their vertical soft tissue (clinical) and vertical skeletal (cephalometric) features but differ in terms of their vertical dental (intraoral) features, where would you plot them on the spectrum of vertical facial development?

A: Since Cassie has normal vertical soft tissue/skeletal development but 50–60% OB, she should be plotted as a *dental* deep bite patient (Figure 1.13). Grace is normal in terms of vertical soft tissue/skeletal development and OB—similar to Emily in Figure 1.5. Since Adair has normal vertical soft tissue/skeletal development but an open bite, he should be plotted as a *dental* open bite patient. Figure 1.13 now illustrates examples of patients covering the complete range of anterior vertical problems: skeletal deep bite, dental deep bite, normal, dental open bite, and skeletal open bite.

Q: Can every patient be classified into one of these five, discrete, vertical spectrum categories—without overlap of features from another category?

A: No. *Many (most) patients present with some overlap of features between categories.* But, the greater the preponderance of features a patient presents with from any one of the categories, the more unambiguously the patient may be classified in that category. For example:

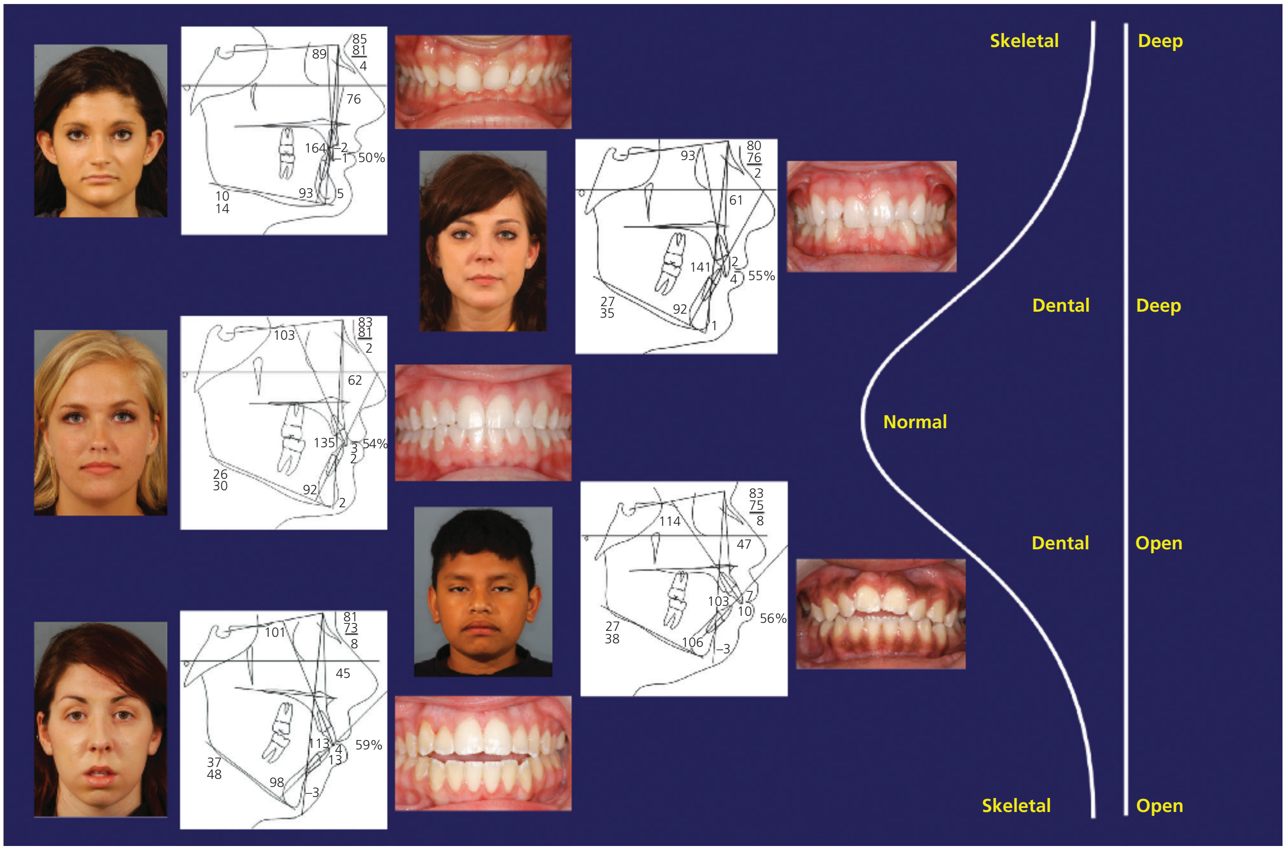


Figure 1.13 Example individuals representing the complete spectrum of vertical facial development.

- A dental deep bite patient can present as a pure dental deep bite—normal vertical facial growth and excess OB due solely to overerupted incisors.
- A dental deep bite patient (overerupted incisors) can present not only as a pure dental deep bite but also as a borderline skeletal deep bite (excess OB partially due to growth pattern).
- A dental open bite patient can present as a pure dental open bite—normal vertical facial growth and open bite solely due to undererupted incisors.
- A dental open bite patient (undererupted incisors caused by habit) can present not only as a pure dental open bite but also as a borderline skeletal open bite (open bite partially due to growth pattern).
- A severe skeletal deep bite patient can present as a pure skeletal deep bite—short LAFH with excess OB due entirely to the growth pattern and not excess incisor eruption.
- A severe skeletal deep bite patient (growth pattern, short LAFH) can also present with a dental deep bite (overerupted incisors) or even a dental open bite (undererupted incisors due to a habit).
- A severe skeletal open bite patient can present as a pure skeletal open bite—long LAFH with open bite due entirely to the growth pattern and not inadequate incisor eruption.
- A severe skeletal open bite patient (growth pattern, long LAFH) can also present with a dental open bite (undererupted incisors caused by habit), a normal OB, or even a dental deep bite (overerupted incisors). Of course, an open bite would not be present in the latter case, just the skeletal open bite growth pattern (LLAFH).

The point is that vertical dimension problems result from interrelationships between vertical condylar growth, ramus growth, sutural lowering of the maxillary corpus, growth rotation of the jaws, dentoalveolar growth, muscle growth, jaw function, and oral habits. The complex three-dimensional interplay of the developing facial structures and developing muscle function produces considerable variation in vertical dimension abnormalities of the facial complex. We've considered vertical dimension problems as a discrete spectrum for the purpose of education, portraying variation from normal vertical relationships due to aberrations in dentoalveolar and skeletal growth—and for more readily establishing principles of treatment.

Q: Mitchell presents to you for treatment (Figure 1.14). What vertical features do you observe? How would you classify Mitchell?

A: Examination in the frontal and profile views reveals an increased soft tissue LAFH (the distance Subnasale to soft tissue Menton is much greater than the distance soft tissue Glabella to Subnasale). There is also noticeable lip strain with the lips closed (Figure 1.14a), but a large ILG exists (Figure 1.14d) with lips at rest. These features are suggestive of a skeletal open bite. Looking at the cephalometric radiograph and tracing (Figure 1.14f, g), several diagnostic features also suggest a skeletal open bite: SN-MP is extremely high at 48°; skeletal LAFH/TAFH ratio, 60%, is over two standard deviations above normal; posterior facial height is short; gonial angle is very shallow at 140°; significant

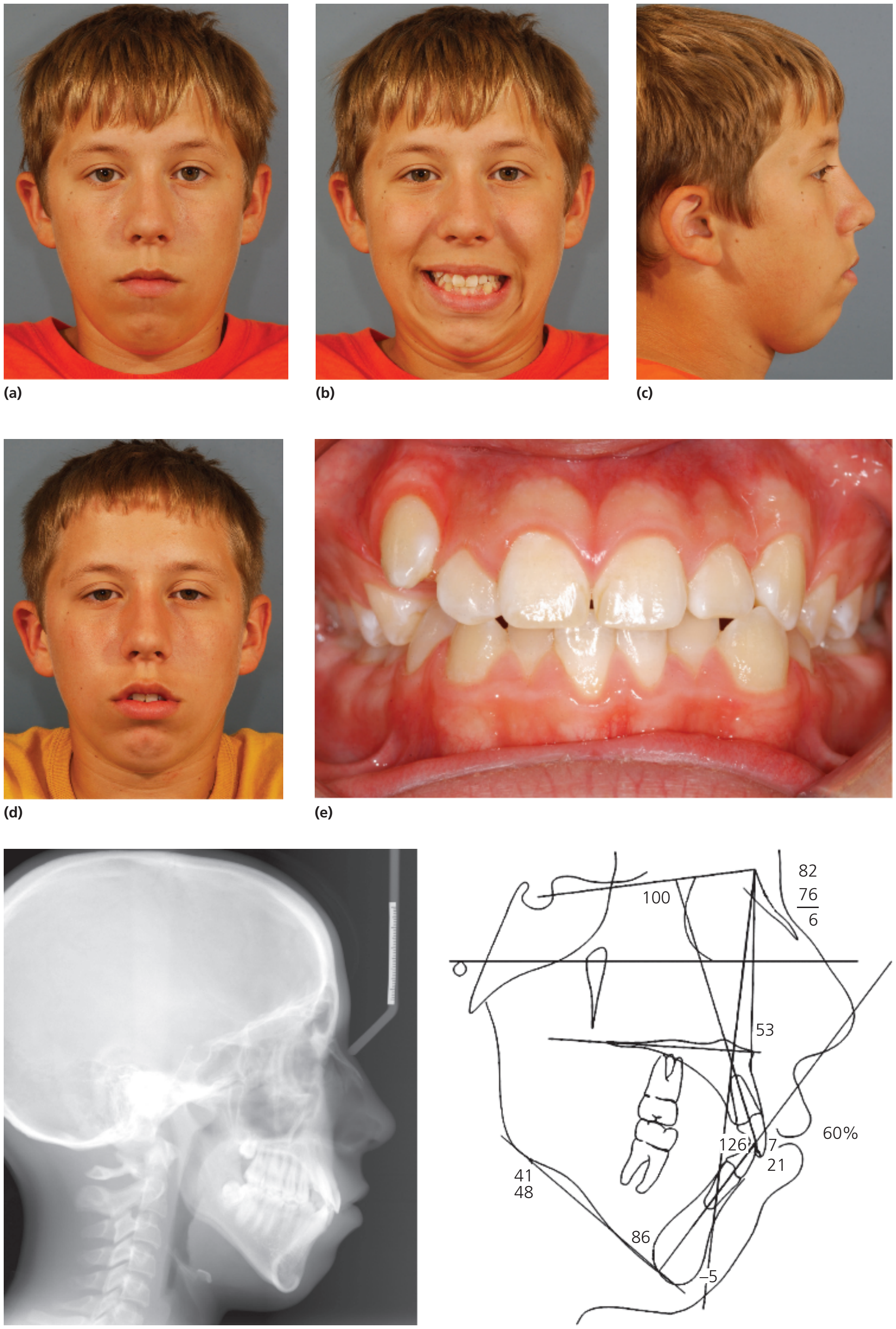


Figure 1.14 Initial records of Mitchell (a) lips straining to close, (b) smile, (c) profile with lips straining to close, (d) lips relaxed revealing an ILG, (e) intraoral photo, (f, g) lateral cephalograph and tracing.

antegonial notching is present; and maxillary first molar apices lie below the palatal plane, suggestive of excessive vertical descent of the posterior maxillary alveolar process and the maxillary molar teeth. Now look at Mitchell's intraoral photograph (Figure 1.14e). With the exception of the ectopic maxillary right canine, anterior overbite appears WRN (20% OB). In other words, *Mitchell has a skeletal open bite development pattern but with normal dental overbite.*

Q: In terms of the vertical spectrum of development, what is Mitchell an example of?

A: Mitchell is an example of overlapping features between categories. He has a preponderance of skeletal open bite features but with normal dental OB. Our point in presenting Mitchell is to emphasize the spectrum of vertical as continuous, with overlapping features between categories, and not discrete.

Q: With the extreme steepness of his LAFH and SN-MP, how did Mitchell achieve 20% OB during growth?

A: The most probable explanation is that *additional eruption of his mandibular anterior teeth compensated for his developing skeletal open bite*. Note how much more Mitchell's lower incisors have erupted (Figure 1.14g) compared to the amount lower incisors erupt in individuals with normal vertical growth (Figures 1.2a, 1.7b, and 1.11a–c).

We can also assume that Mitchell experienced some counterclockwise (forward) mandibular growth rotation, which helped his OB. Bjork [7, 8] showed that clockwise apparent mandibular rotation during growth (SN-MP angle increasing) is associated with increased anterior facial height and a steep MP angle. Schudy [9, 10] reasoned a steep MP angle (facial hyperdivergence) was the result of the vertical condylar growth increment not keeping pace with the increments of maxillary corpus vertical descent and the vertical eruption of posterior teeth, causing backward mandibular rotation and increasing SN-MP angle. However, the majority of *individuals characterized as "skeletal open bite" by their cephalometric and soft tissue characteristics, do not exhibit a net apparent clockwise (backward) mandibular rotation during growth. Most exhibit apparent counterclockwise (forward) mandibular rotation*. For most individuals, even those with hyperdivergent patterns, the SN-MP angle remains the same or decreases slightly during facial growth [11–13]. Karlsen [14] and Chung and Wong [15] demonstrated that individuals with high SN-MP angles followed longitudinally during childhood and adolescence displayed apparent forward (counterclockwise) rotation and decreasing SN-MP angles throughout development, albeit at a lower rate (degrees rotation per year) compared with individuals with low SN-MP angles. This supports the findings of others [16, 17] that a hyperdivergent facial pattern is inherited, and, in the majority of cases, maintained or improved with growth. So for Mitchell, it is plausible that, in spite of his inherited pattern of vertical facial relationships, some counterclockwise apparent mandibular rotation has also allowed him to maintain normal overbite.

Q: How does the concept of feature overlap, or lack of overlap, between vertical spectrum categories affect treatment?

A: On the one hand, the more unambiguously a patient can be classified in one of the vertical categories, the more you should follow the principles of treatment for that category. On the other hand, *the more overlap of features between two categories a patient presents with, the more your care should encompass treatment principles from both categories*. For example, if an open bite patient presents as a pure dental open bite, then you should care for that patient using treatment principles for a dental open bite. But, if a dental open bite patient also presents with borderline skeletal open bite features—then the care you provide should encompass treatment principles for a dental open bite *plus* treatment principles for a skeletal open bite.

Q: Mitchell has overlap of features between categories. How will this overlap affect your treatment decisions?

A: In spite of the fact that he has a normal overbite—you must address the skeletal open bite pattern during treatment—you must control vertical dentoalveolar growth and eruption of posterior teeth. If you do not, then backward mandibular rotation, increase in LAFH, and an anterior open bite may occur.

Q: Let's now turn to consider other aspects of the vertical dimension (and other features impacting facial esthetics as seen in the frontal view). In Figure 1.15a, b, we see two facially symmetric patients with normal vertical proportions. In Figures 1.15c and 1.15d, the amount of incisor display is visible when their teeth are slightly separated and their lips are relaxed. What do these figures illustrate?

A: These figures illustrate the amount of incisal display at rest. Ideal incisal display at rest varies with age and gender [4]. *Maxillary incisal display at rest decreases with age as philtrum length increases (even after age 30), and maxillary incisal display is less in males compared to females* [18, 19]. In terms of treatment, increased incisal display can project a youthful appearance. Decreased incisal display can project an aged appearance.

Q: What feature of dental position can impact incisal display at rest?

A: The presence of a step in the anterior portion of the maxillary occlusal plane, either down (Figure 1.16a) or up (Figure 1.16b), affects the amount of incisal display at rest. If an anterior step is initially present, the amount of incisal display at rest will change after the maxillary arch is leveled.

Q: Lip length also affects the amount of incisal display at rest. What is the range of normal adolescent lip lengths in girls and boys ages 13–15 years? How does age affect lip length?

A: The normal range of maxillary lip length for adolescent girls is 17–23 mm and for adolescent boys 22–26 mm [20]. During the third and fourth decades of life, the maxillary lip length increases approximately 1 mm more than the maxillary incisor edge descends—which explains why older persons show less incisal display than younger persons [19].

Q: The vertical red arrow in Figure 1.17a represents the philtrum length (measured from Subnasale to the maxillary lip vermilion border), while the yellow line represents commissure height (measured along a line parallel to the philtrum length line from Subnasale to the commissure). In adults, the philtrum length–commissure height difference should be small, maybe a few millimeters. In adolescents, the difference may be greater (decreasing as a result of differential lip growth during maturation) [21–23]. What impact can the philtrum length–commissure height difference make on maxillary incisal display?

A: Compare Matt (Figure 1.17b), who has a small philtrum length–commissure height difference, to John (Figure 1.17c), who has a large philtrum length–commissure height difference. Matt shows 1 mm incisor display at rest, while John shows nearly 10 mm of maxillary incisor due to his short philtrum length. John has what is termed an "exaggerated cupid's bow" of his maxillary lip.

Q: How about vertical features during smiling? In a posed smile (Figure 1.18a), the curvature of Ashley's maxillary incisal edges and canines parallel the curvature of her lower lip. What is this called?

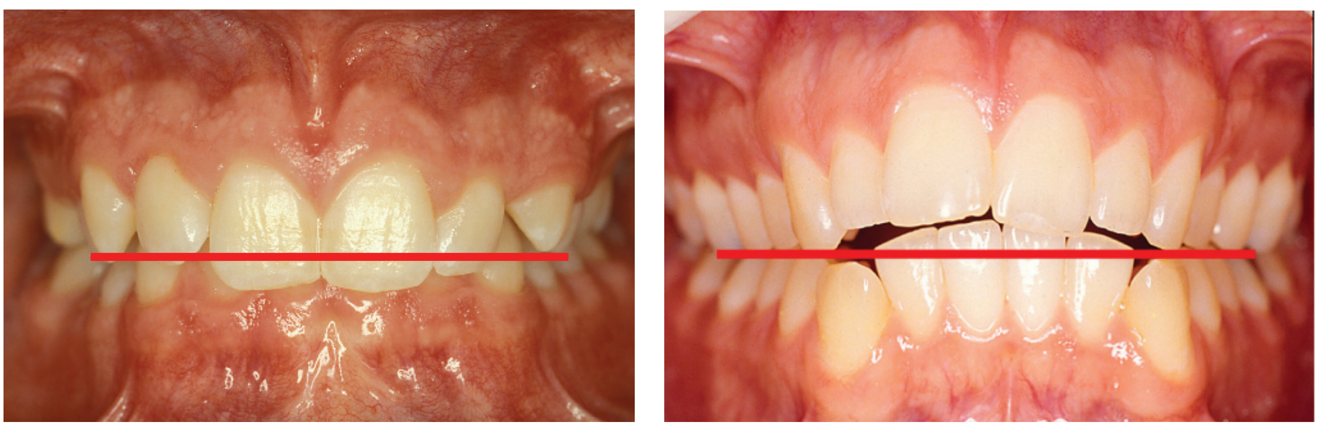
A: This relationship is termed an ideal smile arc [24].

Q: Compare Ashley's ideal smile arc (Figure 1.18a) with Eric's smile arc before (Figure 1.18b) and following (Figure 1.18c) treatment. What do you observe?

A: Whereas Ashley demonstrates an ideal smile arc, Eric initially presents with a *reverse* smile arc (Figure 1.18b). In other words, the edges of his maxillary incisors and canines curve upward, away from his lower lip, instead of curving downward

(a) (b) (c) (d)

Figure 1.15 Incisal display: (a, c) an adolescent girl, (b, d) a young man.



(a) (b)

Figure 1.16 Steps in the anterior portion of the maxillary occlusal plane affect the amount of incisal display at rest: (a) anterior teeth stepped down and (b) stepped up relative to the maxillary occlusal plane, which is defined by the occlusal surfaces of the posterior maxillary teeth bilaterally (red line).

following the curvature of his lower lip. Following treatment (Figure 1.18c), his smile arc is improved.

Q: What are the reasons for a patient presenting with *excessive* incisal and gingival display when smiling?

A: Excess incisal and gingival display can result from the following:

- Excess maxillary vertical growth
- Short maxillary lip (philtrum) length
- Stepped-down (overerupted) maxillary incisors
- Excessively long incisor crowns
- Delayed passive eruption (excess gingiva)
- Hyperactivity of (smiling) facial muscles

Figure 1.17 (a) Effect of philtrum length (red arrow)–commissure height (yellow arrow) difference on incisal display. (b) Matt has a small, 3–4 mm philtrum length–commissure height difference and exhibits 1 mm incisal display at rest. (c) John has a much larger philtrum length–commissure height difference and exhibits 10 mm of incisal display at rest.

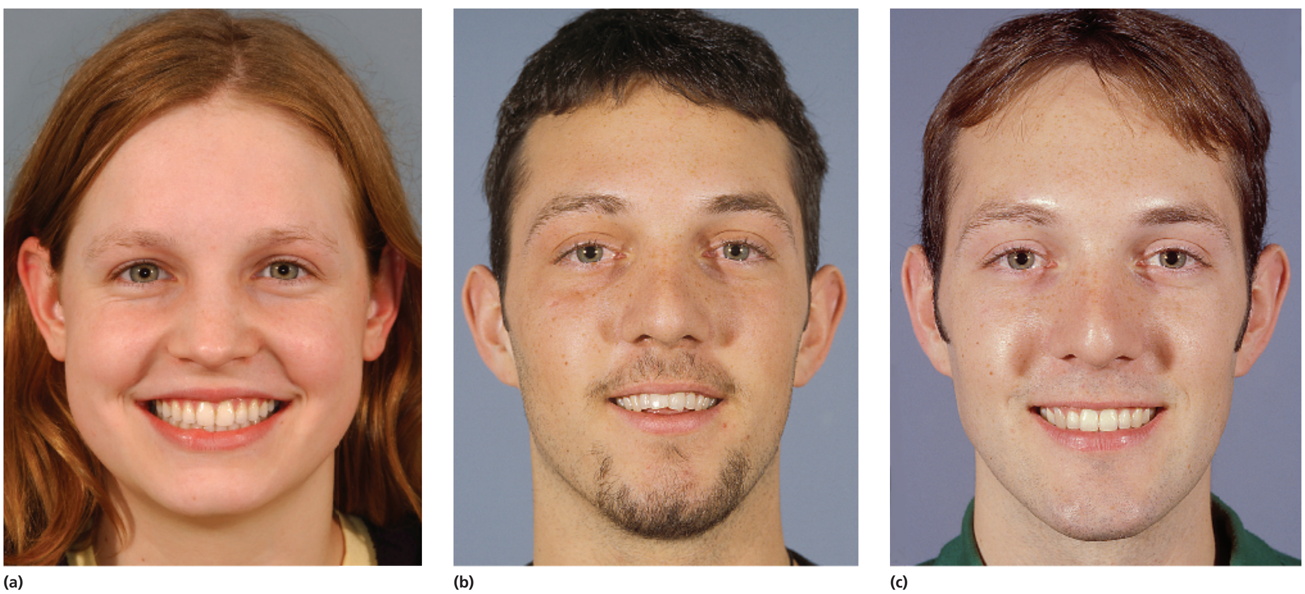


Figure 1.18 Smile arcs during a posed smile: (a) ideal smile arc, (b) reverse smile arc, (c) same patient, improved smile arc following treatment.

Q: What are the reasons for a patient presenting with *inadequate* incisal display when smiling?

A: Inadequate incisal and gingival display can result from the following:

- Inadequate maxillary vertical growth
- Long maxillary lip (philtrum) length
- Stepped-up (undererupted) maxillary incisors
- Worn or short incisor crowns
- Hypoactivity of (smiling) facial muscles

Q: Chris (Figure 1.19a) demonstrates facial symmetry and normal vertical proportions at rest. Compare the two photographs of him smiling (Figures 1.19b, c). What do you note?

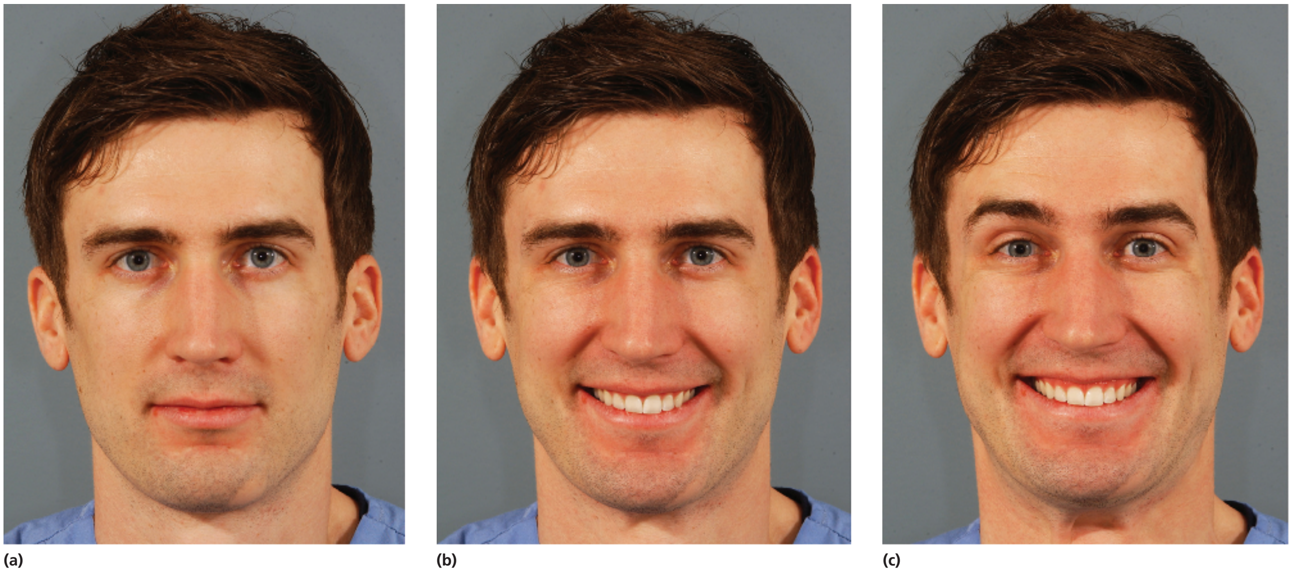


Figure 1.19 Chris: (a) at rest, (b) smiling in a posed smile, and (c) smiling in an unposed smile.

A: In Figure 1.19b, Chris is demonstrating a posed smile—a voluntary, repeatable, and sustainable smile typically made when posing for photographs (not elicited by emotion). In Figure 1.19c, he is demonstrating an unposed smile—a spontaneous smile elicited by joy or humor. We use the posed smile, not the unposed smile, to judge the position of the maxillary lip vermillion to the maxillary incisor gingiva.

Q: What is considered an attractive amount of gingival display in a posed smile?

A: In his posed smile (Figure 1.19b), the (vermillion) border of Chris's maxillary lip is at the same height as the gingival margins of his maxillary central incisors. This is considered ideal, the most attractive, vertical relationship [25–27]. However, it is not until the distance from the maxillary central incisor gingival margins to the maxillary lip is 3.0 mm or more that laypersons perceive a decrease in attractiveness. [28] This fact underscores the concept that attractiveness is a range, not a single value.

Q: Three patients (Figure 1.20; Tanner, Olivia, and Trevor) present to you with LAFHs (reasonably) WRN. As they smile (Figures 1.20d–f), compare their interpupillary lines to their respective right to left maxillary occlusal relationships. What do you observe?

A: They each present with an asymmetrical smile due to a *structural* maxillary occlusal cant. In other words, although each has their maxillary lip vermillion relatively parallel to their interpupillary line, Tanner's right maxilla/right dentition has descended more than his left—resulting in an occlusal cant. Olivia's and Trevor's left maxilla/left dentition has descended (slightly) more than their right.

Q: What percentage of the population has an asymmetrical smile due to a *structural* maxillary occlusal plane cant versus a functional (neuromuscular) canting of the upper lip?

A: Although this phenomenon has not been widely studied, one study (210 subjects) reported that 7% of their sample had an asymmetric smile due to an underlying maxillary (structural) occlusal cant, while 8% of their sample showed similar asymmetry due to a functional (neuromuscular) canting of the upper lip [29].

Q: How large of a right to left structural maxillary occlusal cant must exist to be noticed by laypersons?

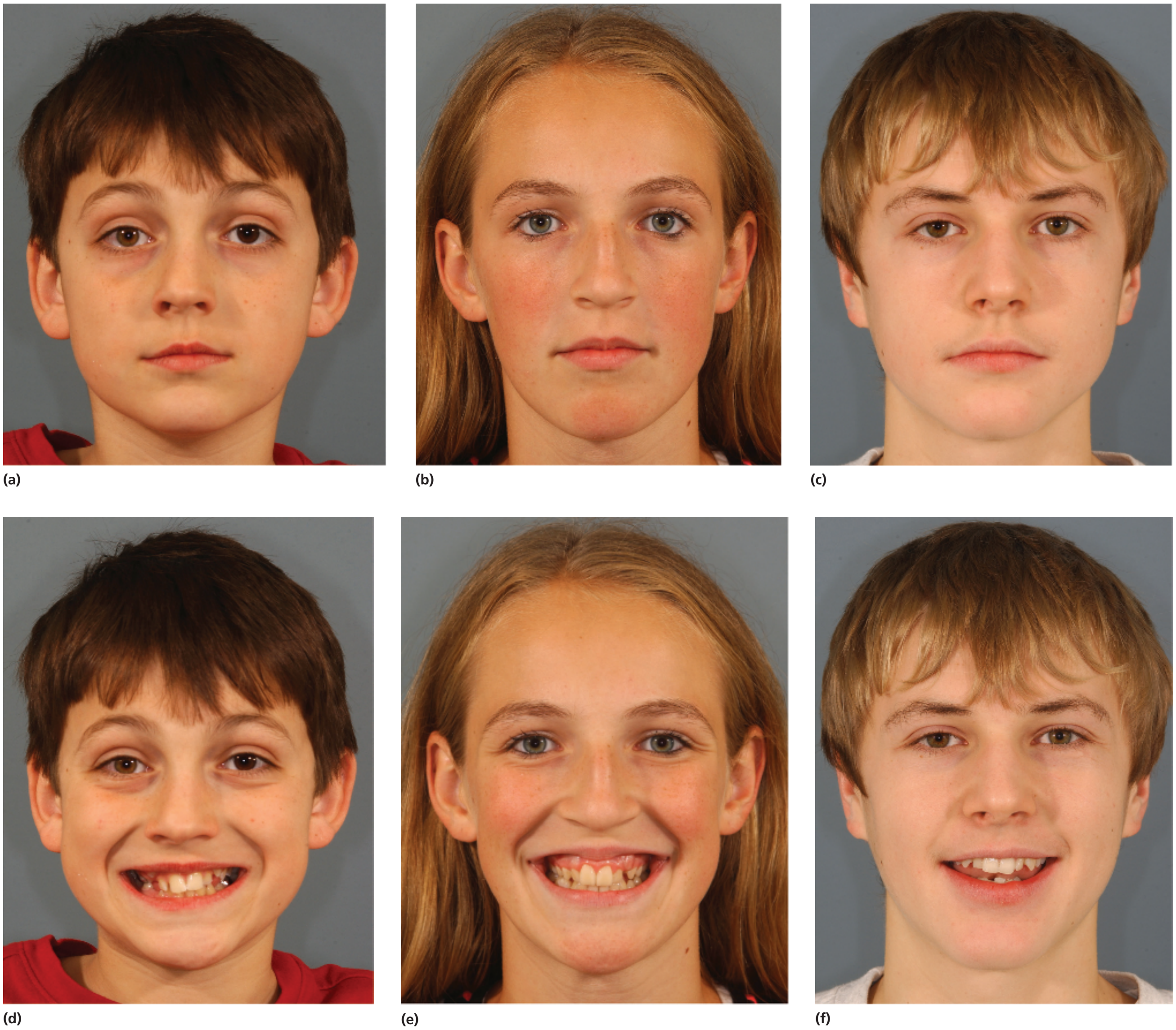
A: A maxillary right canine to left canine cant of at least 3 mm is required for laypersons to rate it as noticeable. An occlusal plane cant can be an overwhelmingly displeasing smile characteristic to health professionals and laypeople, alike [30].

Q: Lynn (Figure 1.21) also has an asymmetrical smile. Is the asymmetrical smile due to a structural maxillary occlusal cant or a functional canting of the upper lip?

A: Closely compare her interpupillary line to her right to left occlusal plane. They are very nearly parallel. There has not been an uneven descent of her maxilla or teeth between left and right sides. Look at her maxillary lip vermillion at rest (Figure 1.21a). It runs right to left in a nearly parallel fashion with her interpupillary line. But, when she smiles (Figure 1.21b), her right maxillary vermillion is elevated higher by her facial musculature than her left. Also notice that her mandibular left lip vermillion is pulled down more than her mandibular right. Lynn's asymmetric smile is due to functional (neuromuscular) canting of her upper lip.

Q: Asymmetrical smiles result from structural or functional asymmetries. How does this impact treatment options?

A: If an asymmetrical smile results from structure (overgrowth of the maxilla on one side, overeruption of teeth on one side), then treatment to correct the asymmetrical structure may be appropriate. Treatment may include a differential Le Fort I maxillary osteotomy (combined with a mandibular osteotomy) to correct the occlusal cant in both jaws, or it may include orthodontic treatment only using TADs to intrude the maxillary or mandibular dentition on one side of the arch (with vertical elastics to correct the corresponding occlusal plane in the opposing jaw). On the other hand, if the asymmetrical smile results from function, then treatment that changes structure would be inappropriate. Treatments such as botulinum toxin injections can be considered to address excess hyperactivity of smiling musculature, but patients can experience unesthetic perioral animation effects with such treatments [31, 32]. Finally, remember that having an occlusal cant may not even be a



Figures 1.20 Patients with asymmetrical smiles due to structural maxillary occlusal cants: (a, d) Tanner, (b, e) Olivia, and (c, f) Trevor.

Figure 1.21 (a, b) Patient with an asymmetrical smile due to unbalanced smile musculature function.

concern for a patient, and as the doctor you must be careful not to draw undue attention to problems that the patient may not be concerned with.

Q: Christina (Figure 1.22) presents with an asymmetrical smile. Is her asymmetry due to structure or function (trick question, look carefully)?

A: Her smile asymmetry is due to *both* structure and function. If you look carefully at her occlusal plane relative to her interpupillary line (Figure 1.22b), then you can see that she has a right to left maxillary occlusal cant (structure). Further, if you look very carefully at her maxillary lip vermillion at rest, you will also notice that it has a very slight right to left cant (structure, Figure 1.22a). Finally, if you look at her during a posed smile, you see that her maxillary lip is raised slightly more on her left than right (function, Figure 1.22b). Her smile asymmetry is due to both structure and function.

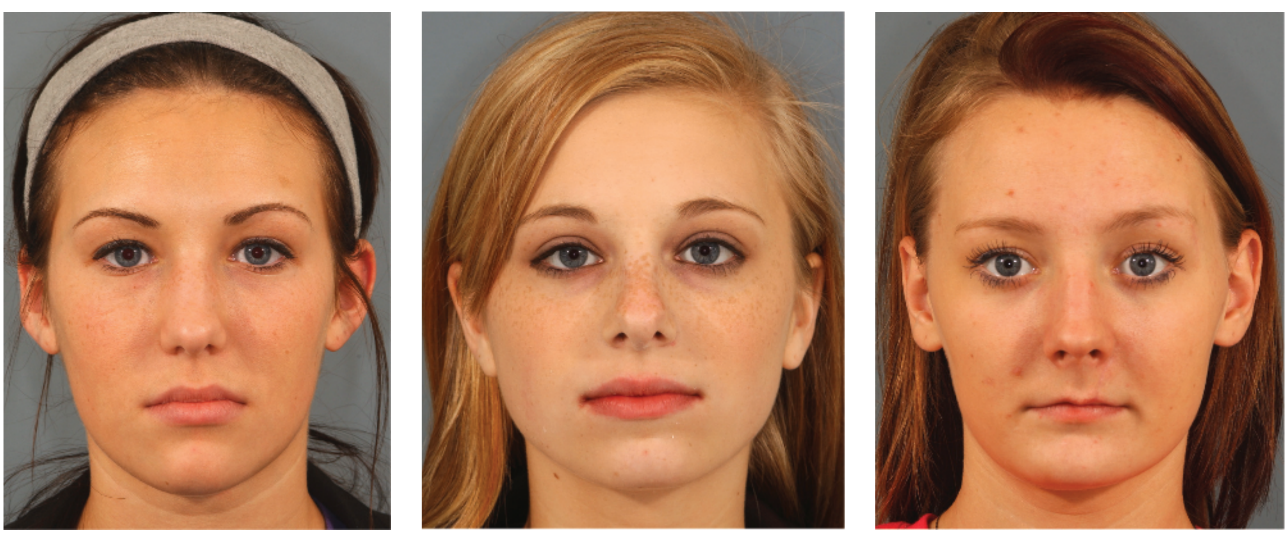
Q: What other features affect smile and lower facial esthetics?

A: Features that affect smile and lower facial esthetics include the following:

- Lip fullness: Scott et al. [33] reported that thicker vermillion (Figures 1.23a, b) are considered more attractive (and more feminine) than thinner vermillion (Figure 1.23c), which are considered less attractive (and more masculine).
- Central incisor midline position: The patient in Figure 1.24a demonstrates ideal central incisor midline positions: maxillary and mandibular central incisor midlines are coincident and in line with the facial midline. The patients in Figures 1.24b, c have midlines that are deviated to the right of their facial midlines by 2 mm and 3–4 mm, respectively. Whereas a recent systematic review reported a dental midline deviation of up to 2.2 mm as being acceptable to laypersons [34], other studies suggest that the majority of laypersons are unable to detect midline deviations up to 4 mm [30, 35].

(a) (b)

Figure 1.22 Christina presents with an asymmetrical smile.



(a) (b) (c)

Figure 1.23 Patients with varying thicknesses of lip vermillion.

(a) (b) (c)

Figure 1.24 Incisor midline positions: (a) ideal, (b) 2 mm right deviation, (c) 3–4 mm right deviation.

(a) (b)

Figure 1.25 Buccal corridors: (a) initially large, (b) reduced following SARME

- Buccal corriodor size: Look at the patient in Figure 1.25. Initially (A), he had a maxillary transverse deficiency (constricted maxilla) and accompanying large buccal corridors (dark intraoral spaces between his cheeks and posterior teeth). Following SARME and orthodontic treatment (Figure 1.25b), his buccal corridors were dramatically reduced. In your opinion, is there an esthetic difference in his smile? Clearly, reduction in his buccal corridors (maxillary expansion) improved his smile esthetics. Although there has been disagreement regarding the influence of buccal corridors on smile esthetics [36], we found that *laypersons judge large buccal corridors (narrow maxillary arches) to be less esthetic than small buccal corridors (broad maxillary arches)* [37]. Large buccal corridors should be included in your problem list.

Q: Considering the fact that the two most important factors in an attractive face are the eyes and the smile [38], the esthetic impact of a well-treated occlusion (Figures 1.26a, b) compared to an untreated malocclusion (Figure 1.26c) is immeasurable. What are some of the factors contributing to an esthetically pleasing occlusion as seen in the frontal view?

A: Tooth shade (ideally, approaching the whiteness of the patient's orbital sclera), absence of crowding, alignment of incisal edges/cusp tips along arch of the occlusal plane, coincidence of maxillary and mandibular midlines, maxillary central incisor crowns having equal widths, maxillary central incisor crowns having ideal proportions (width/height ratio of 4/5) and a lack of wear/attrition, golden proportion of maxillary anterior teeth from anterior to posterior, proper long axis alignment of anterior teeth, absence of a reverse smile arc, ideal overbite and overjet, equal levels of maxillary central incisor gingival margins with canine gingival margins at about the same level and lateral incisor gingival margins slightly incisal, and absence of gingival embrasures (black triangles) or incisal embrasures [5, 30, 31, 35, 39, 40].

Q: Examine each occlusion in Figures 1.27a–o (frontal views). What unesthetic features can you identify in each image?

A: Unesthetic dental features include the following for each of Figure 1.27a–o:

- (a) Dark shade, significant generalized anterior tooth wear, edge-to-edge incisal relationship (lack of normal overbite and overjet), anterior crossbite of right central incisors.
- (b) Severe maxillary central incisor inclination, excessive OB with overerupted maxillary central incisors, gingival margins of maxillary central incisors are stepped down relative to the maxillary lateral incisor gingival margins, dark yellow shade, labial crown torque maxillary left canine, possible gingival recession/caries along the maxillary central incisor gingival margins.
- (c) Dark shade, uneven maxillary central incisor widths, maxillary central incisor crown widths equal to or greater than the incisor crown heights (width/height ratio of 4/5 violated) leading to a disruption in golden proportion of maxillary anterior teeth from anterior to posterior, midlines are not coincident, maxillary midline diastema, uneven maxillary central incisor gingival margins, maxillary left central incisor gingival margin lower than lateral incisor gingival margins, significant tip of maxillary right lateral incisor edge, significant cuspal wear of maxillary right canine.
- (d) Anterior open bite, reverse smile arc, excessive mesial crown tip of maxillary central incisors, malalignment of maxillary and mandibular anterior teeth, isolated white spot hypocalcified areas, excessive maxillary canine lingual crown torque (maxilla appears to be constricted).

(a) (b) (c)

Figure 1.26 Dental features impacting esthetics: (a–b) well-finished occlusions, (c) a malocclusion possessing many unesthetic features.

(a) (b) (c) (d) (e) (f)

Figure 1.27 (a–o) Unesthetic dental features.

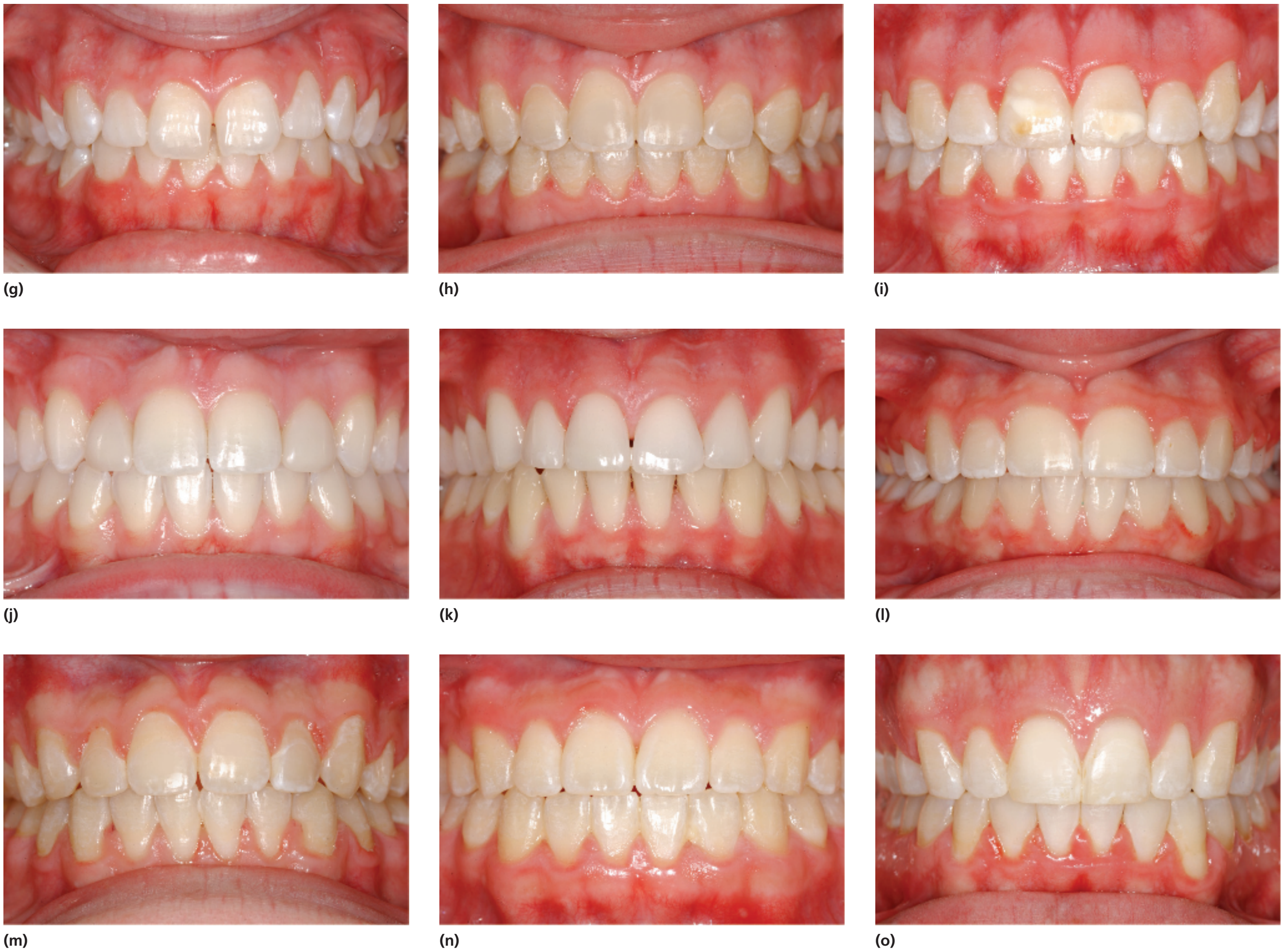


Figure 1.27 (Continued)

- (e) Maxillary central incisor crowns are nearly as wide as they are long (width/height ratio of 4/5 violated) leading to a disruption in golden proportion of maxillary anterior teeth from anterior to posterior, midlines are not coincident, edge-to-edge incisal relationship (lack of minimal overbite and overjet), incisal embrasures, gingival recession over roots of maxillary canines, shade (dark coloration of maxillary central incisors, white spots of maxillary lateral incisors).
- (f) Mottled shade, incisal embrasures, uneven and worn maxillary central incisal edges, malalignment of mandibular incisors.
- (g) Excessively deep OB, maxillary central incisor mesial root tip, maxillary left lateral incisor gingival margin apical to canine gingival margin.
- (h) Dark shade, uneven central incisor gingival margins.
- (i) Discolored central incisors, small incisal embrasure between maxillary central incisors.
- (j) Midlines not coincident, maxillary central incisor gingival margins slightly uneven, mesial root tip of maxillary right lateral incisor.
- (k) Maxillary central incisors gingival embrasure (black triangle), maxillary central incisor gingival margins uneven, maxillary left central incisor stepped down, incisal embrasure between maxillary right lateral incisor and mandibular right canine, gingival embrasures between mandibular incisors.
- (l) Maxillary central incisor crowns are wider than they are long (width/height ratio of 4/5 violated) leading to a disruption in golden proportion of maxillary anterior teeth from anterior to posterior, midlines are not coincident, right canines do not appear coordinated.
- (m) Mild reverse smile arc, central incisor incisal embrasure.
- (n) Excess mesial crown inclination of maxillary left central incisor, incisal embrasures, midlines not coincident.
- (o) A few white spots, maxillary right central incisor is wider than the maxillary left central incisor.

Let's complete this chapter by examining four patients.

Q: Brad presents for a consult because of his deep bite (Figure 1.28). What features would lead you to classify Brad as a dental deep bite patient? What features would lead you to classify Brad as a skeletal deep bite patient?

A: See Table 1.1

Q: Would you classify Brad as a dental deep bite patient, a skeletal deep bite patient, or a combination of both?

A: Dental deep bite. Brad's 80% OB resulted from mandibular incisor overeruption and not from skeletal growth. In the spectrum of vertical facial relationships, Brad lies in the normal skeletal range.

Q: What features would lead you to classify Robert (Figure 1.29) as a dental deep bite or a skeletal deep bite patient?

A: See Table 1.2

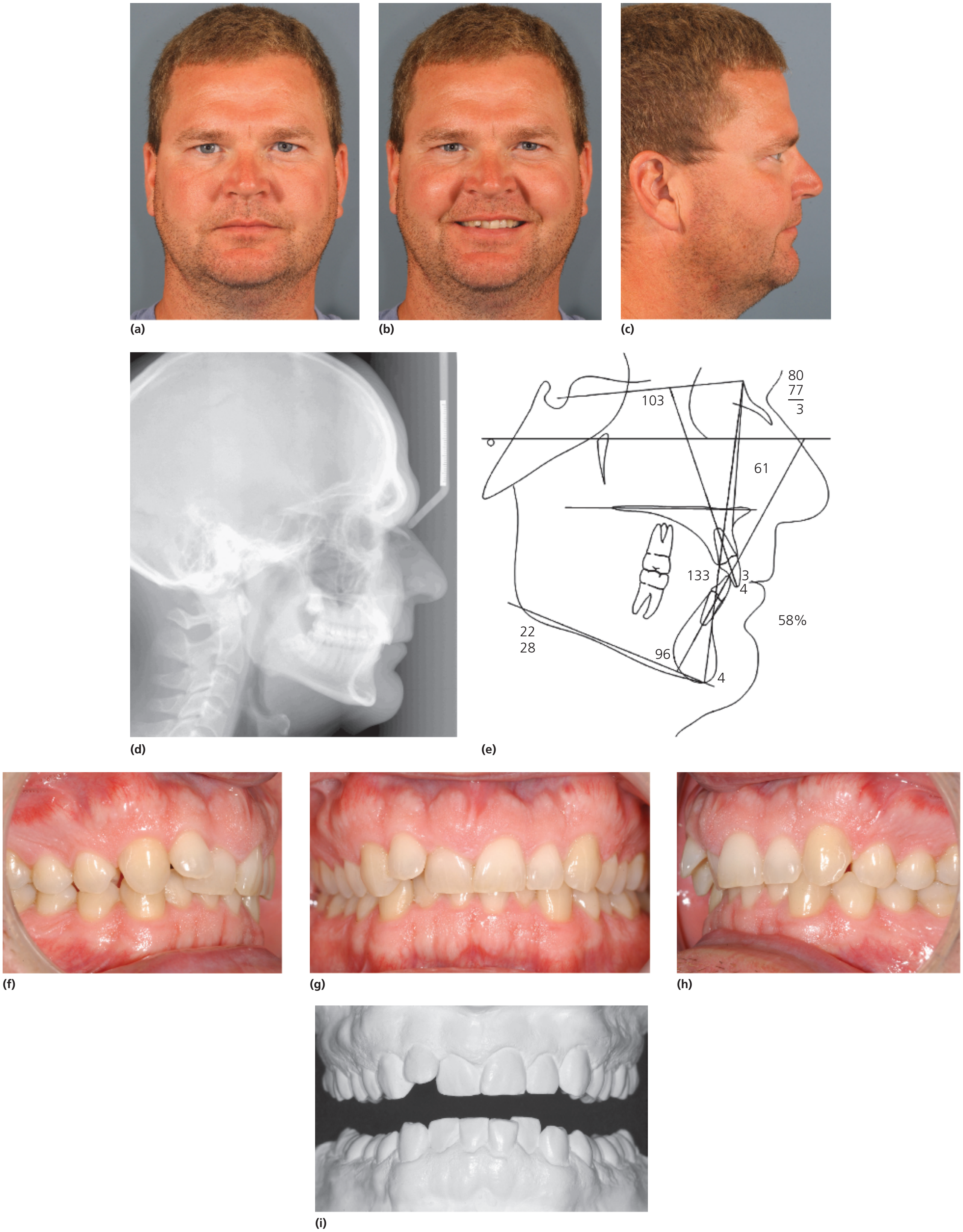


Figure 1.28 Initial records of Brad: (a–c) composite facial photographs, (d) lateral cephalograph, (e) cephalometric tracing, (f–h) intraoral photographs, and (i) models separated.

Table 1.1 Dental and skeletal features of Brad (Figure 1.28).

Skeletal deep bite features	Dental deep bite features
	• Soft tissue LAFH WRN • Skeletal LAFH mildly long (LAFH/TAFH 58%, > 1 standard deviation longer than ideal, 55%) • MPA WRN (FMA = 22° and SNMP = 28°) • Maxillary incisal display WRN (posed smile and as seen on his cephalograph) with maxillary central incisors neither stepped up nor stepped down (Figure 1.28i) • Mandibular incisors overerupted (deep curve of Spee)

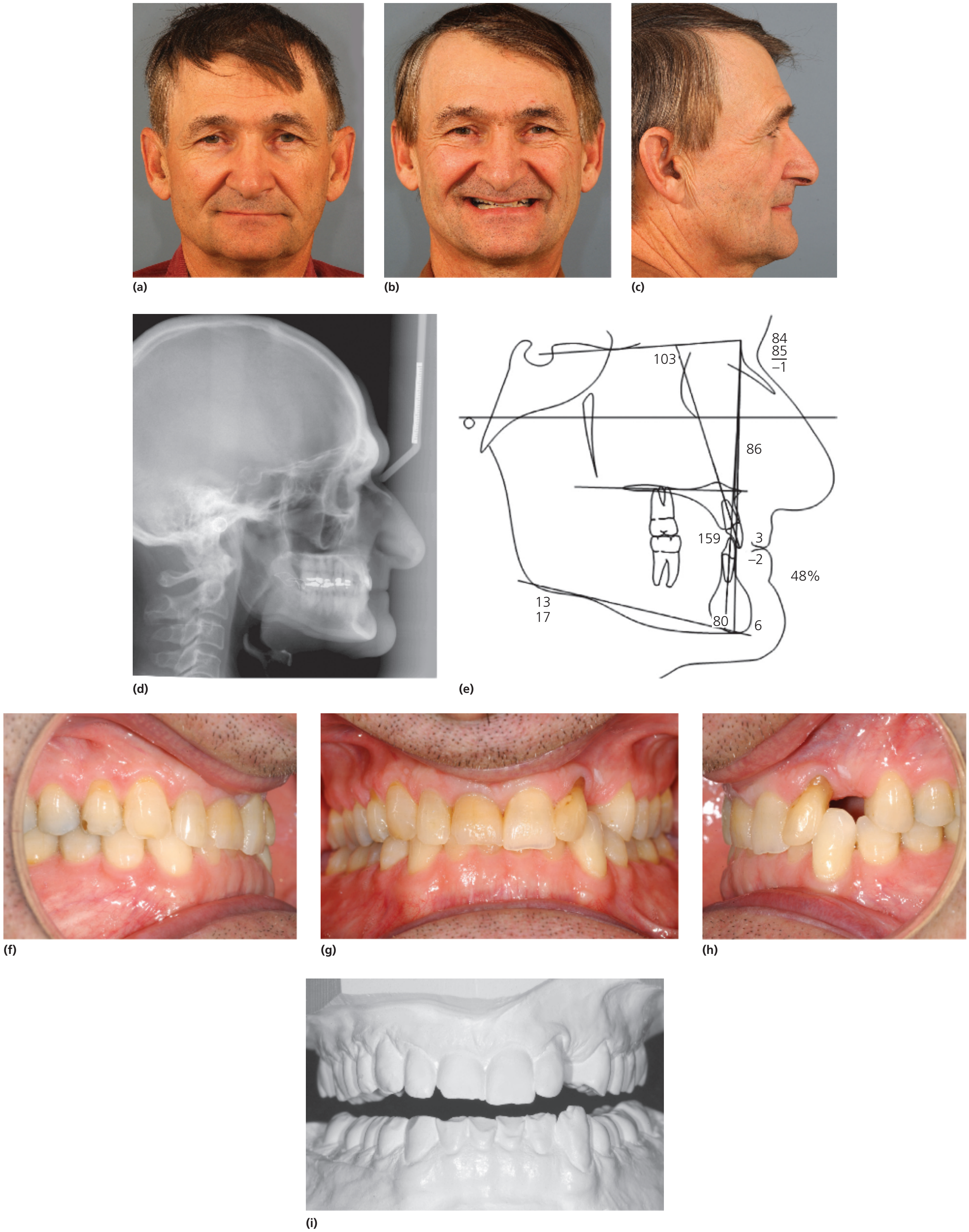


Figure 1.29 Initial records of Robert.

Table 1.2 Skeletal and dental features of Robert (Figure 1.29).

Skeletal deep bite features	Dental deep bite features
• Short soft tissue LAFH • Overclosed appearance • Short skeletal LAFH • Flat MPA • SN, FH, and MP nearly parallel • Maxillary incisal display inadequate (posed smile and cephalometrically) *with* maxillary incisors stepped down	• Overerupted maxillary incisors

Q: Would you classify Robert as a dental deep bite patient, a skeletal deep bite patient, or a combination of both?

A: Robert is a patient with a combination skeletal and dental deep bite. The fact that Robert's facial skeleton never developed vertically to keep pace with his facial soft tissue drape is clearly illustrated by Figures 1.30a–b. Figure 1.30a is, once again, Robert's initial frontal photo in maximal intercuspation. He looks overclosed. In Figure 1.30b, Robert was asked to separate his jaws until his lips were just touching. This is where his facial soft tissue drape is fully expressed (vertically). Do you see the difference in his appearance? In other words, *his facial skeletal vertical development was dramatically less than his facial soft tissue vertical development*. As Robert closes, his mandible rotates upward and forward until his teeth are in occlusion and his facial soft tissues are compressed, leaving him with an overclosed appearance.

This lack of vertical skeletal development also affects his smile (Figure 1.29b). Figure 1.30c illustrates his lack of incisal display at rest even though he has stepped-down maxillary incisors. In the spectrum of vertical facial development, Robert would be plotted in the extreme end of skeletal deep bite. But, he also has a dental deep bite due to overerupted maxillary incisors.

Q: What features would lead you to classify Kelly (Figure 1.31), a 17-year-old, as a dental open bite or a skeletal open bite patient?

A: See Table 1.3

Q: What questions should you ask Kelly? What should you check for?

A: You should check for the following:

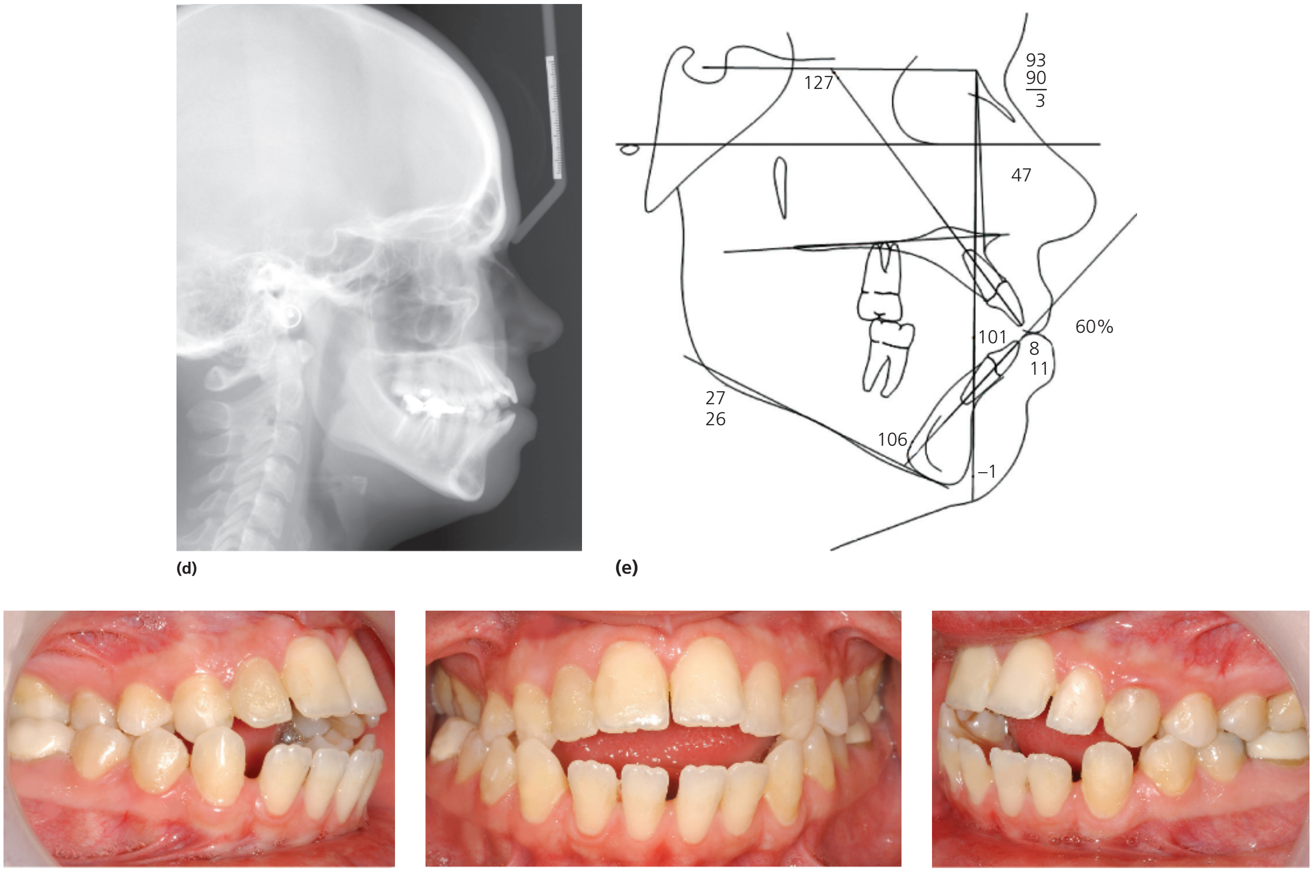
- Although she exhibits lip competence, you should confirm that Kelly is not straining (mentalis strain) to achieve lip competence. This was not done. However, she also exhibits lip competence in her lateral cephalometric radiograph, which is a good sign. Many times, patients who strain to close their lips when photographs are taken will relax when the radiographs are being made. Even if she was straining to achieve lip competence, this could be due to the fact that her incisors are so proclined that her lips must strain to reach around them (and not due to the fact that her LAFH is long).
- Check for an anterior tongue interposition habit by asking Kelly to close her eyes. Then, ask her to open her eyes (to distract her) while you simultaneously draw her lower lip down and look for her tongue to be positioned between her anterior teeth. You find that she does not have an anterior tongue interposition habit. Ask her about a possible digit habit. She says she quit her thumb habit for a number of years but currently sucks her thumb when sleeping at night (the etiology of her dental open bite).

(a) (b) (c)

Figure 1.30 Robert: (a) initial photo again, in maximal intercuspation; (b) with jaws separated until lips relaxed and just touching; (c) opened until lips relaxed and parted to demonstrate degree of incisal display at rest.

(a) (b) (c)

Figure 1.31 Initial records of Kelly.



(d) (e)

(f) (g) (h)

Figure 1.31 (Continued)

Table 1.3 Skeletal and dental features of Kelly (Figure 1.31).

Skeletal open bite features	Dental open bite features
• Long soft tissue LAFH • Long skeletal LAFH (LAFH/TAFH 60%)	• Lip competence (no ILG, mentalis strain?) • MPA WRN • Stepped-up maxillary incisors • Stepped-down mandibular incisors • Open bite limited to anterior teeth with the classic "football shape" indicative of an anterior functional habit

Q: Would you classify Kelly as a dental open bite patient, a skeletal open bite patient, or a combination of both?

A: Kelly should be considered a combination of both. She clearly presents with a dental (functional) open bite due to an active thumb habit. She also exhibits features of a skeletal open bite (long soft tissue LAFH and long skeletal LAFH) but has a normal MPA. In fact, *Kelly's midface appears short (Figure* 1.31*a), which contributes to the findings of long soft tissue LAFH and long skeletal LAFH even though she looks normal*.

Q: Do you recommend *treating* a patient like Kelly as a dental open bite, a skeletal open bite, or a combination of both?

A: This really comes down to questions of how far from normal do you feel Kelly's vertical development is, how much her vertical growth contributed to her open bite, and how much future growth she has left. We concluded that Kelly has a short midface, which contributes to her LLAFH features. In the spectrum of vertical, Kelly lies in the fairly normal skeletal range. Based upon this conclusion, we treated her as a dental open bite. This was reasonable, especially since she was a 17-year-old and had completed growth.

Q: What features would lead you to classify Owen (Figure 1.32) as a dental open bite or a skeletal open bite patient?

A: See Table 1.4

Q: What should you check for? What questions should you ask Owen?

A: First, discuss with Owen and his parents his breathing history. Is he an obligatory mouth breather? Does he snore at night? Does he stop breathing at night (sleep apnea)? He and his parents deny any history of breathing difficulty, and he breaths nasally. Next, check Owen for an anterior tongue interposition habit. You find he has one (Figure 1.33). He denies a history of a thumb or other digit-sucking habit.

Q: Would you classify Owen as a dental open bite patient, a skeletal open bite patient, or a combination of both?

A: A combination of both. In the spectrum of vertical, Owen would be plotted toward the extreme of skeletal open bite, but his dental (functional, tongue interposition habit) component must also be addressed during treatment.

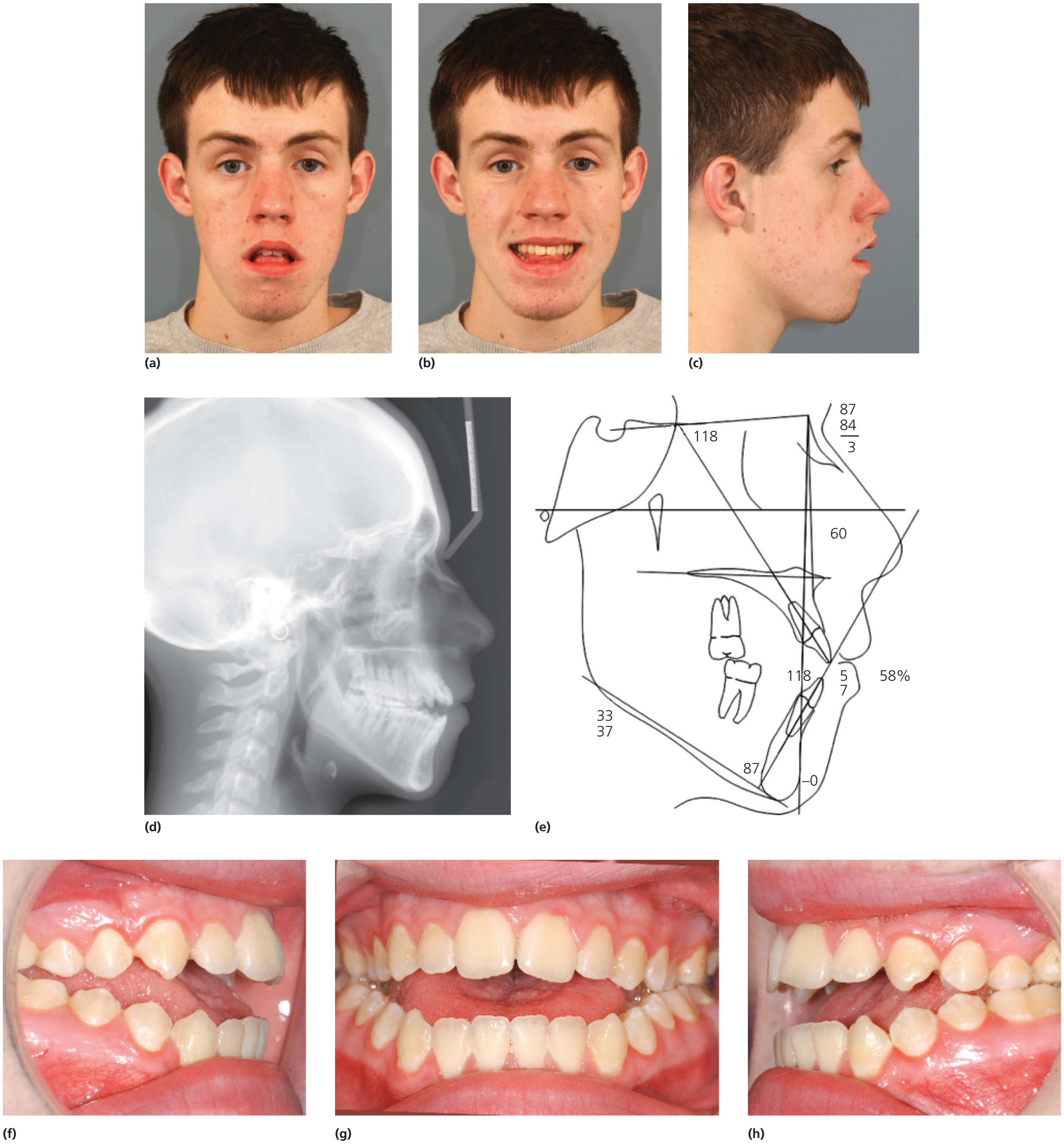


Figure 1.32 Initial records of Owen.

Table 1.4 Skeletal and dental features of Owen (Figure 1.32).

Skeletal open bite features	Dental open bite features
• Long soft tissue LAFH • Long skeletal LAFH • Large ILG • Steep MPA • Maxillary molar root apices erupted well below the palatal plane • Open bite extending to first molars	• Mandibular anterior teeth stepped down in an AP reverse curve (Figures 1.32f and 1.32h)

Figure 1.33 Tongue interposition habit of Owen.

Growth Foundations

Essential to the practice of our specialty is an in-depth understanding of craniofacial growth and development. Most patients are evaluated for orthodontic treatment at a time when their facial structures, particularly the maxilla and the mandible, have their greatest growth potential. Thus, exhaustive knowledge of postnatal maxillary and mandibular growth, heritable and environmental influences on jaw growth, and potential response of the growing jaws to intervention are crucial in order for the specialist to treat patients and to evaluate treatment outcomes. This section provides a brief review of important craniofacial growth and development concepts, with an emphasis on the vertical dimension. A comprehensive foundation can be obtained in other textbooks [41–44].

Q: In general terms, how does the facial skeleton grow?

A: The growing facial skeleton is a composite of *cranial base growth, individual facial bone growth, and drift of the teeth*. Each of these components has its own unique pattern of change in space and time but does not generate its unique characteristics independently. Instead, change in size and shape of each growing facial component is the result of complex genetic, spatial, temporal, and functional interrelationships among the components [41–43]. A composite of these growth changes seen in the sagittal view is depicted in Figure 1.34.

Q: What are the underlying mechanisms responsible for facial bone size and shape changes?

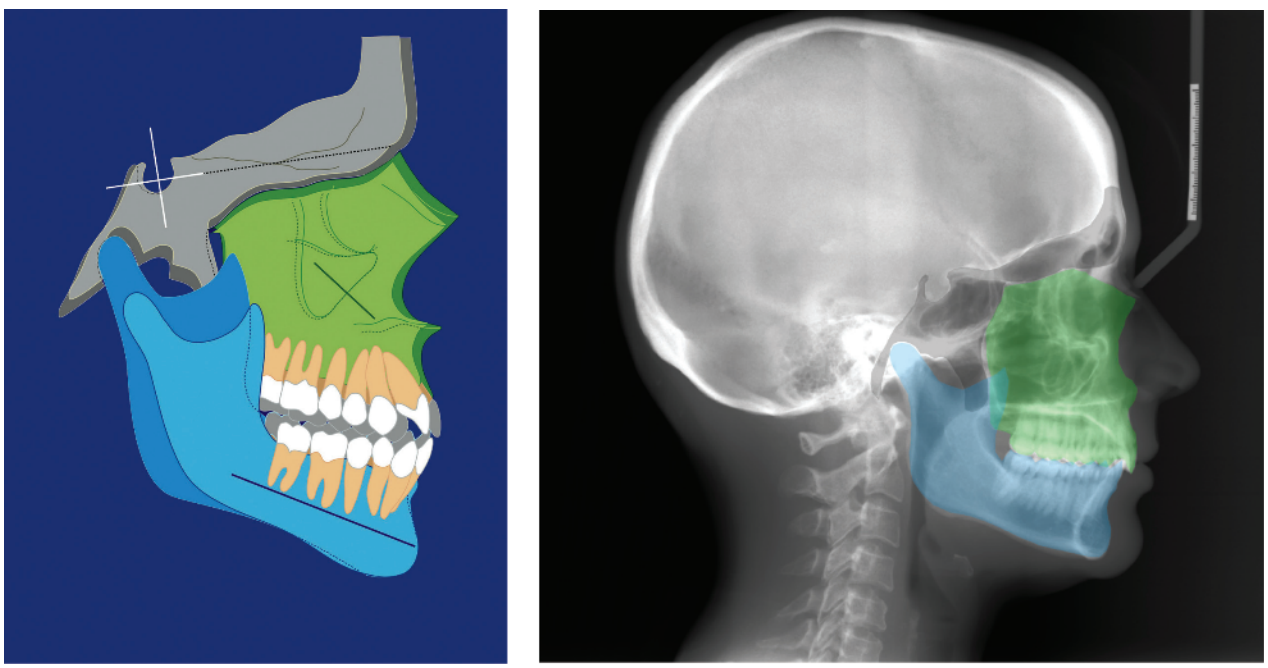
A: Underlying mechanisms include the following [42]:

- Appositional bone growth at the facial bone *sutures* (sutural growth) causing spatial displacement. This displacement is termed *primary displacement* of the facial bones.
- Appositional bone growth at the facial bone *surfaces* (periosteal growth) causing individual facial bone enlargement and change in shape. This is defined as facial bone *surface drift*.
- Interstitial growth of cartilage in the synchondroses of the cranial base and nasal septal cartilage associated with spatial displacement of the adjoining bones of the face. The displacement of the adjoining facial bones is termed *secondary displacement*.
- Chondrogenesis and endochondral ossification of secondary cartilage of the mandibular condyle associated with primary displacement of the mandible.

Important concept: With the possible exception of secondary displacement mediated by growth at synchondroses and the nasal septal cartilage [41], bone displacement and drift does not cause bones to "push" against each other. Facial bone displacement and drift is, by and large, driven by forces created by growth of the enveloping soft tissues.

Q: How do temporal relationships among the growing components of the facial skeleton affect maturation of facial size and shape?

A: Maturation of facial size and shape occurs, by and large, along a superior–inferior gradient [42]. Cranial structures mature first, followed by cranial base structures, then facial bones,



(a) (b)

Figure 1.34 (a) Changes in size, shape, and position of the major skeletal components contributing to the growth of the face as viewed in the sagittal. Approximate outline of the cranial base (gray), the maxilla (green), and the mandible (blue) as seen on a lateral cephalogram. Darker shades of color indicate the approximate location and direction of changes in size and shape due to drift and displacement of the cranial base, maxilla, mandible, and teeth during growth (Duterloo HS, Planche PG. Handbook of Cephalometric Superimposition. Hanover Park: Quintessence, 2001. Figure 3.23. Reprinted with permission from Quintessence Publishing Company Inc, Chicago.) (b) The location of the cranial base (gray), maxilla (green), and mandible (blue) on a lateral cephalogram.

and finally the mandible completes maturation after the maxilla. Aberrations in the temporal sequence result in significant craniofacial deformities. Some have characterized the gradient with distinct tiers (cranial, cranial base, facial), while others have argued for a continuous gradient with maturation of anatomic structures between tiers showing overlap [45]. Adding to the complexity of facial growth, the mediolateral structures of the cranial base do not mature in the same "tier," or at the same time, as those along the superior–inferior gradient. Midline cranial base structures, presumably influenced by maturation of the spheno–occipital synchondrosis, appear to mature well in advance of structures in the lateral cranial floor. Lateral cranial floor structures mature in a similar time frame to the mandible, suggesting a possible functional integration of the masticatory apparatus and the development of lateral cranial floor [45].

Q: How does the cranial base grow? How does cranial base growth contribute to facial growth? Provide a detailed review.

A: The cranial base provides the foundation for postnatal facial development. In the midline, the cranial base undergoes lengthening by primary displacement to accommodate the growing brain. Frontal and temporal lobe brain expansion corresponds with the endochondral bone replacement of cartilage in the sphenoethmoidal and spheno–occipital synchondroses. Frontal and temporal lobe expansion also drives anterior and middle cranial fossae anteroposterior, vertical, and lateral enlargement via sutural growth and drift. The resulting forward movement of the anterior cranial base has a forward displacement (secondary) effect on the nasomaxillary complex. The pterygoid processes move downward by drift and displacement. As frontal lobe expansion and sphenoethmoidal synchondrosis growth cease (approximately age 7), temporal lobe expansion continues, displacing the frontal lobe anteriorly with a continued secondary displacement effect on the anterior cranial base and the nasomaxillary complex. Temporal lobe expansion laterally and vertically also influences the position of the glenoid fossae, which are displaced posteriorly and inferiorly [46] until approximately age 15–16 years [45]—and to a greater degree during adolescence compared to childhood [47]. In general, a more obtuse cranial base angle (angle formed by Nasion–Sella and Sella–Basion) and/or a long anterior (relative to posterior) cranial base length is associated with a skeletal Class II relationship. A more acute cranial base angle and/or a short anterior cranial base length is associated with a skeletal Class III relationship [44]. A composite of these growth movements in the sagittal view is depicted in Figure 1.35.

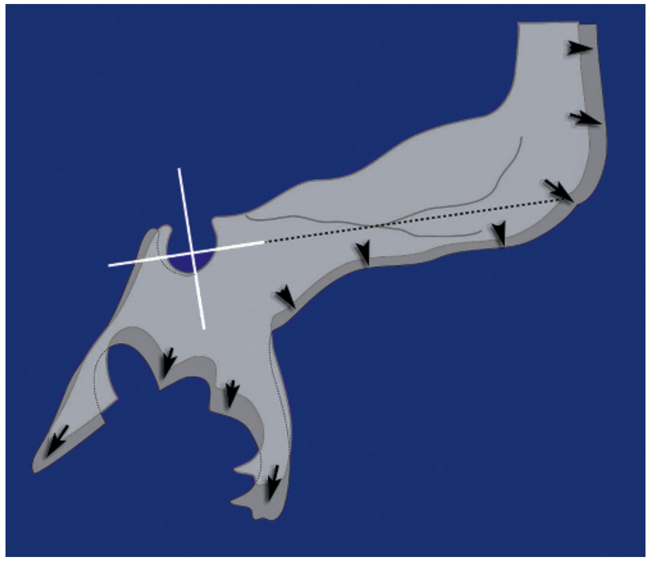


Figure 1.35 Changes in size, shape, and position of the cranial base during growth—as viewed in the sagittal. The intersection of the white lines indicates Sella point. Darker gray and dark arrows indicate approximate magnitude and direction of changes due to growth-driven drift and displacement. (Duterloo HS, Planche PG. Handbook of Cephalometric Superimposition. Hanover Park: Quintessence, 2001. Figure 3.25. Reprinted with permission from Quintessence Publishing Company Inc, Chicago.)

Q: How does the nasomaxillary complex grow?

A: Downward and forward growth of soft tissues creates tension that drives downward and forward nasomaxillary complex movement. That is, as a result of this tension, primary displacement occurs by growth at the sutures joining the maxilla with the frontal, zygomatic, sphenoid, ethmoid, nasal, lacrimal, and palatine bones. This is accompanied by backward downward drift at the posterior maxilla (tuberosity) with new bone available for eruption of permanent molar teeth and downward-directed drift of the hard palate. Nasomaxillary surface drift occurs in response to changing soft tissue and facial bone spatial relationships. Palatal inferior drift and development/vertical drift of permanent teeth contribute approximately 66% of the vertical height change in the maxilla [48, 49]. Vertical drift of maxillary teeth is controlled by forces of eruption (exact mechanism unknown) and forces opposing eruption (e.g., functional/parafunctional loading with mandibular teeth). The contribution of nasal cartilage interstitial growth to downward forward translation of the nasomaxillary complex (secondary displacement) remains equivocal and is thought to have early postnatal influence that diminishes as other displacement forces supersede in later postnatal development [42]. A composite of these growth movements in the sagittal view is depicted in Figure 1.36.

Q: How does the mandible grow?

A: The mandible is carried downward and forward by growth expansion of the enveloping soft tissues. In response, the mandible grows upward and backward largely by a combination of bone surface drift (membranous ossification) and primary displacement at the condyle (endochondral replacement of cartilage).

Q: The mandible consists of five developmentally important regions. What are they, and what role does each play?

A: The mandible consists of the condyle, coronoid process, ramus, corpus, and alveolar process (Figure 1.37). The role of each is as follows:

- Condyle: Along with the ramus, it is a major site of compensatory mandibular growth during downward and forward facial soft tissue expansion. It provides articulation with the temporal bone to mediate function between the maxillary and mandibular teeth. It performs these roles by virtue of the

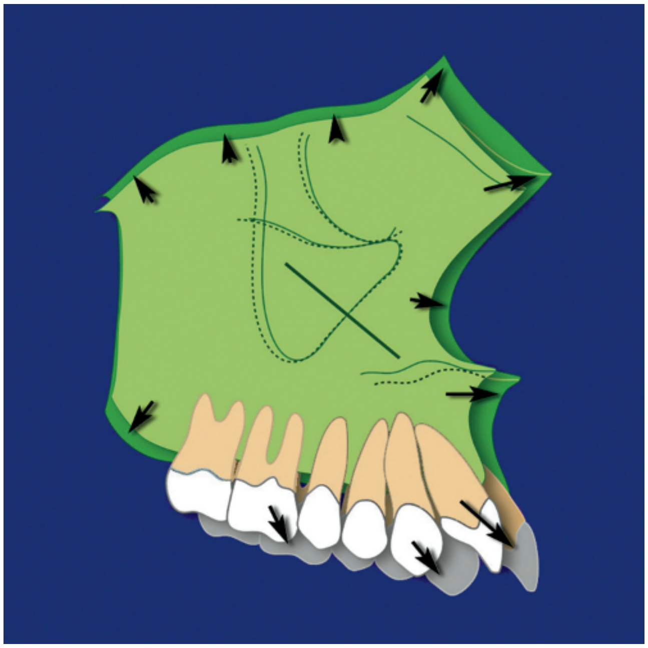


Figure 1.36 Changes in size, shape, and position of the maxilla and maxillary teeth during growth—as viewed in the sagittal. Darker color and dark arrows indicate approximate magnitude and direction of changes due to growth-driven drift and displacement. (Duterloo HS, Planche PG. Handbook of Cephalometric Superimposition. Hanover Park: Quintessence, 2001. Figure 3.29. Reprinted with permission from Quintessence Publishing Company Inc, Chicago.)

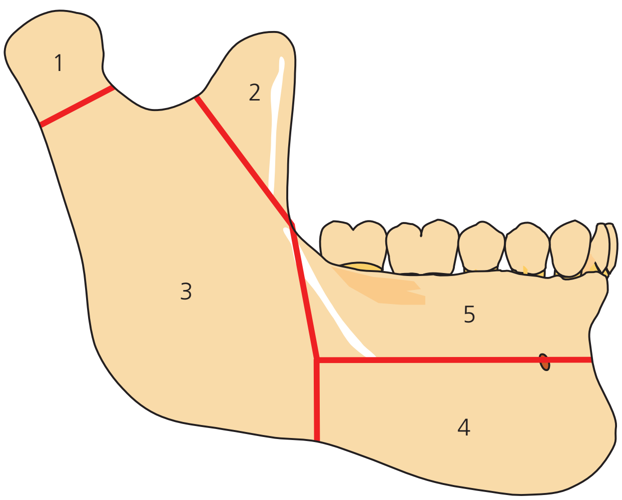


Figure 1.37 The mandible consists of five regions: 1, condyle; 2, coronoid process; 3, ramus; 4, corpus; 5, alveolar process.

condylar cartilage, a specialized secondary cartilage allowing endochondral bone growth to occur in the presence of masticatory compressive forces on the condylar head. The arrangement of secondary cartilage allows condylar growth to be adaptive, facilitating change in growth direction as pressure and tension on the condylar head change, and to maintain the relationship of the condyle and glenoid fossa as the mandible is carried downward and forward.

- Coronoid process: It provides attachment for the temporalis muscle, which supports the process of mastication. The coronoid process undergoes extensive drift upward, backward,

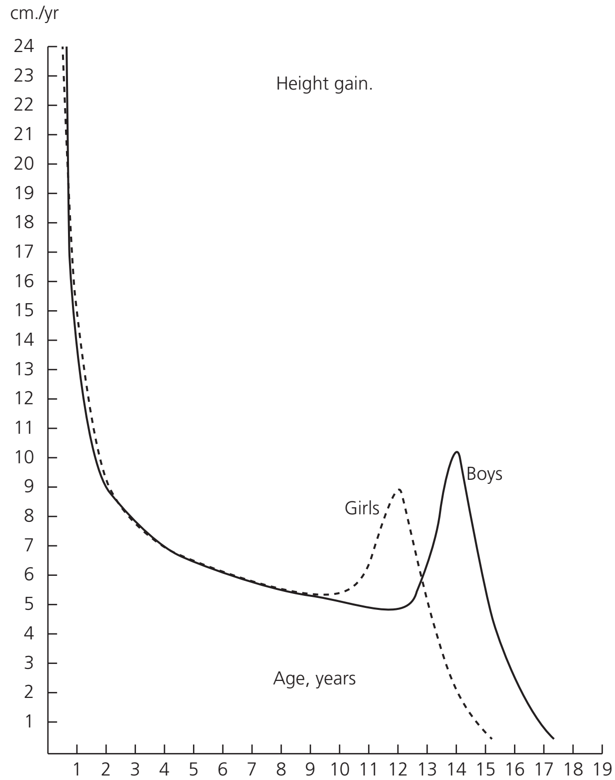


Figure 1.38 Plot of change in growth velocity with age. (Tanner JM, Whitehouse RH, Takaishi M. Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children, 1965. Arch Dis Child. 1966 Oct;41(219):454–71. Reproduced with permission from BMJ Publishing Group Ltd.)

and laterally during the downward and forward translation of the mandible.

- Ramus: As a major site of adaptive growth and growth compensations, it is of equal importance to the condyle during downward and forward translation of the mandible. In order to maintain the appropriate spatial and functional relationship of the mandibular dentition with the maxillary dentition during growth, the ramus undergoes complex changes in width, breadth, height, and uprighting (gonial angle becoming more acute). Although it is common to encounter the term "condylar growth," signifying the condyle as the most important mandibular growth center, the importance of ramus growth is coequal in establishing balance or imbalance in mandibular position [42]. Also, the ramus provides attachment for the masseter and medial pterygoid muscles.
- Corpus: It supports the alveolar process and teeth during development and mastication. It provides attachment for many muscles, including the mentalis muscle.
- Alveolar process: It provides a bony housing for the eruption, drift, and function of teeth.

Q: Is facial growth coordinated with statural growth?

A: Yes. By and large, the temporal sequence of maturation is synchronized throughout the body. General body (somatic) growth, measured by change in stature with time (growth velocity), is an indicator of velocity changes in craniofacial dimensions during growth [50, 51]. This is in contrast to the neurocranium, which is more closely coordinated with neural growth.

Q: Do facial and statural growth proceed with constant velocity during development?

A: No. A typical plot of statural growth velocity (Figure 1.38) shows that change is not uniform. One-half of adult stature is attained within the first two years of life during a rapid deceleration of growth velocity. After which, a much slower deceleration of growth velocity occurs until the onset of puberty. The inflection point where deceleration of growth velocity changes to acceleration is the beginning of pubertal growth acceleration, often termed the "pubertal growth spurt." This pubertal growth acceleration increases growth velocity to a maximum, termed the peak height velocity (PHV), followed by a rapid deceleration in growth velocity [50, 52].

Q: What is the duration of pubertal growth acceleration?

A: Approximately 2–2.5 years [50].

Q: Does pubertal growth acceleration occur at the same age for males and females?

A: No. On average, females reach PHV at age 11.5–12 years and males reach PHV at age 14 years [50, 52, 53].

Q: Is peak facial growth velocity coordinated with PHV?

A: Yes. On average, females reach peak facial growth velocity at 10.9–12.3 years and males reach peak facial growth velocity at 14.1–14.3 years, [54–56] supporting the close association between change in stature and facial dimensions [50].

Q: How much growth occurs during pubertal growth acceleration?

A: On average, males add 20 cm in stature and females add 16 cm in stature. On average, the chin moves downward and forward, away from the cranial base (measured Sella to Gnathion), 1.8–2.0 cm in males and 1.1–1.2 cm in females [57].

Q: Are males and females similar in the downward and forward facial growth pattern during pubertal growth acceleration?

A: Possibly not. A recent longitudinal study of 111 untreated French-Canadian females demonstrated that during the period of pubertal growth acceleration, beginning at approximately 11.5 years of age, measures of vertical skeletal change showed acceleration, but measures of horizontal skeletal change did not. Moreover, in this sample, measures of horizontal skeletal change decelerated during adolescence. For this sample, female pubertal growth acceleration did not provide anteroposterior improvement in chin projection [58].

Q: Is the pattern and rate of forward and downward growth during pubertal growth acceleration the same as found prior to puberty?

A: No. Studies have shown that horizontal and vertical translation of the chin varies between prepubertal and pubertal growth periods. [16, 59–63] Growth-driven *anterior* movement of the chin has a greater velocity during childhood compared to the period of adolescence. In contrast, growth-driven *inferior* movement of the chin has less velocity during childhood compared to the period of adolescence. These findings suggest a tendency for facial height to increase more than facial depth during adolescence compared to childhood [63–65].

Q: Is there a measurable indicator of pubertal growth acceleration and PHV?

A: There is no one best indicator of pubertal growth acceleration and PHV. Maturation of hand–wrist and cervical spine bones has been studied as potential predictors of PHV but give unreliable estimates of PHV timing and peak facial growth velocity [54, 66–71]. Skeletal development of hand–wrist bones has been shown to be useful in estimating whether the pubertal growth acceleration has already occurred [54], but the method of assessing a patient's skeletal development by hand–wrist radiography against a radiographic atlas of normal hand–wrist skeletal development has low sensitivity in predicting whether the pubertal growth acceleration is impending or ongoing [50, 66]. That being said, measurement of growth acceleration itself, measured as *change in stature with time, may be considered the least invasive and most reliable predictor of pubertal growth acceleration and PHV* [54, 72].

Q: Can other periods of growth acceleration occur before puberty?

A: Yes. Statural growth acceleration has been reported to occur in the age range 6.5–8.5 years for some individuals (termed the midgrowth spurt) [73, 74]. Reports on the magnitude of change in statural growth velocity vary in characterization from "less deceleration" to mild acceleration, and are of considerably lower magnitude, compared to pubertal growth acceleration [73–76]. Midgrowth spurts in facial growth velocity have also been shown in some individuals. [54, 59, 77]

Q: How does "normal" vertical skeletal development occur?

A: "Normal" vertical skeletal relationships arise from a sequential coordination of the major elements of the craniofacial growth process:

- Cranial base growth
- Sutural lowering of the maxilla with drift of the palate/nasal floor
- Growth of the mandibular corpus, ramus, and condyle
- Maxillary and mandibular growth rotation
- Dentoalveolar development

These processes are directed by complex interrelationships between factors under genetic control and environmental (epigenetic) factors influencing this control.

Q: Is "normal" skeletal development a prerequisite for "normal" interocclusal dental relationships?

A: No. A Class I molar relationship is considered "normal," as is 1–2 mm of incisor overjet and 10–20% of incisor overbite. However, "normal" interocclusal dental relationships can occur in spite of abnormal skeletal relationships. For example, it is estimated that 50–55% of the US population have Class I malocclusion [78], meaning that they have Class I molars in spite of possible anteroposterior (AP), vertical, or transverse skeletal problems (in addition to dental problems, such as crowding). Among all human populations, there are individuals who vary significantly from others in vertical, AP, and transverse skeletal relationships yet have similar interocclusal relationships [42].

Q: The preceding answer raises the following question: what is the growth mechanism providing "normal" interocclusal molar relationships despite significant variation in skeletal relationships?

A: Simply put, it is the *compensatory growth potential* of each major element of the facial growth process. In other words, compensatory growth of one element can overcome unbalanced growth of another element, allowing for attainment and maintenance of normal interocclusal relationships [42]. For example, decreased cranial base flexure can be associated with skeletal mandibular retrusion; but, an individual with decreased cranial base flexure and increased ramus breadth growth will maintain a normal interocclusal relationship.

Q: By what one mechanism do skeletal problems develop?

A: This is a trick question. By and large, there is not one *single* causative mechanism for the development of skeletal problems. The development of skeletal problems is multifactorial, and "abnormal" skeletal relationships arise from aberrations in genetic and/or environmental influences. Aberrations produce altered growth intensity and/or altered temporal coordination of one or more major growth elements. Aberrations, sufficient in magnitude and/or duration to disrupt the interrelationships of these elements, result in adaptive growth movements of the maxilla and mandible that may lead to abnormal changes in the spatial relationship of the jaws [42, 79].

Q: With respect to craniofacial development in the vertical dimension, abnormal skeletal relationships are characterized using various cephalometric measures. Can you list, and describe, three of these measures?

A: Three cephalometric measures describing vertical development include the following:

- Ratio of posterior facial height (linear measurement from Sella to Gonion) to anterior facial height (linear measurement from Nasion to Menton) or PFH/AFH, viewed in the sagittal plane. A reduced PFH/AFH ratio is associated with greater divergence between the MP and cranial base, indicating a "skeletal open bite" facial pattern. Conversely, a large PFH/AFH ratio tends to indicate a "skeletal deep bite" facial pattern [80].
- Ratio of lower anterior facial height to total anterior facial height (linear measurement from Menton to ANS superimposed on the linear measurement of Menton to Nasion) or LAFH/TAFH. A reduced LAFH/TAFH ratio indicates a skeletal deep bite facial pattern, and a large ratio is associated with greater facial divergence and a skeletal open bite pattern.
- Angular measurement between the SN line and MP, or SN-MP angle. An elevated SN-MP angle indicates greater facial divergence and skeletal open bite facial pattern. A reduced SN-MP angle indicates a skeletal deep bite facial pattern.

Q: Extreme variations of craniofacial vertical growth appear to result from adaptive jaw displacement and remodeling. Skeletal open bites may result from adaptive jaw displacement and remodeling due to what?

A: Skeletal open bites may result from the following:

- Decreased cranial base flexure leading to superior spatial positioning of the temporomandibular fossae. However, this influence is not well understood and requires more study before it can be used reliably in diagnosis.
- Excessive sutural lowering (vertical growth/descent) of the maxilla
- Diminished vertical growth of the mandibular condyles (posterior-superior condylar growth)
- Backward mandibular (true/internal) rotation
- Excessive vertical posterior dentoalveolar development (excessive posterior tooth eruption)

Q: Conversely, skeletal deep bites may result from adaptive jaw displacement and remodeling due to what?

A: Skeletal deep bites may result from the following:

- Increased cranial base flexure leading to inferior spatial positioning of the temporomandibular fossae. However, this influence is not well understood and requires more study before it can be used reliably in diagnosis
- Deficient sutural lowering (vertical growth/descent) of the maxilla
- Excessive vertical growth of the mandibular condyles
- Forward mandibular (true/internal) rotation
- Deficient vertical posterior dentoalveolar development

Q: According to Schudy [9], how does vertical condylar growth relate to the sum of vertical maxillary descent *plus* maxillary/mandibular molar eruption during normal, hyperdivergent, and hypodivergent facial development?

A: Schudy suggests the following:

- When vertical condylar growth *matches* the sum of maxillary descent and posterior dentoalveolar development, *normal vertical development results.*
- With diminished vertical condylar growth relative to the sum of maxillary descent and posterior dentoalveolar development, facial hyperdivergence, decreased PFH/AFH, increased LAFH/TAFH, increased SN-MP, and a skeletal open bite pattern result.
- With excessive vertical condylar growth relative to the sum of maxillary descent and posterior dentoalveolar development, facial hypodivergence, increased PFH/AFH, decreased LAFH/TAFH, decreased SN-MP, and a skeletal deep bite pattern result.
- It is noteworthy that extreme patterns of vertical skeletal growth develop early and, by and large, remain relatively stable throughout craniofacial growth.

Q: Do we know the *exact* mechanisms by which aberrations in craniofacial growth processes create vertical skeletal abnormalities?

A: No. The exact mechanisms by which aberrations in craniofacial growth processes create vertical skeletal abnormalities remain unknown. Epigenetic (environmental) factors are thought to influence the interrelationship of these processes, but this influence is currently not well-defined. For example, structural and/or environmental conditions that restrict nasal breathing have long been associated with facial hyperdivergence (skeletal open bite). Enlarged adenoids being a conspicuous etiologic agent for restricted breathing, the term "adenoid facies" was widely used to describe the clinical picture of affected individuals. Extreme facial hyperdivergence, narrow maxillary arch, and lip incompetence are prominent clinical features. The theory of epigenetic influence proposed for the development of this skeletal pattern has been largely mechanistic: restricted nasal breathing demands mouth breathing and mouth breathing alters the tongue position, mandibular posture, and head posture. Altered postures lead to muscle imbalance, and muscle imbalance influences craniofacial growth—leading to decreased vertical condylar growth, increased sutural growth of the upper face, and increased posterior dentoalveolar development with negative (elongating) effects on vertical facial growth [81–85]. Although much study has been applied to this theory, a direct cause and effect relationship remains equivocal [86].

More recently, a new theory has been proposed that suggests restricted nasal breathing may be related to abnormal nocturnal secretion of growth hormone (GH) [87]. Children with obstructive sleep apnea share similar craniofacial characteristics to those earlier characterized with "adenoid facies" and also show abnormal nocturnal GH secretion. GH is known to have a positive mediating effect on mandibular ramus height during growth [88]. Further, recent information suggests that faulty GH receptors are a genetic marker for reduced ramus height during growth in certain populations [89–91]. Taken together, this information suggests that the mechanism by which restricted nasal breathing affects craniofacial form is due to a more complex sequence of epigenetic events than envisioned previously. Our point is that, even using one example, the *exact* mechanisms by which aberrations in craniofacial growth processes create vertical skeletal abnormalities are unknown.

Q: Is there a relationship between masticatory function and vertical facial dimensions?

A: Yes, variation in vertical facial dimensions is closely associated with masticatory performance. Studies have documented that an increase in the relative height of the anterior lower facial skeleton is predictably associated with a decrease in bite force magnitude, smaller cross-sectional area of the masticatory adductors (i.e., masseter, medial pterygoid, and temporalis), and reduced masticatory muscle activity. The precise influence of masticatory performance on facial form is not well understood, and there is debate regarding the causal nature of this relationship [92]. In a manner similar to habitual mouth breathing, reduced muscle function may alter the posture of the mandible and thus affect the pattern of mandibular rotation during development resulting in a long-face phenotype [93].

Q: What is meant by growth rotation of the jaws?

A: Viewed in the sagittal plane, the maxilla and mandible undergo patterns of rotation during facial growth and development. Björk [7, 94] identified three components of mandibular rotation and remodeling (matrix, intramatrix, and total rotation, Figure 1.39 illustrates an example) that have been more recently redefined [78]:

- Apparent rotation (“matrix” rotation) is a measure of angular changes of the mandibular inferior border relative to the anterior cranial base (usually measured as changes in SN-MP). It is the absolute change in MP angle during growth.
- Angular remodeling (“intramatrix” rotation) is a measure of change in the MP relative to the mandibular reference line representing the mandibular corpus (determined by mandibular structures stable during growth). It reflects remodeling along the mandibular lower border during growth.
- True rotation (“total” rotation, “internal” rotation) is a measure of change in the mandibular reference line (mandibular corpus) relative to cranial base reference line. True rotation is the sum of apparent rotation and angular remodeling. Apparent rotation is often less than true rotation due to the angular remodeling that minimizes SN-MP change during growth.

Q: How does growth rotation of the jaws influence the vertical dimension of the face?

A: Björk was the first to discover true growth rotation, defined as rotation of the maxillary or mandibular corpus relative to the cranial base, measured from stable references (implanted bone markers) in the sagittal view. [94, 95, 8] His studies demonstrated a relationship between true mandibular growth rotation and condylar growth:

- Greater true forward mandibular growth rotation is associated with vertical condylar growth of larger magnitude directed more anteriorly.

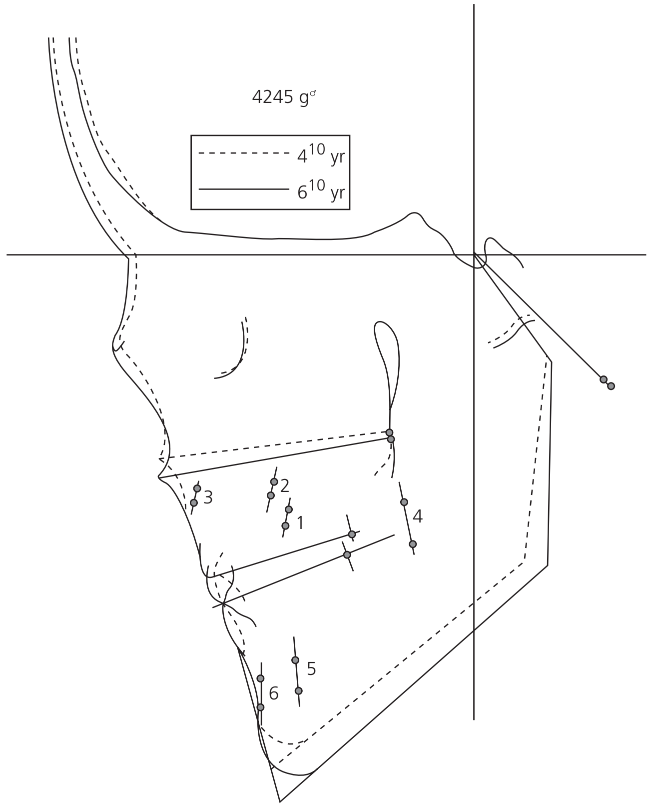


Figure 1.39 Overall superimposition of cephalometric tracings depicting 2 years of growth in a young male. Black dots indicate location of implant markers. Numbers 1, 2, and 3 indicate maxillary implant markers. Numbers 4, 5, and 6 indicate mandibular implant markers. Black lines bisecting dot pairs indicate the direction of implant marker displacement during growth. Note the equivalence in magnitude and direction of maxillary implant displacement indicating nominal true rotation. In contrast, the pattern of changes in magnitude and direction of the mandibular implants indicates true mandibular anterior rotation. The pattern of inferior mandibular border change suggests nominal apparent mandibular rotation due to angular remodeling. (Björk A. Facial growth in man, studied with the aid of metallic implants. Acta Odontol Scand. 1955;13:9–34. Reproduced with permission from Taylor & Francis Ltd.)

- Greater backward mandibular growth rotation is associated with vertical condylar growth of smaller magnitude directed more posteriorly.
- True forward mandibular rotation during growth is also associated with decreases in the gonial angle [96–99], decreases in MP angle [100], and increases in posterior facial height [95]. True forward rotation results in adaptive modeling of the mandible, including increased posterior ramus deposition, increased posterior lower border resorption, and increased anterior lower border deposition [101, 102].
- Extremes of true forward or backward mandibular rotation are associated with skeletal deep bite and skeletal open bite, respectively [80].

Q: What is the occurrence of forward or backward growth rotation of the jaws?

A: Measurement of selected populations longitudinally has suggested that *true mandibular growth rotation is, by and large, forward for most* individuals [14, 15, 103]. High-angle (increased SN-MP angle) subjects show less forward true rotation (degrees per year) and less condylar growth magnitude (mm per year) compared with low-angle (decreased SN-MP angle) subjects. In addition, these studies support the findings of Björk, namely, that apparent forward rotation (decreasing SN-MP) during growth occurs even for hyperdivergent subjects. The take-home message is that the vast majority of individuals with hyperdivergent skeletal patterns experience net forward jaw rotation during growth.

Q: Is rotation of the jaws uniform during craniofacial growth?
A: No. In a manner similar to the velocity of condylar growth, individuals show variation in amount, duration, and direction of growth rotation [14, 98, 100]. This means that some hyperdivergent individuals that experience a net forward rotation may experience backward rotation during some period of growth.

Q: Are there diagnostic indices that foretell an individual's maxillary or mandibular future growth rotation?
A: No. Björk [104] identified seven specific structural features that might develop as a result of remodeling during a particular type of growth rotation. His suggestions for predicting condylar rotation have, however, not been widely used by the specialty because (1) some of the indicators cannot be easily seen on the average cephalogram, (2) the use of the indicators is very time-consuming for the clinician, and (3) there has been no scientific validation of the suggested indicators because of difficulties encountered in study design [105, 106].

Q: Does growth rotation affect dentoalveolar development?
A: Yes. Greater mandibular incisor proclination, greater mandibular molar mesial drift, and relatively greater mandibular molar than incisor eruption have been related to greater true forward rotation [95, 107].

Q: Does dentoalveolar development and tooth eruption *cause,* or only adapt to, skeletal problems in the vertical dimension?
A: Dentoalveolar development and tooth eruption *adapt* to skeletal problems in the vertical dimension. Issacson [108] was the first to report on dentoalveolar development in three groups of subjects—those with short anterior facial height (hypodivergent), those with average anterior facial height, and those with excessive anterior facial height (hyperdivergent). Maxillary posterior alveolar development was found to decrease with decreasing SN-MP angle, with a difference of 5.1 mm of dentoalveolar development between hypodivergent and hyperdivergent individuals. This finding has subsequently been confirmed by others [109, 110]. Björk [95] suggested dentoalveolar development was coupled to vertical facial growth, a suggestion later verified in a longitudinal female sample. In this study, eruption of mandibular teeth followed vertical growth displacement of the mandible. Also, mandibular molar eruption showed greater plasticity than vertical mandibular growth, indicating that molar eruption *adapts* to vertical mandibular growth displacement [111].

Q: What causes abnormalities in vertical dentoalveolar development?
A: Vertical dentoalveolar development abnormalities manifest as supraeruption or infraeruption of teeth. Since vertical dentoalveolar development/tooth eruption is controlled by forces of eruption (exact mechanism unknown) and forces opposing eruption (e.g., functional/parafunctional occlusal loading), any disturbance of these forces may create abnormalities. Such disturbances may include the following:

- Diminished masticatory performance/diminished loading: This has been shown to be associated with increased posterior alveolar development, increased posterior tooth eruption, severe hyperdivergence, and skeletal open bite and may influence tooth eruption and jaw growth [92].
- Altered spatial relationship of the jaws: This can result in teeth left unopposed and absence of forces opposing eruption. For example, in a large Class II discrepancy patient (mandibular hypoplasia, large ANB angle), the mandibular incisors are unopposed by the maxillary incisors. The mandibular incisors can supraerupt creating an exaggerated curve of Spee.
- Tongue interposition, digit-sucking, and lip-biting habits: This can increase the duration/magnitude of forces opposing tooth eruption and result in anterior or posterior open bites.
- Ankylosis or tooth loss: This results in elimination of forces opposing eruption and supraeruption of teeth in the opposing arch.

Q: What is dentofacial orthopedics?
A: Orthopedic change can be defined as alteration in size, shape, growth velocity, or growth rotation of bones. Modifying craniofacial growth with orthodontic forces is termed dentofacial orthopedics.

Q: Which craniofacial elements are potentially alterable by dentofacial orthopedics?
A: Forces of significant magnitude and duration can potentially influence the following:

- Glenoid fossa portions of the cranial base
- Cranial base sutures adjoining the maxilla
- Corpus, dentoalveolar process, and teeth of the maxilla
- Condyle, ramus, dentoalveolar process, and teeth of the mandible

Q: Can you provide examples, or cite studies, of orthopedics influencing these elements?
A: One vivid example is the use of a "Milwaukee brace" to treat patients with scoliosis of the spinal column. Use of this device transmits significant force to the lower border of the mandible—producing changes in shape and growth direction for both the maxilla and mandible [112]. Various animal studies [113–116] have demonstrated that application of relatively high force over an extended period of time can produce measurable changes in the flexure of the cranial base, sutural growth of the maxilla, proliferation of condylar cartilage, shape and position of the glenoid fossae, growth rotation of the mandible, and position of teeth/dentoalveolar bone. Orthodontic treatment, with or without the extraction of permanent teeth, can redirect the drift of teeth and alveolar bone, providing treatment-induced adaptation or compensation of the maxillary and mandibular dentoalveolar processes [117].

Q: Can orthopedic treatment effects demonstrated in animal studies be extrapolated to humans?
A: Animal study findings, generally, cannot be directly extrapolated to humans. For example, differences in force magnitude are frequently found between animal studies and clinical application. Elder and Tuenge [113] used 1400 grams of force to produce measurable change in cranial base flexure in monkeys (small cranium), but typical clinical headgear force ranges from 200 to 400 grams per side and typical clinical maxillary protraction or mandibular chin cup forces range from 300 to 600 grams [118]. Also, differences in force duration are frequently found between animal studies and clinical application. For example, McNamara and Bryan studied the orthopedic effects of a mandibular protrusive appliance worn in monkeys from early

mixed dentition until maturity [119], but clinical application of protrusive appliances in persons is for much shorter periods (e.g., 6- to 9-month wear for a Herbst appliance [120, 121]).

Diagnosis and Treatment Planning Principles

Q: When evaluating a patient for orthodontic treatment, what steps are needed to obtain a comprehensive diagnosis and appropriate treatment plan?

A: We recommend a sequence of 10 steps in order to establish a final treatment plan:

1. Obtain a thorough history and establish the patient's chief complaint (CC).
2. Examine the patient clinically and with appropriate records.
3. Exhaustively list diagnostic findings/problems and make your diagnosis.
4. Define your treatment goals.
5. Focus on solving the *primary* problem in each dimension (plus other major problems).
6. Consider surgical options.
7. Establish unknowns you face—strive to reduce significant unknowns *before* presenting final treatment plan to the patient and before doing anything irreversible.
8. Consider benefits/costs of treating nonextraction.
9. Consider benefits/costs of treating with extraction of anterior teeth or posterior teeth.
10. Weigh benefits/costs of all viable treatment options.

Based upon all of the aforementioned steps, you should choose a recommended treatment plan (what you would do if you were the patient or if the patient was a member of your family).

These 10 steps will be used for all the cases presented in this text. Let's examine these steps in detail, using patients to exemplify how information is obtained and used in this process.

Step 1: Obtain a thorough history and establish the patient's CC.

Q: How do you establish the patient's CC?

A: By asking the direct question when discussing their history, "What is your chief concern?" and following with open-ended questions that elicit the patient's description of their concern and their feelings regarding why they are seeking change. For example, Monica (Figure 1.40) is a 41-year-old woman who presents to you with the CC, "I would like my crossbite corrected." Many patients are not so candid. When a patient first presents to you, it can be helpful to ask them questions that draw out their concerns, such as, "Hello Monica, thank you for

Figure 1.40 Initial records of Monica: (a–c) facial photographs, (d) lateral cephalograph, (e) cephalometric tracing, (f) pantomograph, (g–k) intraoral photographs, (l–q) model photographs.

Figure 1.40 (Continued)

coming to see us. Is there any particular problem with your bite or your smile that has brought you here today?" The way you phrase such a question can make all the difference in helping to elicit their concerns. Many patients can benefit greatly from orthodontic treatment in terms of facial and smile esthetic improvement and self-image improvement but have difficulty communicating this. For example, a patient may present for an initial examination and ask you if they should have treatment. Providing the patient a hand-held mirror during the examination will facilitate a discussion about their concern regarding their bite or smile.

Step 2: Examine the patient clinically and with appropriate records.

Q: What should be included in your clinical examination, and what records are appropriate for diagnosis?

A: Your clinical examination should include the following: a review of the patient's past medical history (PMH) and past dental history (PDH), a temporomandibular joint (TMJ) examination, a complete head and neck examination, a periodontal/mucogingival examination, determination of their upper dental midline (UDML) position with respect to the midline of their face, evaluation of the incisal display and upper lip length in relaxed lip position, and a determination of any AP or lateral slide between centric relation and centric occlusion (CR–CO shift).

Appropriate records include facial and intraoral photographs, models or intraoral digital scans of the teeth and gingiva, and radiographic imaging of the facial bones and teeth. Radiographic imaging may include a cone-beam CT scan (CBCT), or separate lateral cephalometric and panoramic radiographs, and a complete mouth series of dental radiographs as you deem appropriate. Also, identify the patient's general dentist and physician and any dental specialists providing treatment, to allow you to communicate with other doctors involved in the patient's care.

For Monica, you perform a clinical examination and make a lateral cephalometric radiograph, panoramic radiograph, models, and photographs (Figure 1.40). Her PMH, PDH, and TMJs are WRN. You observe that CR=CO (there is no shift). Her periodontal examination is WRN.

Step 3: Exhaustively list diagnostic findings/problems and make your diagnosis.

Q: Do you have any suggestions to help make an exhaustive list of diagnostic findings and problems?

A: Yes, using a table to systematically list pertinent diagnostic data and problems that vary significantly from normal can help. An example is shown in Table 1.5.

Q: Looking at Monica's diagnostic records (Figure 1.40), what are her diagnostic findings and problems? (A blank Diagnostic Findings/Problem List table for you to copy is found in the Appendix.)

A: See Table 1.6

Q: Why is it important to view study models from the lingual aspect?

A: Some patients with mesially rotated maxillary first molars will appear Class II when viewed from the buccal but actually have their maxillary first molar lingual cusps properly seated in the mandibular first molar central fossa (Class I) when viewed from the lingual. As shown, Monica is clearly Class I when viewed from the lingual (Figure 1.41).

Table 1.5 Diagnostic findings and problem list with some common features to examine.

Full face and profile	**Frontal view**
	Facial symmetry
	Vertical facial balance
	Relaxed lip posture
	Buccal corridors
	Upper dental midline
	Incisal display during smile
	Gingival display during smile
	Profile view
	Overall profile curvature (convex, straight, concave)
	Nasal shape
	Nasolabial angle (NLA)
	Labiomental sulcus
	Chin position (retrusive, effective, protrusive)
	Lip–chin–throat angle
	Chin–throat length
Ceph analysis	**Skeletal**
	Maxillary position (anteroposterior (AP) and vertical)
	Mandibular position (AP and vertical)
	Bony chin position and form
	Vertical balance
	Dental
	Maxillary and mandibular incisor angulation
	Maxillary and mandibular incisor AP position
Radiographs	Dentition stage of development
	Root morphology
	Pathology
Intraoral Photos and Models	Angle classification
	Iowa classification
	Dental midlines
	Dental arch symmetry
	Dental arch crowding or spacing
	Absence of teeth
	Maxillary occlusal plane alignment
	Mandibular occlusal plane alignment
	Posterior tooth torque
	Transverse occlusal/skeletal discrepancies
	Gingiva and frena
Other	CR–CO shift, syndromes, systemic disease, impacted teeth, poor oral hygiene, etc.
Diagnosis	Angle classification of malocclusion
	Vertical and/or AP skeletal problems
	Vertical and/or AP dental arch problems

Q: How do you use study models to assess posterior tooth torque?

A: By looking at the models down the arch, from anterior to posterior (Figure 1.42).We see that Monica has slight buccal crown torque of the maxillary second molars and lingual crown torque of the mandibular molars. For Monica's treatment these torques may not be critical, but in many patients such torques will play a role in helping you to decide whether or not to expand the maxilla (providing overjet to later remove the torque).

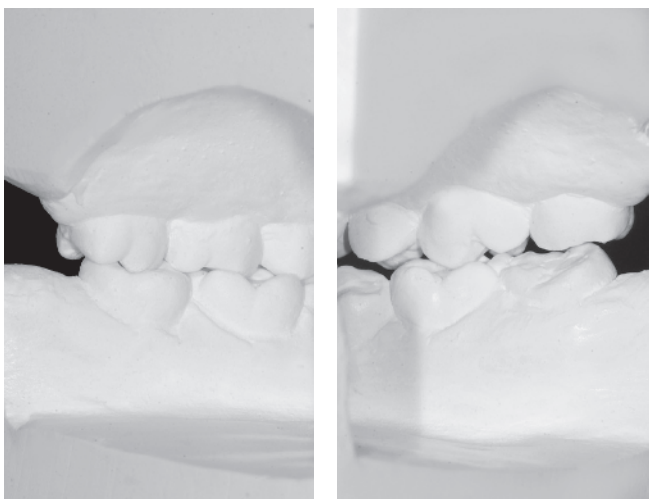
Step 4: Define your treatment goals.

Q: What are "treatment goals"?

A: Treatment goals are the "big picture" objectives you have for a patient and should address the patient's CC and all problems that can feasibly be treated. Some problems, of concern to the patient or found during diagnosis, may not be amenable to treatment or require treatment for which the outcome is not certain. Therefore, compromises involving alternative treatment or no treatment for these problems should be discussed with the patient. Treatment goals are *not* the specific procedures used to achieve big picture objectives. For example, extraction of first premolars is not a treatment goal. It is a treatment procedure.

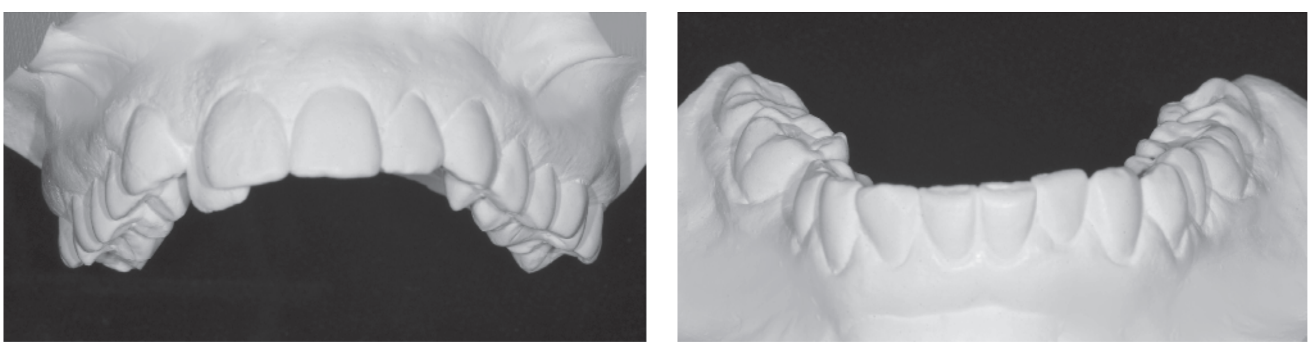
Table 1.6 Diagnostic findings and problem list for Monica (Figure 1.40).

Full face and profile	**Frontal view**
	Face is symmetric
	Vertical facial proportions appear WRN (Glabella–Subnasale ≈ Subnasale–soft tissue Menton)
	Deep paranasal folds
	Absence of an interlabial gap
	Upper dental midline is to the right of her facial midline ≈ 1 mm (UDML R ≈ 1 mm)
	Inadequate incisal display in a posed smile (central incisor gingival margins are hidden above vermillion border of maxillary lip)
	Profile view
	Upturned nose
	Obtuse NLA
	Straight profile
	Prominent chin
	Lip–chin–throat angle WRN
Ceph analysis	**Skeletal**
	Maxillary AP position is mildly deficient (A-Point lies behind Nasion-perpendicular line)
	Mandibular AP position appears WRN
	Effective bony Pogonion
	Vertical skeletal proportions WRN (FMA = 21°; SNMP = 29°; LAFH/TAFH = 57%)
	Dental
	Upright maxillary incisors (U1-SN = 99°)
	Mandibular incisors mildly proclined (FMIA = 62°)
Radiographs	Adult dentition
	Missing third molars
Intraoral photos and models	Angle Class I
	Iowa Classification: I II (2–3) I I
	OJ ≈ 1 mm
	OB ≈ 60%
	Curve of Spee (COS) depth ≈ 2 mm
	Midlines are not coincident (LDML to right of UDML by 1–2 mm)
	Mild mandibular and maxillary crowding
	Increased buccal crown torque maxillary second molars
	Increased lingual crown torque mandibular molars
Other	Anterior crossbite (single tooth)
Diagnosis	Angle Class I malocclusion
	Single tooth anterior crossbite



(a) (b)

Figure 1.41 Lingual view of Monica's occlusion: (a) left, (b) right.



(a) (b)

Figure 1.42 Separated models view of Monica.

Q: What would be appropriate goals for Monica? Do you anticipate any treatment compromises?

A: The most important goal is addressing her CC—correction of her crossbite. Other goals may include achieving a Class I AP relationship bilaterally (molars *and* canines since her right canines are initially Class II), deep bite correction (obtaining minimal overbite), and obtaining minimal OJ. No treatment compromises are anticipated.

Step 5: Focus on solving the *primary* problem in each dimension (plus other major problems).

Q: What is the importance of identifying the *major (primary)* problem in each dimension and other major problems?

A: Major problems having a direct impact on your treatment goals must be identified. This is one point we will emphasize throughout this text. If you learn only one thing from us this is it. You must never lose sight of the major problem you face in each dimension (plus other major problems). Avoid getting sidetracked during treatment on small issues (such as marginal ridge discrepancies between adjacent teeth) when you have larger AP, vertical, and transverse issues to deal with. Correct your major problems first, and then move on to addressing smaller problems. During your initial workup, *and as you examine the patient at each treatment appointment*, you should ask yourself: "What are the primary AP, vertical, and transverse problems in this patient (plus other major problems, such as impacted teeth)?" Then, focus on correcting these major problems.

Q: Why is this concept so important?

A: The following case is an example of why this concept is so important and why you can end up in trouble if you ignore this principle. Look at Corey (Figure 1.43), a 14-year-old boy who presents for treatment with the CC, "I have crowded teeth." His diagnostic findings and problem list are found in Table 1.7.

Q: Given your diagnostic findings and problem list, what are Corey's *primary* problems in the AP, vertical, and transverse dimensions? Are there other significant problems that will affect your treatment plan? (A blank Primary Problems table for you to copy is found in the Appendix.)

A: See Table 1.8

Q: Given your diagnostic findings and problem list, what are your treatment goals for Corey?

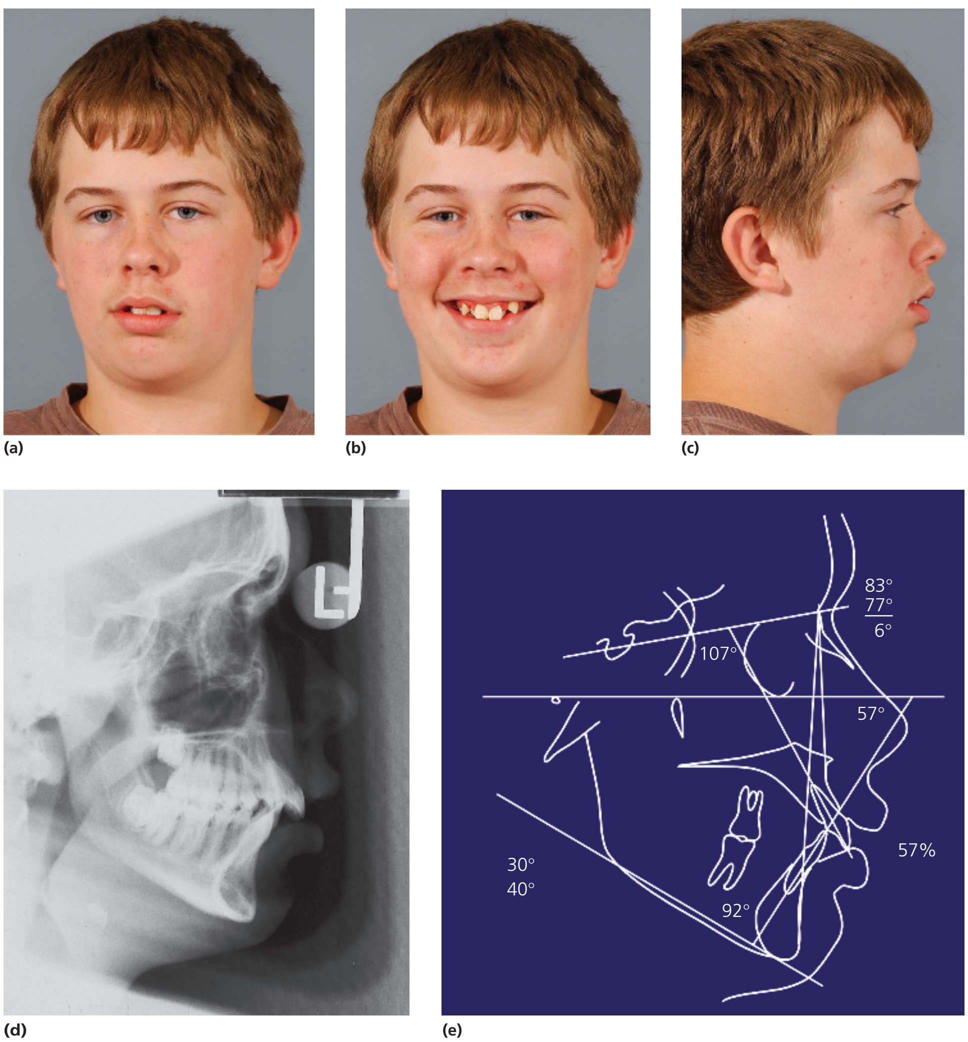


Figure 1.43 Initial records of Corey.


(f)


(g)


(h)


(i)


(j)


(k)


(l)


(m)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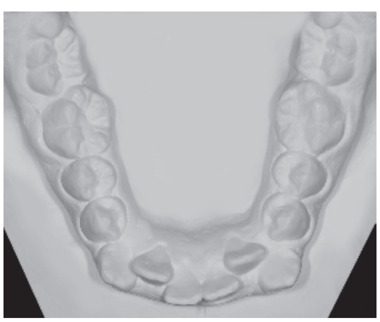

(p)


(q)

Figure 1.43 (Continued)

Table 1.7 Diagnostic findings and problem list for Corey (Figure 1.43).

Full face and profile	**Frontal view**
	Long lower anterior soft tissue height (LLAFH) (soft tissue Glabella–Subnasale < Subnasale–soft tissue Menton)
	Interlabial gap in relaxed lip position (ILG)
	Buccal corridors evident
	UDML to left of facial center
	Excessive incisal display at rest and during smile
	Excessive gingival display during smile
	Profile view
	Upturned nose
	NLA—WRN
	Convex profile
	Deep labiomental sulcus
Ceph analysis	**Skeletal**
	Maxillary AP position WRN
	Retrusive mandible with ineffective Pogonion
	Steep mandibular plane angle
	Short posterior facial height
	Dental
	Maxillary incisor proclination (U1-SN = 107°)
	Mandibular incisor proclination (FMIA = 57°)
Radiographs	Full permanent dentition
Intraoral photos and models	Angle Class II division 1
	Iowa Classification: II (6) II (6) II (6) II (6)
	Moderate maxillary and mandibular anterior crowding
	Mandibular incisors stepped up
	Narrow constricted maxillary arch
	Mandibular posterior lingual crown torque
Other	Poor oral hygiene
Diagnosis	Angle Class II division 1 malocclusion
	Mandibular skeletal retrusion
	Mildly excessive vertical anterior facial height
	Transverse maxillary skeletal deficiency with mandibular posterior transverse compensations

Table 1.8 Primary problems list for Corey *(apical base/skeletal discrepancies italicized).*

AP	II (6) II (6) II (6) II (6) *Mandibular skeletal retrusion*
Vertical	*Mildly excessive vertical anterior facial growth* (ILG, excess incisal display, steep MPA)
Transverse	*Constricted maxillary arch* with significant mandibular posterior lingual crown torque (mandibular posterior transverse dental compensations)
Other	Poor oral hygiene

A: Treatment goals include the following:

- Improve profile/chin projection.
- Obtain Class I canine relationship.
- Obtain normal overbite and overjet.
- Increase maxillary skeletal width/eliminate transverse posterior compensations.
- Relieve maxillary and mandibular dental arch crowding.
- Improve poor oral hygiene.

Q: Keeping his AP, vertical, transverse, and other major problems in mind (Table 1.8), how would you proceed with Corey's treatment?

A: Considering Corey's age, it would be wise to *begin orthopedic treatment immediately* in order to take advantage of any remaining growth. Further, it would be wise to delay orthodontic tooth movement until his major AP, vertical, and transverse problems have been addressed via dentofacial orthopedics. His maxilla should be skeletally expanded with an RME appliance, and he should be placed on a high-pull headgear (HPHG; to restrict his maxillary forward and downward growth) or a Herbst appliance (to restrict his maxillary growth and accelerate his mandibular growth). At the same time, every effort should be made to address his poor hygiene before he has orthodontic appliances placed and begins tooth movement. This would be the smart approach—focusing on his major problems.

However, this was not the approach taken. Yes, his maxilla was expanded with an RME appliance to begin, but he was then placed in fixed appliances to align his arches (Figure 1.44) for nearly a year (with the thought that he would be placed in a headgear or Herbst appliance sometime later).

Beginning with an RME appliance was prudent, but the lack of focus on his AP and vertical problems was not. By delaying headgear or Herbst treatment, any possible orthopedic benefits to improve Corey's AP and vertical problems were lost. Ignoring his primary AP and vertical problems could have necessitated a later need for orthognathic surgery to achieve treatment goals. Further, not immediately addressing his poor hygiene resulted in areas of decalcification (Figures 1.45d–f). In patients with high caries susceptibility, poor oral hygiene can force discontinuing treatment early leading to compromised results.

Q: It was at this time that Corey is transferred to you (Figure 1.44). How would you proceed?

A: Even at this late stage, you need to *focus on the primary problems in each dimension*. Corey still has skeletal problems in two dimensions (deficient mandibular AP growth and excess vertical maxillary growth). Since his skeletal problems are found in the basal bone of the jaws (in the bone apical to the alveolar process bone of the jaws), we can refer to Corey's primary problems as "apical base discrepancies" between the jaws. Apical base discrepancies are treated in three different ways: orthopedics, masking (camouflage), and surgery.

Figure 1.44 Progress records of Corey.

Orthopedics is the modification of apical base discrepancies by modifying jaw growth. Two factors (in addition to patient compliance) determine your chances for success with orthopedics: the magnitude of the apical base discrepancy and the time remaining for growth to occur. If the patient has a mild apical base discrepancy with years of potential growth remaining, then you have a reasonable chance of correcting the problem. If the child has a severe apical base discrepancy with very little future growth potential, then you have a poor chance of correcting the problem (but with some growth you may achieve enough correction to be able to mask the problem).

Masking (camouflage) is orthodontic treatment directed at correcting the dental/occlusal relationships without addressing the underlying apical base discrepancies.

Surgery can correct moderate-to-severe apical base discrepancies when growth is considered complete.

In Corey's case, we lost the opportunity to use orthopedics to correct his Class II relationship (his parents state that he has now finished growing), and his parents are opposed to surgery. We are left with the possibility of masking (camouflage) if we think that a reasonable outcome can result. This is what was done. Corey's maxillary first premolars were extracted, and the extraction spaces were closed by retracting his maxillary canines and incisors distally. His deband photographs are shown in Figure 1.45.

Q: What do you think of Corey's outcome?

A: This was a compromised result. Notice his AP occlusion (Figures 1.45d–f). His canine relationship is slightly Class II

Figure 1.45 Deband records of Corey.

and his posterior teeth do not fit together in maximum intercuspation (he exhibits what is termed "Class II windows"). What could have been an excellent finish is now less than that—because *primary problems were not addressed initially and at every treatment visit.*

Q: Returning to Monica (Figure 1.40, Table 1.6), what are her primary problems in each dimension plus other problems relevant to your treatment plan?

A: Monica does not have overt apical base discrepancies. Her AP, vertical, and transverse skeletal relationships are reasonably normal. But, she does have primary problems in each dimension, as shown in Table 1.9. The correction of these problems should be considered at every appointment.

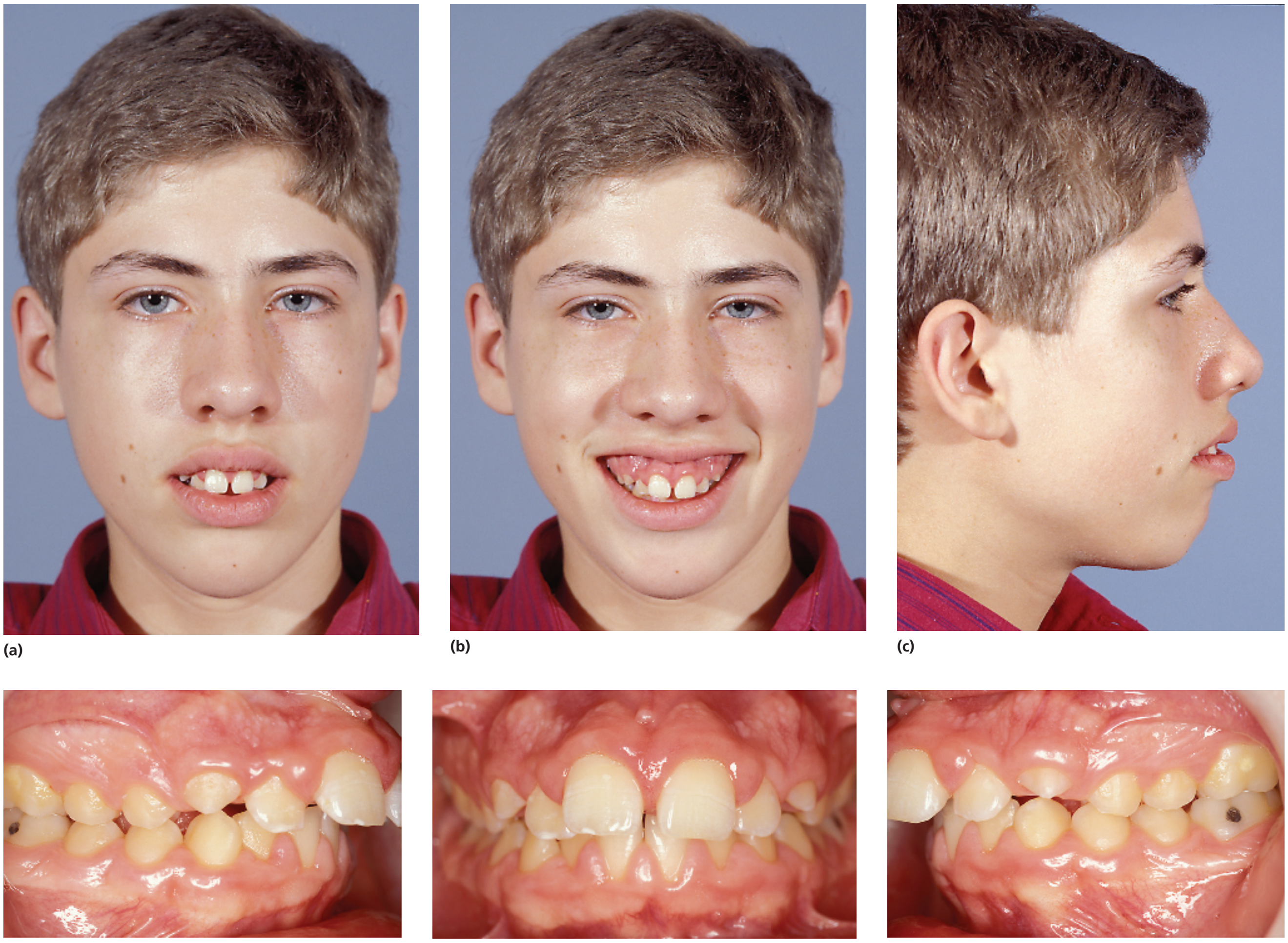
Table 1.9 Primary problems list for Monica.

AP	I II (2–3) I I
Vertical	60% deep bite (dental)
Transverse	–
Other	Anterior crossbite

Step 6: Consider surgical options.

Q: Why must you consider surgical options?

A: Many patients have severe apical base discrepancies and can benefit from orthognathic surgery. But *with every patient, you should always ask yourself whether orthognathic surgery would*



(a) (b) (c) (d) (e) (f)

Figure 1.46 Initial records of Martin.

be a viable treatment option. With most patients you will quickly answer no, with some patients you will quickly answer yes, but with some borderline patients you will want to investigate the patient's concerns and desires further.

For example, look at Martin in Figure 1.46. He is a 19-year-old college student who is not happy with his appearance. It is quite apparent that he exhibits increased anterior facial growth. He shows too much maxillary incisor at rest (excessive incisal display), he shows too much gingiva when smiling (excessive gingival display), and he has a convex profile (inadequate chin projection/mandibular deficiency). After establishing a problem list and diagnosis, you must sensitively discuss his facial features that relate to his vertical and AP skeletal relationships and discuss the anticipated outcomes of orthodontic treatment alone versus treatment that includes jaw surgery.

This was done, and a surgical option was chosen by Martin. Surgery included a Le Fort I maxillary impaction osteotomy to move his maxilla superiorly and bilateral sagittal split mandibular advancement osteotomy to correct his Class II dental relationship and increase his chin projection. Figures 1.47a–h show his final result. Figures 1.47d–e show his initial and post-surgery lateral cephalometric radiographs, respectively. Note the significant improvement in his appearance and occlusion.

Another example is Jackie (Figure 1.48). Jackie is 15 years old and, according to her parents, has finished growing.

Q: What are Jackie's primary problems in the AP, vertical, and transverse dimensions, plus other problems relevant to your treatment plan?

A: See Table 1.10

Q: Jackie's mandibular skeletal deficiency is reflected in her convex profile. Should you discuss this problem with Jackie and her parents? If she would benefit from surgery, then what type of surgery would you recommend?

A: Yes, any time surgery is a viable alternative; you must discuss this option with the patient and parents. What you consider a minor concern, the patient may consider significant. Likewise, what you consider a significant concern may be unimportant to the patient. Jackie and her parents said they were not interested in surgery (mandibular advancement osteotomy), but you must document in her record that this option was presented and declined. Due to the fact that Jackie has finished growing, orthopedics was not an option in Jackie's treatment. The final option presented (masking) included extraction of the maxillary left first premolar with retraction of the maxillary anterior teeth into a Class I left canine relationship.

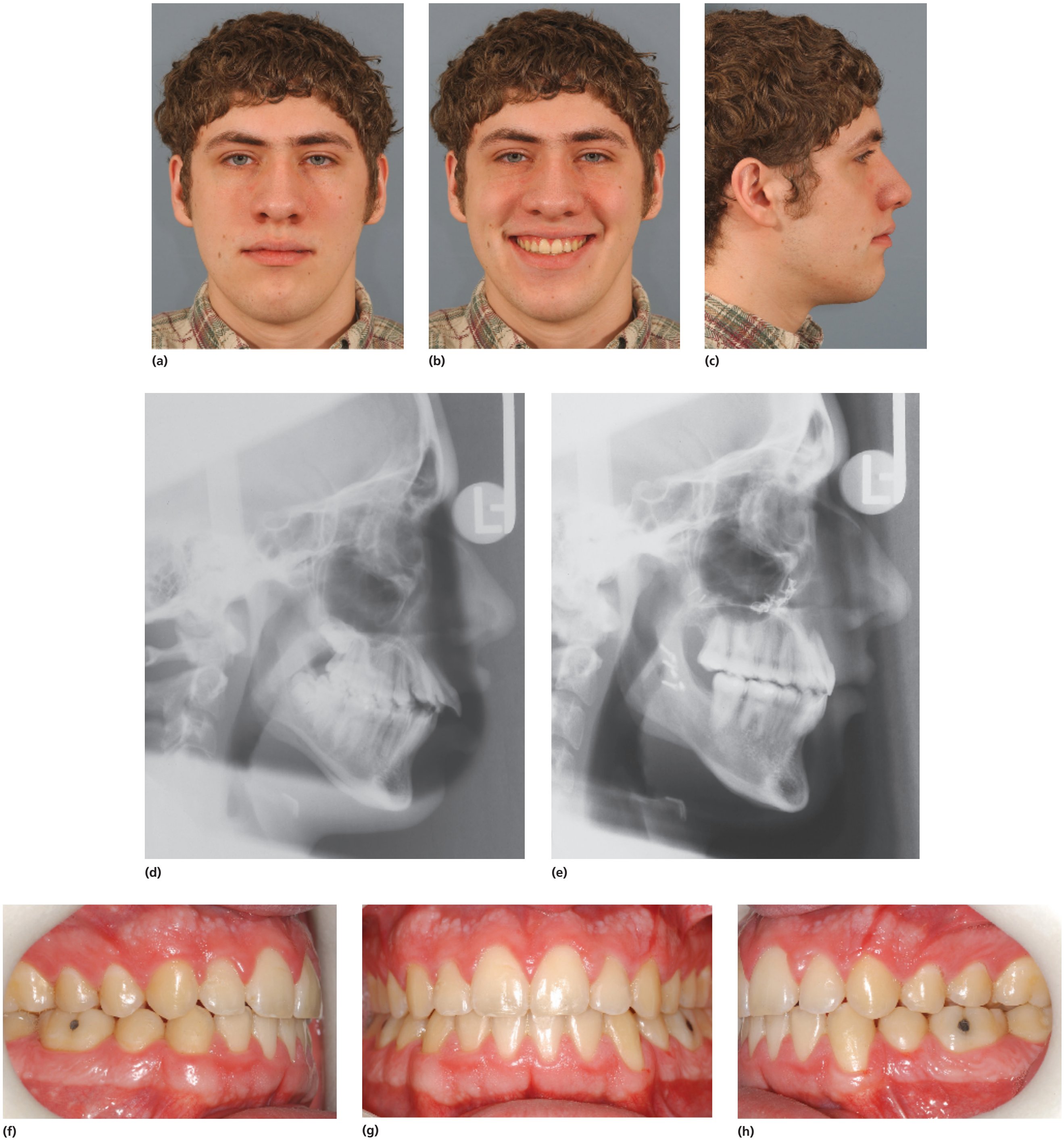


Figure 1.47 Deband records of Martin.

Note: You must discuss with the patient and parents treatment compromises that will occur if surgery is recommended but not chosen, that is, if masking the apical base discrepancy is chosen instead of surgery. If you are attempting to treat adolescent patients with orthopedics, it is usually wise to inform parents of potential future surgical needs if satisfactory growth, or satisfactory patient compliance, is not forthcoming during treatment. Be very considerate of the patient's feelings when bringing up potential surgical needs. In our eyes, every patient we have ever treated has been beautiful even before undergoing orthodontic/surgical care. As an orthodontist, you should inform the patient of structural discrepancies you observe that may limit correction without jaw surgery, but you should also phrase your comments in a thoughtful and sensitive manner.

Q: Returning to Monica (Figure 1.40, Table 1.6), should you discuss possible surgical options with her?

A: No, Monica is not a candidate for orthognathic surgery, and there is no need to discuss surgical options with her.

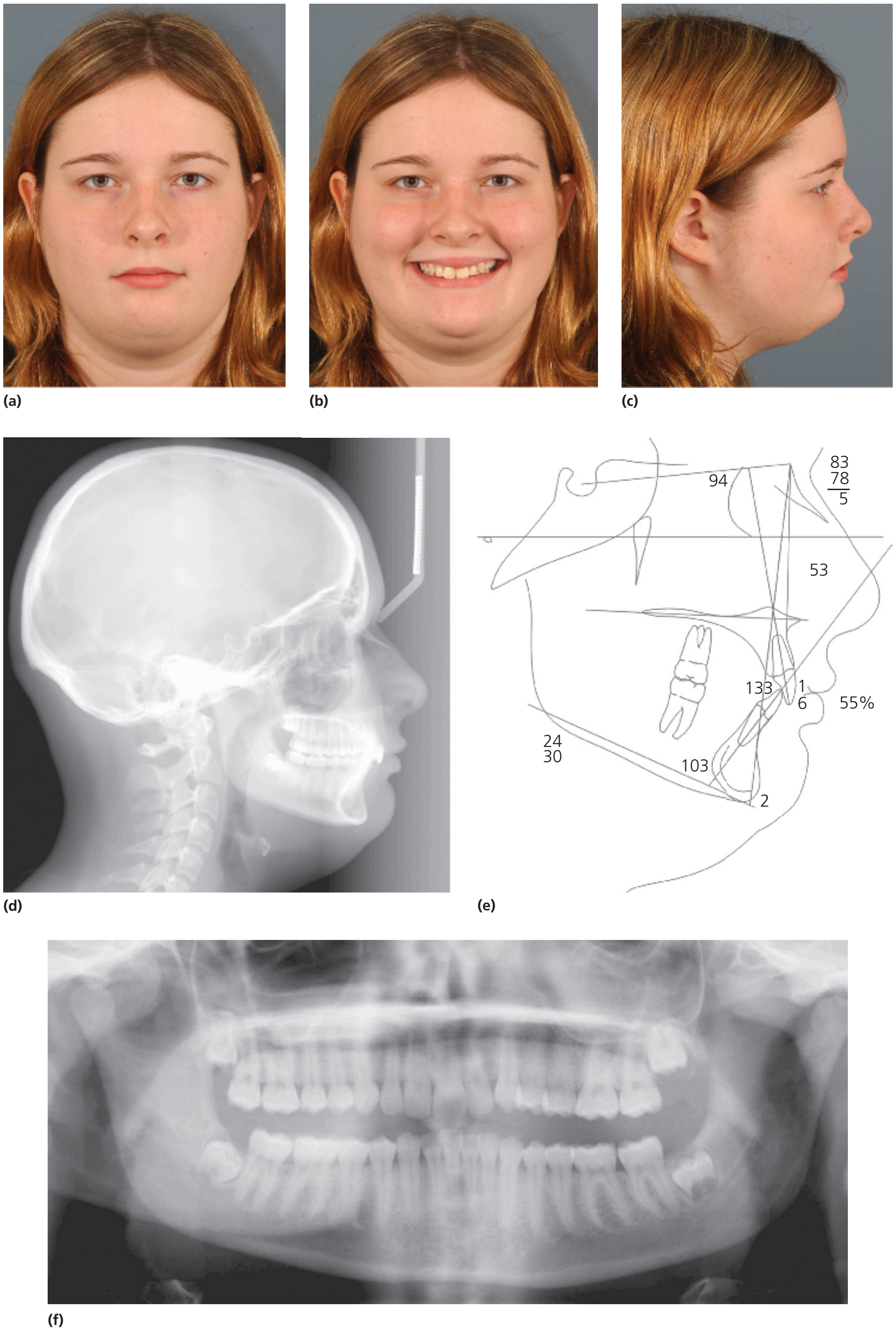
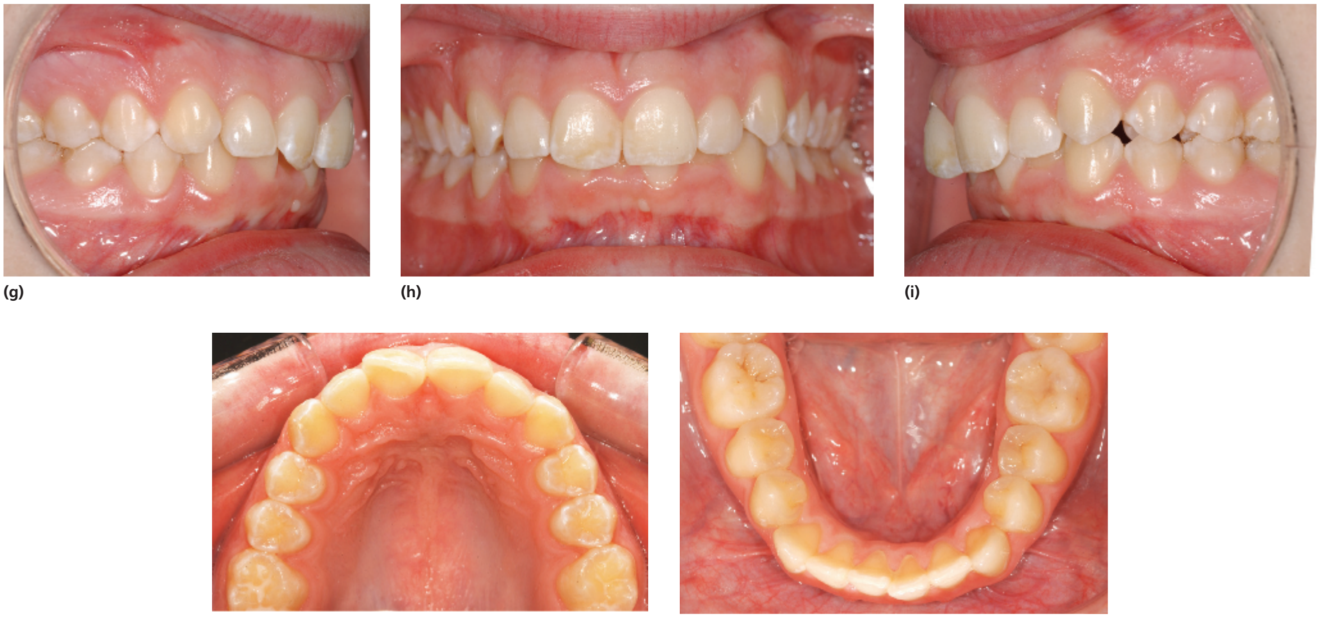


Figure 1.48 Initial records of Jackie.

Step 7: Establish unknowns you face—strive to reduce significant unknowns *before* presenting the final treatment plan to the patient and before doing anything irreversible.

Q: How do unknown conditions affect your treatment plan and the application of treatment?

A: If you only learn one thing from us—this is it: after you have defined the problems you will address with your treatment plan, always force yourself to answer this question, "what unknowns are present in treating this patient and what unknowns should I eliminate before I define my final treatment plan or do something irreversible?" Unknowns typically include variables such as magnitude and direction of jaw growth in adolescent patients, undetected CR–CO shifts, treatment cooperation, whether the midpalatal suture will split in late adolescence, possible tooth ankylosis, patient hygiene, and so forth. Explain to patients and parents the unknowns you face—they will be more understanding if you later need to modify



(g) (h) (i) (j) (k)

Figure 1.48 (Continued)

Table 1.10 Primary problems list for Jackie *(apical base/skeletal discrepancies italicized).*

AP	I I II (3) II (3) *Mandibular skeletal retrusion*
Vertical	70–80% deep bite
Transverse	–
Other	–

your treatment plan as a result of these unknowns. Begin treatment by trying to reduce these unknowns before committing to irreversible treatment (e.g., extracting teeth).

As an example, what unknowns did you face earlier in treatment planning for Corey (Figure 1.43)?Unknowns include the following:

- If you could achieve maxillary midpalatal suture separation with his RME appliance
- How much mandibular (and maxillary) AP growth he had remaining
- How much vertical maxillary growth he had remaining (which might promote rotation of his mandible down and back—worsening his Class II relationship)
- How compliant Corey would be with treatment
- If he would improve his hygiene

Which of Corey's unknowns should have been eliminated before Corey and his parents were given a final treatment plan? Within a 9- to –12-month evaluation period, the unknowns of midpalatal suture opening, jaw growth, patient compliance, and hygiene should have been reduced/eliminated before a final, definitive treatment plan was established. As we discussed, Corey should have immediately been placed in an RME appliance and a HPHG or Herbst appliance while his hygiene was followed. Instead, the principle of reducing unknowns was ignored, resulting in compromised care for Corey.

As another example, Jesse (Figure 1.49) is 16 years, 6 months old. Because of his age, his significant mandibular retrusion (normal maxillary AP position, but ANB = 6°), and his profile concerns, Jesse's treatment plan included extraction of mandibular first premolars, mandibular space closure to upright his proclined mandibular incisors, and bilateral sagittal split osteotomy mandibular advancement. But his parents objected to extracting teeth and requested that a decision to extract teeth be made after Jesse's arches were leveled and aligned.

Q: What is the major unknown in Jesse's case?

A: His remaining facial growth potential. Even though Jesse is 16 years, 6 months old, he could still be growing. In fact, during the time that braces were put on Jesse, and his arches were being leveled and aligned, Jesse's mandible grew enough to where his profile was no longer a concern (Figure 1.50). The need for surgery was eliminated! Our point is this—unless Jesse's parents were absolutely sure that he had stopped growing for some time, orthopedic treatment should have at least been initially considered (maybe for a 6-month trial period while Jesse was being leveled and aligned) before a decision to commit to surgery was made. We would not have lost any time proceeding this way. Thankfully, nothing irreversible (e.g., mandibular premolar extractions) was done until the unknown (growth) was eliminated, even though no one planned it this way.

Q: Was the improvement in Jesse's profile really the result of growth or was it simply a posturing forward of his mandible once his bite was "unlocked" during leveling and aligning?

A: The improvement was due to growth. No CR–CO shift was noted before or after leveling and aligning of Jesse's arches. Interestingly, in a study of Class II division 2 children, proclination of upper incisors and deep bite correction (phase one of the treatment, unlocking the bite) did *not* result in mandibular anterior positioning [122].

Q: Regarding patients like Jesse, what is the current state of the art in predicting the amount and direction of facial growth in adolescents?

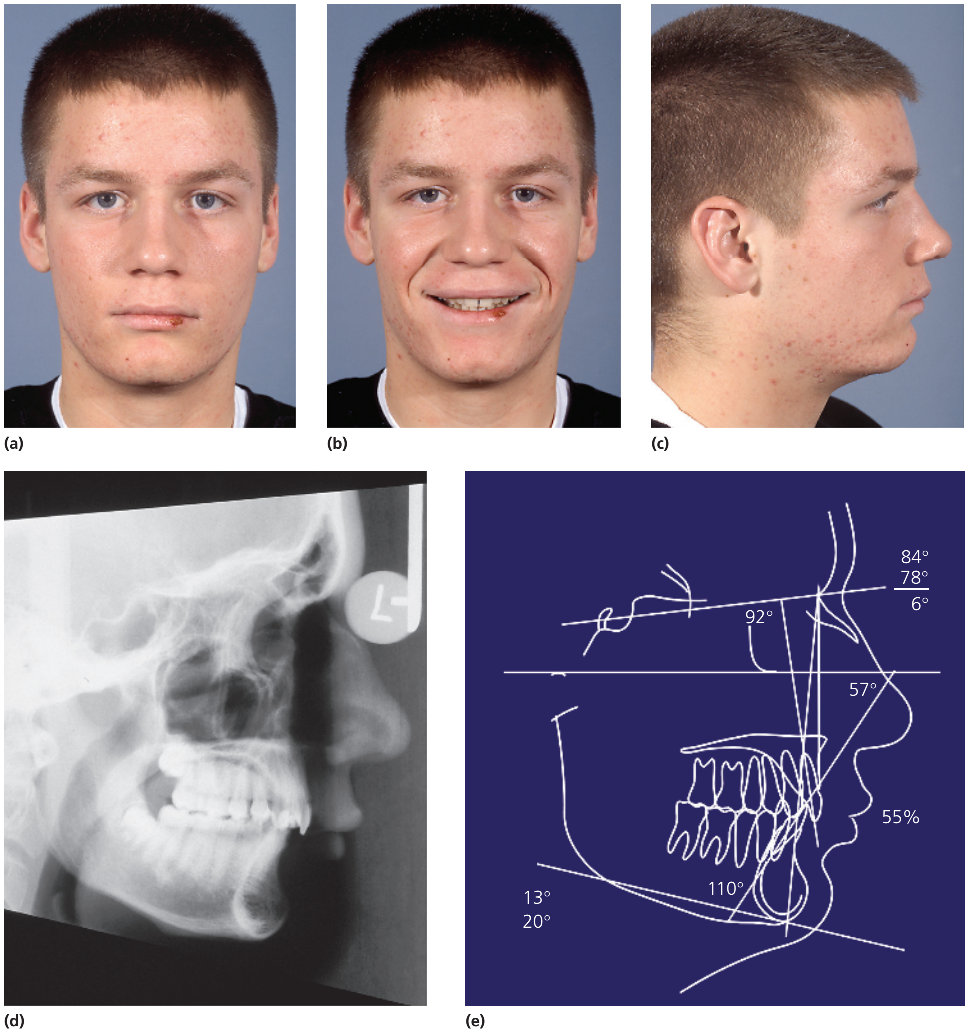


Figure 1.49 Initial records of Jesse.

A: In a recent systematic review [68], a number of salient points were made concerning facial growth prediction, which are as follows:

- Each individual's skeletal maturity is influenced by genetic and environmental factors.
- The same pattern of skeletal growth can be found in almost everyone, but the initiation, duration, and amount of growth varies considerably during the pubertal growth spurt. Some persons mature early with a relatively short pubertal growth spurt, whereas other persons mature late with a large pubertal growth spurt.
- Staging of skeletal development has been assessed using peak growth velocity in standing height, pubertal markers (voice changes in males, menarche in females, breast development, appearance of pubic hair, and appearance of axillary hair), radiographic assessment of bone maturation, and staging of dental development.
- Peak growth velocity in standing height forms a useful historic longitudinal measure of an individual's growth pattern but has little predictive value for future growth rate or percentage of total growth remaining.
- Ossification sequence and timing of skeletal maturity within the hand–wrist area show polymorphism and sexual dimorphism, which can limit their clinical predictive use.
- Skeletal maturity analysis of hand–wrist radiographs for use in predicting facial growth velocity should include bone staging as well as ossification events.
- Overall facial horizontal and vertical growth velocity is related to skeletal maturation indicators determined by analysis of hand–wrist radiographs.
- Maxillary and mandibular growth velocities are related to skeletal maturity, but the correlations are less robust than those for overall facial growth velocity.
- Dental development indicators are not reliable predictors of an individual's stage of skeletal development.
- The relationship between cranial base growth velocity and skeletal maturity has not been definitively established.

In perhaps the most extensive study of the pattern of facial growth and its relationship to maturity indicators [54] yet published, results demonstrated that

- The onset of average pubertal growth spurt in height was *9.3 years in girls and 11.9 years in boys*.
- Average stature (height) peaked *earlier* than facial skeletal growth (*girls 10.9 years, boys 14.0 years*).
- Hand–wrist stages provided the best indication that maturation had advanced to the peak.

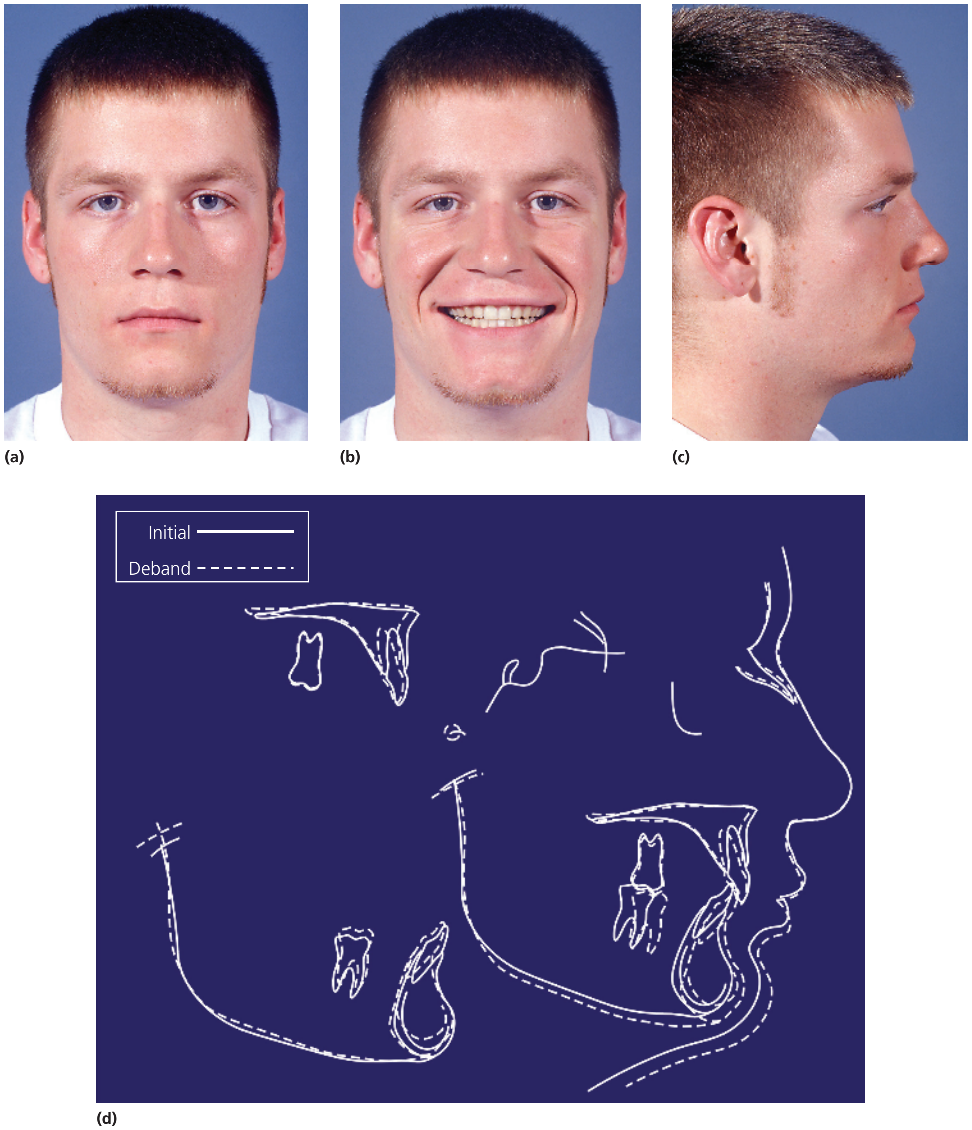


Figure 1.50 Deband records of Jesse. Note the additional growth he had.

- Vertebral stages were consistently the worst predictors (intrinsic lack of predictive ability). Recent evidence suggests that reliability of vertebral staging is questionable [70].
- Of the methods tested for the prediction of peak growth velocity, statural onset had the lowest errors.
- Chronologic age was nearly as good.
- Stature can be measured repeatedly—and may lead to improved prediction of the timing of the adolescent growth spurt.

The point is that the cervical vertebrae method has little clinical predictive value, but hand–wrist measurements do have value as indicators of maturation advancing to peak. Even hand–wrist measurements cannot predict the direction of future facial growth. Chronologic age, but especially repeated measures of stature, can be used clinically (and noninvasively) to improve indication of the adolescent growth spurt.

For girls:

- Peak rate of stature increase is reached prior to menarche [123].
- With the onset of menses, the rate of change in height drops to prepubertal levels and decreases sharply to an adult rate of change during the subsequent 2 years. Note: our clinical impression is that, for girls with hyperplastic mandibular growth, growth may continue far longer than this rule of thumb would suggest.

Q: Why is the prediction of remaining facial growth magnitude and direction so important in adolescents?

A: With a large amount of growth remaining (plus favorable growth direction plus excellent patient compliance), you have a better chance of orthopedically improving an apical base discrepancy (AP, vertical, or transverse) than with little growth remaining. Conversely, with a large amount of growth remaining following orthopedic treatment, it is more difficult to maintain a corrected occlusion afterwards (growth is not been normalized with orthopedic treatment).

Q: List “take-home pearls” in predicting the timing, amount, and direction of adolescent facial growth.

A: Take-home pearls include the following:

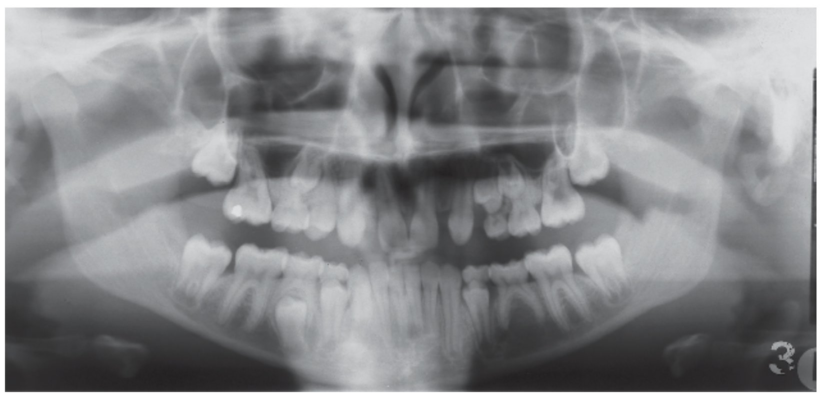
- We would like to predict the adolescent growth spurt so as to time orthopedic treatment and “bracket” or capture this growth period. Further, we would like to be able to predict the amount of remaining growth so that we can best judge when it is safe to discontinue orthopedic treatment (e.g., highpull chincup treatment in Class III patients) or when it is safe for a patient to undergo surgical correction—without fear of relapse from continued growth.

- The state of the art in predicting individual amount and direction of facial growth in adolescents is imprecise. For over 40 years, various radiographic schemes have purported the ability to predict *individual* facial growth. None have survived the test of time, and we are left with "rules of thumb" to guide us during treatment, such as the following:
  - For maximum orthopedic effect, apply an RPHG to an anteroposteriorly deficient maxilla at the time the maxillary incisors are erupting.
  - Wait to start Class II correction until the late mixed dentition stage of development.
  - Once a highpull chincup has corrected a Class III malocclusion, it should be worn until growth is complete.
  - To maximize transverse skeletal correction with RME appliance, apply it before or during puberty.
  - Surgery to correct excess jaw growth should be performed after growth is complete.
  - Surgery to correct a deficient mandible can be instituted earlier.
- Average onset of the statural growth spurt is a little more than 9 years for girls and a little less than 12 years for boys; peak of statural growth spurt is about 1½ to 2 years later; facial skeletal growth peaks later; but, there is considerable variation in these times among patients.
- At an adolescent's initial examination, ask the parents if they are measuring their child's height regularly and if the child has entered his or her growth spurt. Begin making height measurements. For girls, inquire about menarche—once menarche is reached you have about 2 years to capture any remaining growth. *As soon as you or the parents detect the growth spurt onset, consider beginning orthopedic Class II (headgear or functional appliance) or Class III (high-pull chin-cup) treatment*—regardless of whether the patient is still in early mixed dentition or not. It is generally better to error by beginning orthopedic treatment too early than by beginning too late.
- *For patients in late adolescence, if there is any chance that the patient is still growing, you must take this residual growth potential into consideration when treatment planning.* Otherwise, you may miss the chance either to improve the apical base discrepancy orthopedically or to maintain a correction, posttreatment.

Now back to our principle of establishing unknowns. As another example, look at the initial panoramic image and periapical radiographs of an 11-year-old girl shown in Figure 1.51.

Q: What do you see?

A: She is in the late mixed dentition stage of dental development, she is missing her maxillary lateral incisors and mandibular left second premolar, and the roots of her maxillary second premolars are very poorly developed.



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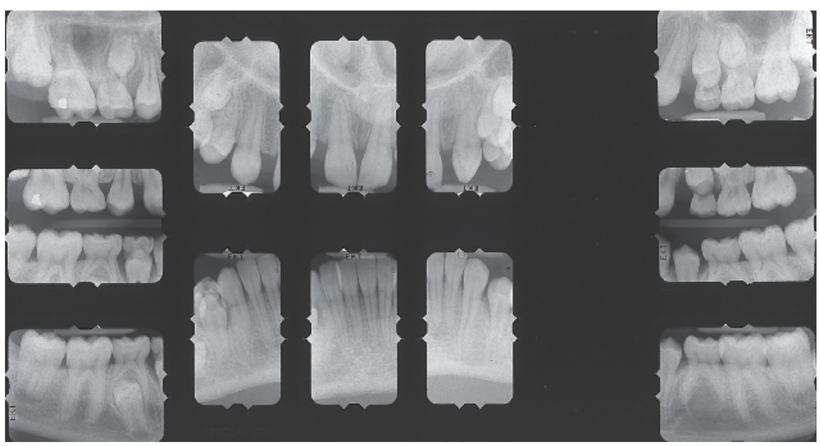


(b)

Figure 1.51 Panoramic and complete mouth series radiographs for an 11-year-old female presenting for orthodontic evaluation.

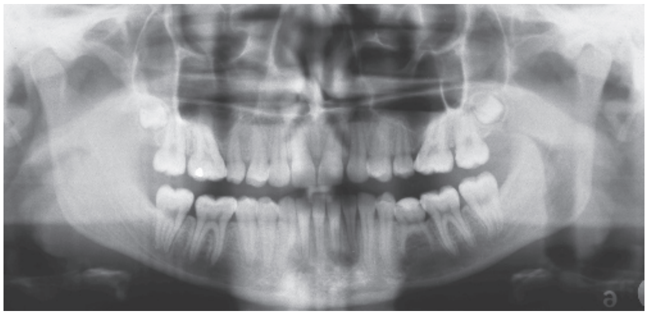


Figure 1.52 Posttreatment panoramic radiograph of the patient presented in Figure 1.51.

Q: So, in terms of these radiographs what are her unknowns?

A: Her unknowns include potential future ankylosis or loss of the retained mandibular left second primary molar and whether her maxillary second premolars will ever develop/erupt (whether they should be retained or extracted).

Q: How should you handle these unknowns?

A: *Wait and monitor*. Give the premolars some time to develop. Assuming there are no outstanding issues with facial growth that must be addressed, you should wait to begin treatment. Waiting is a very hard thing to do for most orthodontists, but waiting can potentially reduce this patient's unknowns to the point where you can finalize your treatment plan. This was done and the maxillary second premolars eventually developed and erupted and were aligned with orthodontic treatment (Figure 1.52). Given adequate time, if they had not developed complete roots, you could have considered extracting them. Note that the mandibular primary left second molar does appear to have ankylosed (slight step in the alveolar process bone between it and the adjacent teeth), and it has been covered with a composite buildup.

Q: Returning to our patient Monica, what are her unknowns?

A: Monica is an adult, so future growth is not a variable. However, her unknowns include the possibility of an undetected CR–CO AP or lateral shift. You must take the possibility of this shift into account and, if present, try to eliminate it before committing to a final treatment plan. In some patients, an undetected shift will not be discovered until habitual biting patterns are "broken up" during leveling and aligning with fixed appliances. Check for a CR–CO shift again at Monica's case presentation appointment and at every treatment appointment thereafter.

Step 8: Consider benefits/costs of treating nonextraction.

Step 9: Consider benefits/costs of treating with extraction of anterior teeth or posterior teeth.

Decisions to extract permanent teeth or not to extract permanent teeth depend on a myriad of factors, including magnitude of the AP apical base or dental discrepancy, amount and location of dental crowding, degree of proclination or uprightness of the anterior teeth, presence or absence of dental arch asymmetries, condition of the periodontium, condition of the teeth, frontal/profile soft tissue esthetics, and the decision to treat surgically or nonsurgically. A decision to extract teeth should never be taken lightly and should be made *only* after significant unknowns are eliminated and after considering the costs/benefits of treating the patient nonextraction, with extraction of different combinations of anterior teeth, with extractions of different combinations of anterior and posterior teeth, and with extraction of different combinations of posterior teeth.

Step 10: Weigh benefits/costs of all viable treatment options and establish the recommended plan and treatment objectives.

This is the time for "putting it all together." Consider every viable treatment option, exhaustively. If necessary, determine what preliminary treatment you must initiate in order to eliminate unknowns before you can decide on a final treatment plan. Many patients generally appreciate that you present a range of options to them at the case presentation, and they realize that you have taken the time to consider the costs/benefits of each. Be prepared to recommend which you consider to be the best treatment option.

The following two cases exemplify application of Steps 8, 9, and 10. Connie is a 33-year-old woman (Figure 1.53) who presents to you for treatment to eliminate her severe dental crowding. She is Class I with a deep bite and relatively straight profile.

Q: Should Connie be treated nonextraction, with extraction of anterior teeth only, with extraction of some combination of anterior and posterior teeth, or with extraction of some combination of posterior teeth only?

A: On the one hand, she has moderate-to-severe mandibular anterior crowding. As a general guideline, tooth extraction to alleviate anterior crowding is generally warranted when the amount of dental crowding is 10 mm or greater. Connie appears to meet this guideline. On the other hand, Connie's cephalometric tracing reveals that she has *very* upright incisors, and her lips are not protrusive. Moreover, she has a Class I dental relationship and a straight profile. These facts support a nonextraction treatment approach since space for her anterior teeth can be gained by proclining her incisors to a more normal inclination.

Proclination of anterior teeth to gain arch length is generally reasonable when the anterior teeth are initially upright. Look at Figure 1.54, top, where the mandibular incisors are upright. Proclining the incisors moves the crowns forward increasing arch length (Figure 1.54, bottom) and places the incisor long axes in a more ideal angulation. Of course, this assumes that the mandibular anterior periodontium can support forward root movement. In Connie's case (Figures 1.53g–i), there appears to be robust periodontium.

All things considered, Connie could benefit from nonextraction treatment, and there are no real costs to treating her nonextraction. But you should never get in the habit of considering only one treatment option. You should always force yourself to exhaustively consider all of your treatment options—including extraction of anterior and posterior teeth.

Q: So, exploring the cost/benefit of extractions, should Connie be treated with extractions?

A: Start by considering extraction of her anterior teeth. The only reasonable anterior tooth to extract would be a mandibular incisor. Extracting a mandibular incisor would help alleviate the anterior crowding and would not upright her anterior teeth more because the extraction space would be used entirely for dental alignment. However, because her maxillary anterior teeth are not small, extraction of a mandibular incisor would result in a mismatch in cumulative mesiodistal widths between maxillary anterior teeth and mandibular anterior teeth (anterior Bolton discrepancy). This mismatch would result in excess anterior overjet at the end of treatment.

Q: How about extracting posterior teeth?

A: Posterior extractions would alleviate her crowding, but you would need to extract symmetrically to keep her dental arches symmetric following space closure (since her arches are already symmetric). Extracting four first premolars would create over 14 mm of extraction space in each arch. Retracting anterior teeth through so much extraction space would result in further uprighting of her already upright incisors. The resulting incisor movement could retract the lips relative to her nose and chin causing a negative impact on her profile (a possible "dished in" profile).

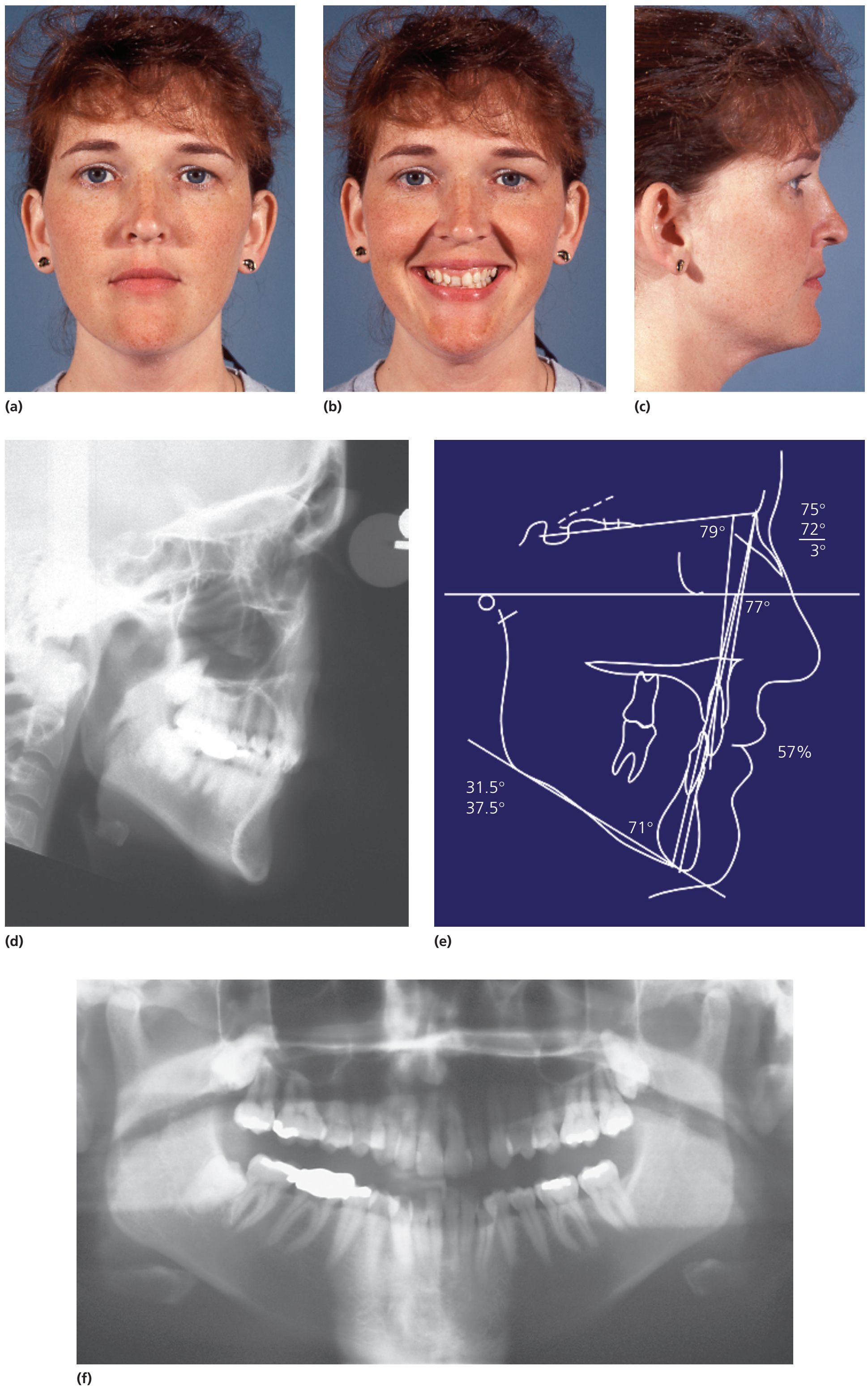


Figure 1.53 Initial records of Connie.

(g) (h) (i)

(j) (k)

Figure 1.53 (Continued)

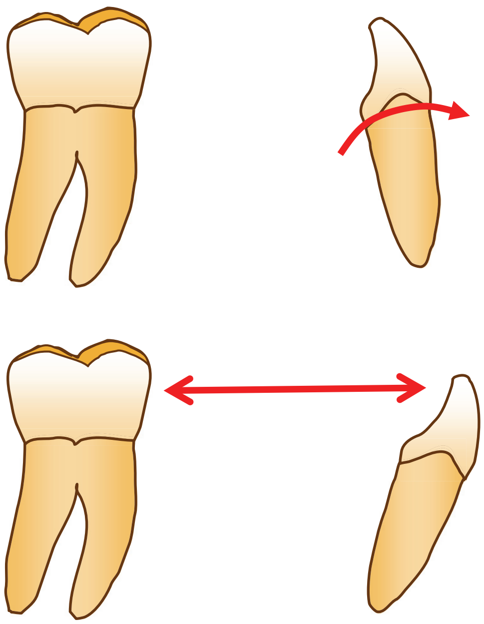


Figure 1.54 By proclining initially upright mandibular incisors (top) additional arch length can be gained (bottom).

Q: In cases where you are retracting maxillary incisors, what *ultimately* limits how far you can retract the maxillary incisors?

A: The position of the mandibular incisors limits the amount of maxillary incisor retraction (i.e. zero overjet). You cannot retract maxillary incisors beyond the mandibular incisors' edges, as you will put the patient in cross bite.

Connie was successfully treated nonextraction by aligning/proclining her incisors (Figure 1.55). The point we are trying to make is that these options must be considered in deciding whether to treat a patient nonextraction or with extractions. With every patient, you must weigh the benefits and costs of nonextraction treatment and different combinations of extraction treatment.

Pearl: With some borderline extraction patients, where you are having difficulty deciding whether to extract or not extract teeth, it makes sense to begin treatment nonextraction and reevaluate for extractions after leveling and aligning the arches. This is another example of reducing unknowns. However, in patients with extreme crowding, proclined incisors, lip protrusion, and steep MP angles, it may make sense to extract teeth (make space) at the very beginning of treatment.

Q: Your next patient is Phoebe (Figure 1.56) who is 11 years old. What are her primary problems in the AP, vertical, and transverse dimensions plus other problems relevant to your treatment plan?

A: See Table 1.11

Q: In terms of your decision to treat Phoebe nonextraction or with extraction of permanent teeth, can you list the factors that will play a role in your decision?

A: Factors to consider include Phoebe's

- Upturned nose (her nasolabial angle could become obtuse if maxillary incisors are retracted).
- Minimal overbite (which will tend to deepen if anterior teeth are retracted).
- AP relationship is normal (there is no need to extract teeth to achieve a Class I canine relationship).
- Moderate-to-severe crowding in the maxillary anterior (blocked-out maxillary permanent canines) and mild-to-moderate crowding in the mandibular anterior (mandibular primary second molars are still present).
- Bimaxillary dental protrusion with mild lip protrusion (both of which could improve if anterior teeth were retracted).

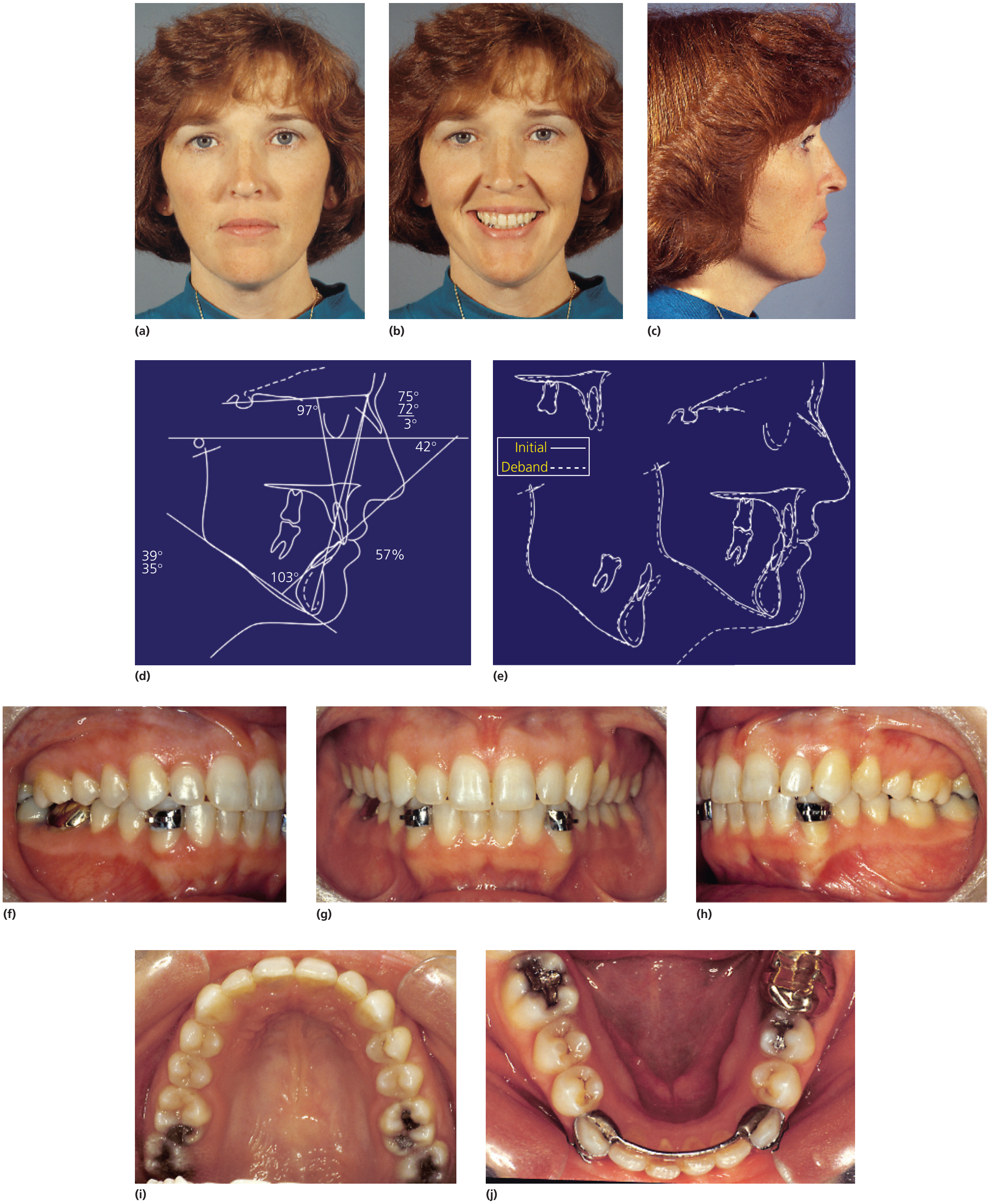


Figure 1.55 Deband records of Connie.

- Thin attached tissue covering roots of mandibular central incisors (will recession occur if the incisors are proclined?).

Q: Is Phoebe a good candidate for nonextraction treatment?

A: Although you can argue that every patient can be treated nonextraction, the cost of treating Phoebe without extractions could be high. Even if you expanded her maxillary arch to gain maxillary arch perimeter and make space for her canines, if she is treated nonextraction then her maxillary incisors could end up very proclined after they are aligned. Her lips could be very protrusive, and she could end up with mandibular anterior gingival recession. However, you could make a case for initially treating her nonextraction, placing a lower lingual holding arch (LLHA), expanding the maxillary arch, and reevaluating for extractions after all the permanent teeth erupt and you observe how much crowding you still have (reducing unknowns).

Q: Is Phoebe a good candidate for extracting anterior teeth?

A: Consider the following:

- If you initially placed an LLHA, allowed all her permanent teeth to erupt, then deemed mandibular tooth extraction still necessary, you could consider extracting a mandibular incisor to alleviate crowding. But, depending upon the cumulative mesiodistal widths of the remaining maxillary and mandibular anterior teeth, you could end up with excess anterior overjet (anterior Bolton discrepancy).
- In the maxilla, you could consider extracting the permanent canines (substituting the maxillary first premolars as canines) to alleviate crowding. If you did this, you would first need to confirm (CBCT scan) that the left lateral incisor root has not been resorbed by the canine crown.
- Or, you could consider extracting maxillary lateral incisors (substituting maxillary permanent canines as laterals and

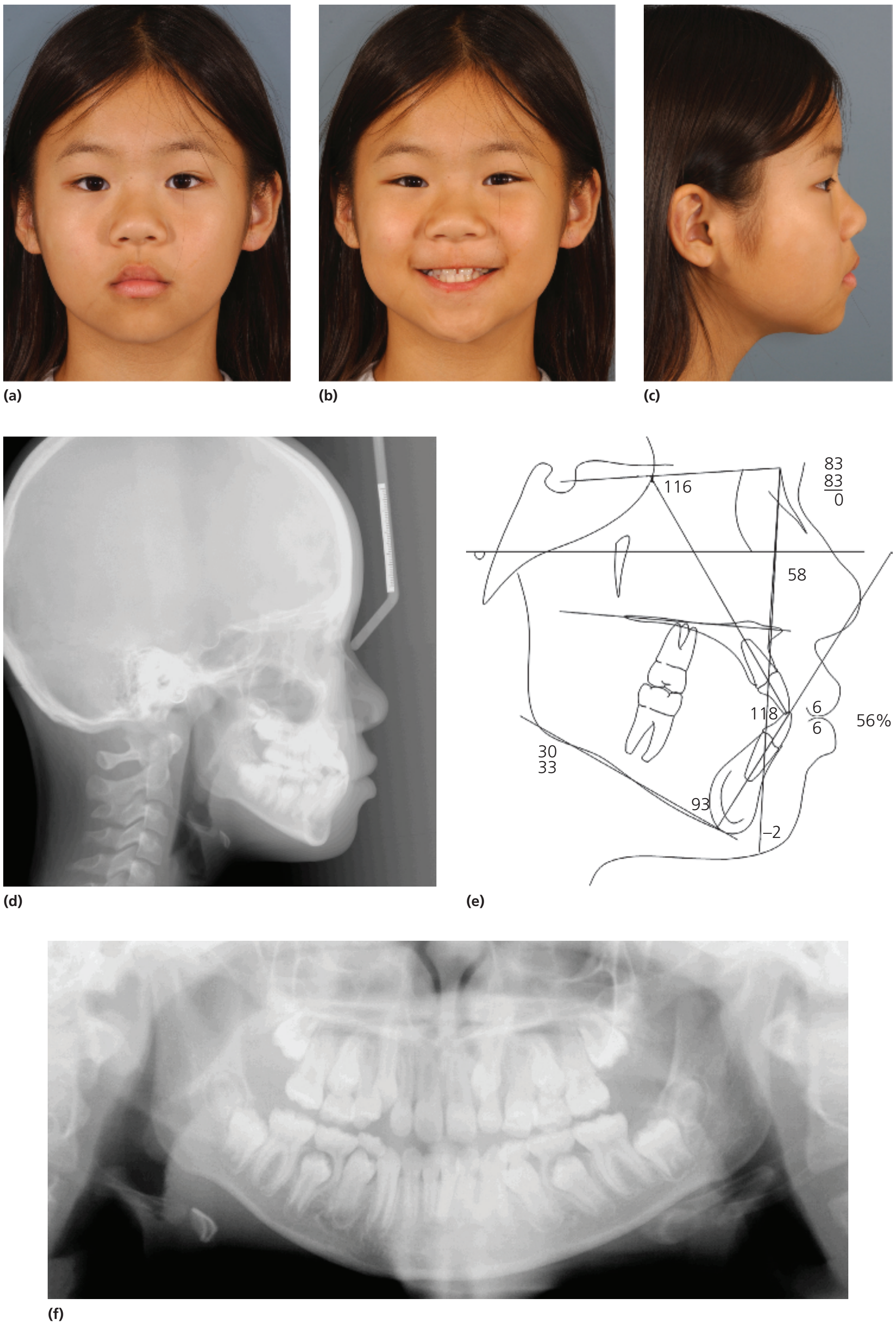


Figure 1.56 Initial records of Phoebe.

Figure 1.56 (Continued)

Table 1.11 Primary problems list for Phoebe (Figure 1.56).

AP	I I I I
Vertical	Minimal overbite
Transverse	–
Other	Anterior crossbite Blocked-out maxillary canines with the left canine overlapping the lateral incisor on panoramic image

substituting maxillary first premolars as canines). If you then ended up treating the mandibular arch nonextraction, you would need to protract the maxillary posterior teeth to place the maxillary first premolars in a Class I canine position.

Q: Patients with severe anterior crowding (incisor protrusion/lip protrusion) may be candidates for first premolar extractions, patients with moderate crowding (relatively acceptable profile) may be candidates for second premolar extractions [124], and patients with severe mandibular incisor crowding (with a corresponding anterior tooth–size discrepancy/peg-shaped maxillary lateral incisors) may be candidates for mandibular incisor extraction. Focusing on just the mandibular arch, what tooth movements are observed with reciprocal space closure (no use of TADs for anchorage) following second premolar extractions, first premolar extractions, and incisor extractions?

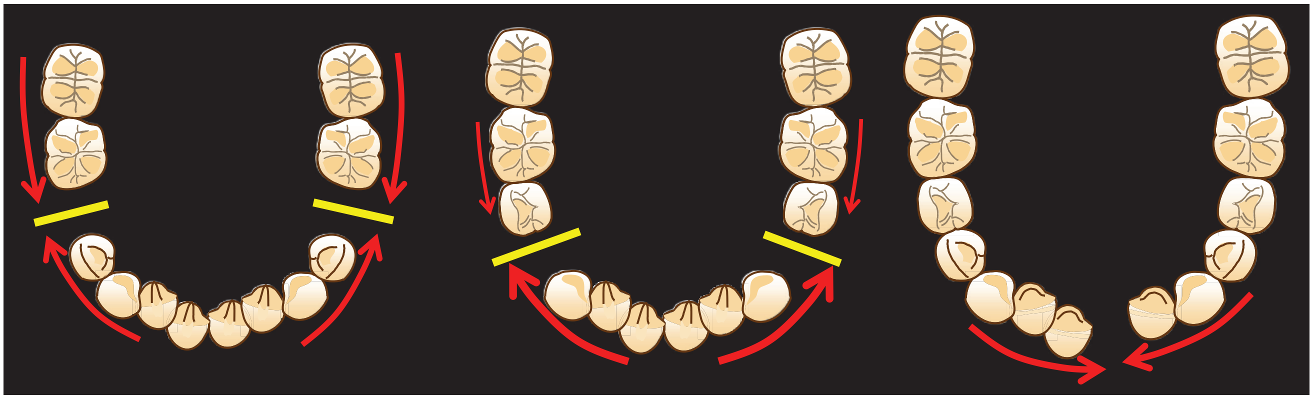
A: As a rule of thumb, when second premolars are extracted one can expect the posterior teeth to move mesially one-half the extraction space, leaving the remaining half for canine retraction, relief of crowding, and incisor retraction (Figure 1.57a). When first premolars are extracted, one can expect the posterior teeth to move mesially one-third of the space, leaving the remaining two-thirds of the space for canine retraction, relief of crowding, and incisor retraction (Figure 1.57b) [125, 126]. When a mandibular incisor is extracted (Figure 1.57c), the entire space is available for relief of crowding. *With incisor extraction, mesial movement of posterior teeth should not be anticipated.* Of course, when TADs are incorporated for anchorage, the entire tooth movement equation changes with the possibility of complete space closure in one direction.

Q: Is Phoebe a good candidate for extracting posterior teeth?

A: Extraction of all four first premolars would provide space to align (and upright) anterior teeth. However, before you extract all first premolars, you should examine the maxillary lateral incisor roots (CBCT scan) to confirm that the maxillary left lateral incisor root has not been severely resorbed by the permanent canine crown. Extraction of premolars may also necessitate surgical exposure of the maxillary left canine. Extraction of all four second premolars offers no advantage over extracting all four first premolars: space closure will result in less incisor retraction and more posterior tooth mesial movement.

Q: Is Phoebe a good candidate for extracting a combination of anterior and posterior teeth? What is your recommended treatment for Phoebe?

A: She was treated with extraction of a combination of anterior and posterior teeth. An LLHA was initially placed, but mandibular crowding was later deemed too severe to treat the lower arch nonextraction. Mandibular first premolars were removed. Due to the position of the maxillary left canine crown (and probable maxillary left lateral incisor root resorption), the maxillary lateral incisors were also extracted. All permanent teeth were allowed to erupt. Figure 1.58 illustrates Phoebe's final result after completion of fixed orthodontic treatment and canine composite veneers. As shown, she has a beautiful smile and profile. A small space exists distal to the mandibular left canine.



(a) (b) (c)

Figure 1.57 Illustration of the effect of (a) second premolar, (b) first premolar, or (c) incisor extraction on movement of teeth (red arrows) adjacent to mandibular extraction sites using conventional orthodontic mechanics without the aid of supplemental anchorage. Yellow lines in (a) and (b) indicate approximate location where translation of tooth segments will stop when extraction spaces are completely closed, reciprocally. Note in (c), protraction of posterior teeth is not seen.

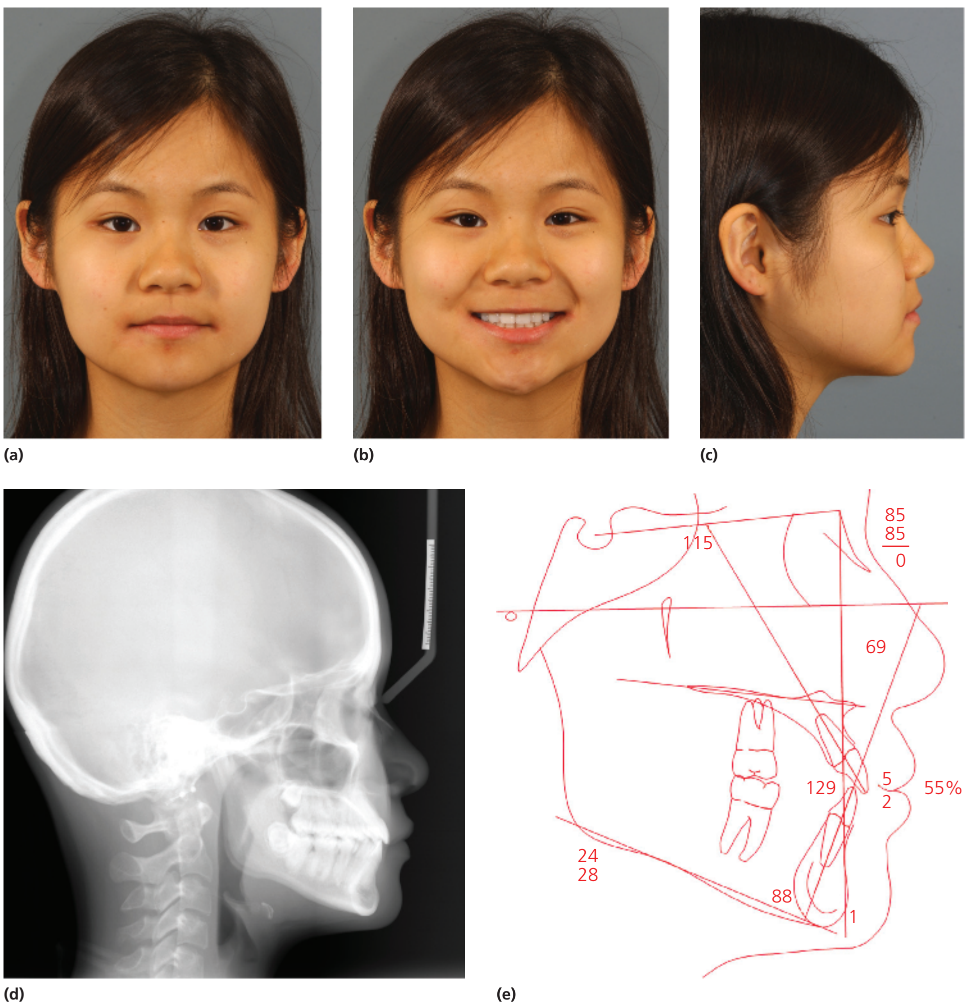
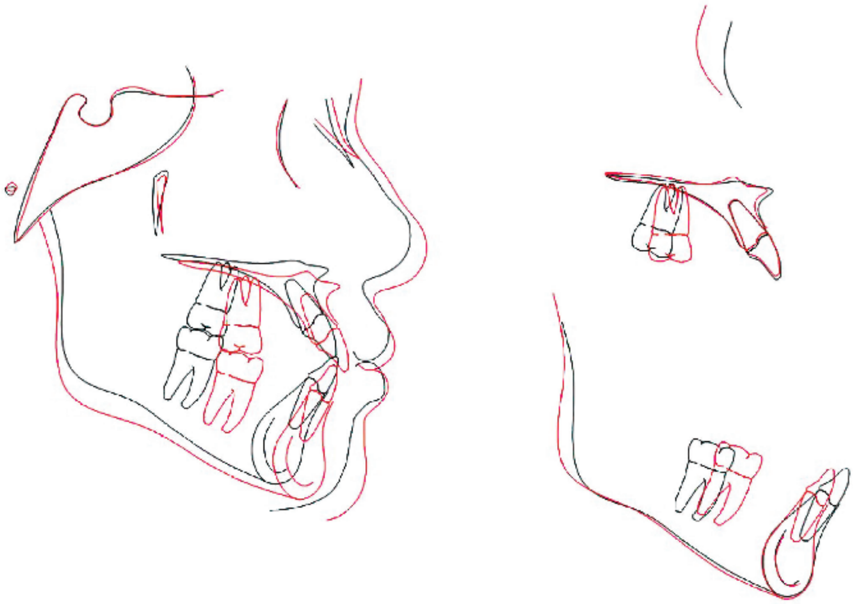


Figure 1.58 Deband records of Phoebe.

Once again, our point is to encourage you to consider all possible options in deciding whether to treat a patient nonextraction or with extractions. You must eliminate unknowns (e.g., placing an LLHA and evaluating space needs after the permanent teeth erupt), consider the costs/benefits of nonextraction treatment, and weigh the costs/benefits of every reasonable combination of anterior and posterior tooth extraction pattern.

Now let's look at a case by proceeding through all 10 steps of diagnosis and treatment planning. Lauris (Figure 1.59) presents to you for orthodontic treatment.



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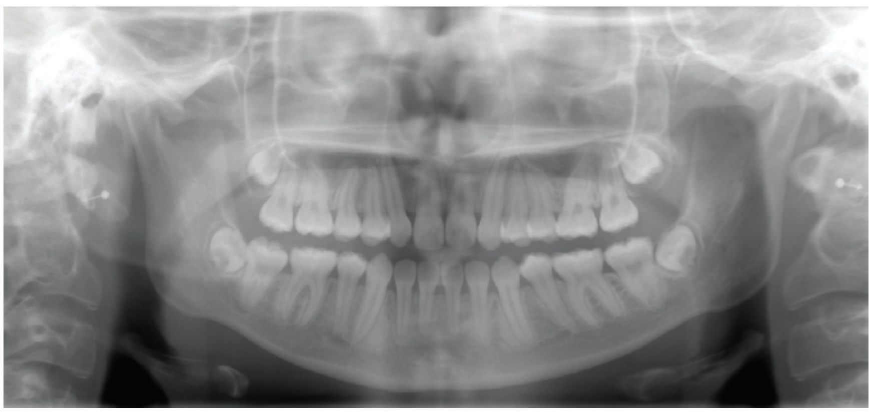

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Figure 1.58 (Continued)

Q: Step 1: What is Lauris' history and CC?

A: Lauris is 42 years old with PMH WRN. PDH includes significant restorative dentistry experience. He states he wants his crossbite corrected.

Q: Step 2: What does your examination reveal? What records are required?

A: Periodontal and TMJ findings are WRN. UDML is centered with the face. No functional shifts are detected. Panoramic and cephalometric radiographs, a full photographic series, and study models are made (Figure 1.59, study models not shown).

Q: Step 3: After a thorough review of Lauris' history, examination, and records, what are his diagnostic findings and problems? Please prepare a list.

A: Lauris' diagnostic findings/problem list and diagnosis is shown in Table 1.12.

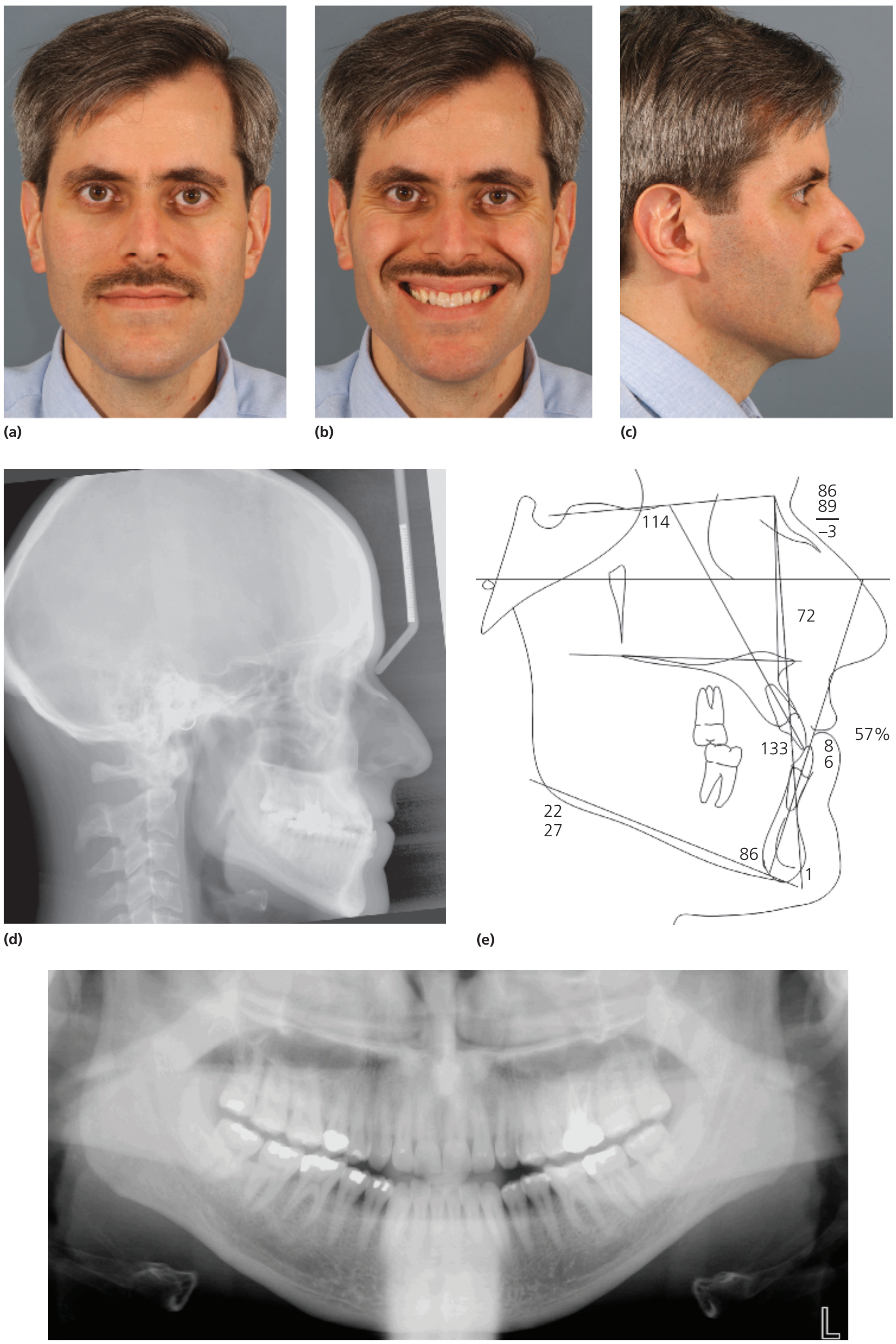

Figure 1.59 Initial records of Lauris.

Figure 1.59 (Continued)

Table 1.12 Diagnostic findings and problem list for Lauris (Figure 1.59).

Full face and profile	**Frontal view**
	Face is symmetric
	Soft tissue long lower anterior facial height (soft tissue Glabella–Subnasale < Subnasale–soft tissue Menton)
	Absence of an interlabial gap (ILG)
	UDML WRN
	Incisal display during posed smile WRN
	Profile view
	Straight to slightly concave/anteriorly divergent profile
	NLA WRN
	Prominent chin
	Lip–chin–throat angle WRN
Ceph Analysis	**Skeletal**
	Maxillary AP position WRN
	Mandibular AP position protrusive (ANB = −3° with maxillary AP position WRN)
	Effective bony Pogonion
	Mildly increased skeletal lower anterior facial height: LAFH/TAFH = 57%
	FMA (22°) and SNMP (27°) mildly flat
	Dental
	Proclined maxillary incisors (U1-SN angle of 114°)
	Upright mandibular incisors (FMIA = 72°)
Radiographs	Adult dentition
Intraoral photos and models	Angle Class III
	Iowa Classification: III (4–5) III (4–5) III (4–5) III (4–5)
	Anterior crossbite occlusion
	OB 20%
	Midlines are not coincident (LDML to right of UDML by 1–2 mm)
	Moderate mandibular anterior crowding
Other	
Diagnosis	Angle Class III malocclusion
	Skeletal mandibular protrusion
	Anterior crossbite occlusion

Q: Step 4: What are your treatment goals?

A: Achieve Class I canines, achieve ideal overbite and overjet and correct his anterior crossbite.

Q: Step 5: What are Lauris' primary problems in the AP, vertical, and transverse dimensions (plus other significant problems)?

A: See Table 1.13

Table 1.13 Primary problems list for Lauris *(apical base/skeletal discrepancies italicized).*

AP	III (4–5) III (4–5) III (4–5) III (4–5) *Mandibular skeletal protrusion*
Vertical	–
Transverse	–
Other	Anterior crossbite

Q: Step 6: Should you discuss surgical options with Lauris?
A: Yes. Lauris is a good example of the challenges you face in deciding treatment for patients with *moderate* apical base discrepancies. On the one hand, you could consider a mandibular setback osteotomy to address his mandibular protrusion and his dental Class III relationship. On the other hand, setting back his mandible could result in an unsatisfactory chin–throat length and profile convexity. It is important to discuss this with him, underscoring the limitations of treatment to ideal occlusion (treatment with surgery) versus treatment to acceptable occlusion with less dramatic effects on his profile (treatment without surgery). After your presentation and discussion, Lauris states that he does not want jaw surgery. Note: With any mandibular setback osteotomy, you must inquire whether the patient has a history of snoring or sleep apnea as the airway cross-section could be reduced with surgery [127]. A sleep study (polysomnography) may be warranted before beginning treatment in such a case.

Q: Step 7: What unknowns do you face? Specifically, what unknowns does he have that you should eliminate before doing anything irreversible (e.g., extracting permanent teeth)?
A: A previously *undetected CR–CO shift*. This underscores an important point regarding examination of patients with crossbites. Crossbite occlusions may occur as a result of a functional shift to avoid tooth interferences. In some instances, growth and dental drift may "convert" the shift to a stable mandibular position. However, in many instances, the shift is not stable and is due to neuromuscular control of the mandible to avoid interferences. Detecting a true path of closure may be difficult in such cases. Therefore, if you are uncertain whether Lauris has a shift, attempt to discover it at a later examination [128]. This was done and it was noted that Lauris has an anterior centric slide of 1 mm. This information will modify the cephalometric diagnosis, as the cephalometric radiograph is taken in maximum intercuspation. Note: As an aid in detecting CR–CO shifts, you can consider "deprogramming" the habitual occlusion by having the patient wear a bite plate or leveling the arches with fixed appliances.

Q: Steps 8 and 9: Because of his age, you cannot treat Lauris' apical base discrepancy with orthopedics. Further, he declined surgery. Therefore, your remaining option is masking. In terms of treating Lauris by masking his apical base discrepancy (either nonextraction or with extraction of permanent teeth), can you list the factors that will play a role in your decision?
A: Factors include the following:

- A 4–5 mm Class III AP dental relationship *but with a 1mm anterior shift from CR to CO (which reduces the Class III relationship to only 3–4 mm)*
- Upright mandibular incisors and proclined maxillary incisors
- Mild-to-moderate crowding in the mandibular anterior
- Robust attached tissue covering roots of mandibular incisors

Q: Step 8: Is Lauris a good candidate for nonextraction treatment?
A: No. Achieving a Class I canine relationship without surgery or extractions may be heroic. Class III elastic wear can be effective in correcting a Class III patient a few millimeters but may be challenging with Lauris' 3–4 mm Class III discrepancy. The costs outweigh the benefits.

Q: Step 9: Is Lauris a good candidate for treatment with anterior tooth extractions?
A: No. Extracting anterior teeth (e.g., a lower incisor) has the benefit of relieving anterior crowding, but extracting an incisor will not help correct his Class III relationship. The cost is not finishing Lauris with a Class I canine relationship.

Q: Is Lauris a good candidate for treatment with posterior tooth extractions?
A: Yes. Extraction of mandibular premolars could provide space to retract the mandibular canines into a Class I relationship and unravel his mild mandibular anterior crowding. The cost is further uprighting his mandibular incisors.

Q: Step 10: Weighing the benefits/costs of these options, which is (are) best for Lauris? What is your recommended treatment plan?
A: The cost of treatment without extractions or with anterior tooth extractions outweighs their benefits. The cost of extracting mandibular premolars and retracting mandibular anterior teeth can be further uprighting of his mandibular incisors. However, the benefits of mandibular premolar extractions are correction of his anterior crossbite (his CC), improvement of his mandibular anterior tooth alignment, and finishing in Class I canine relationship. Extraction of mandibular first premolars was the option that was chosen.

Q: What are the specific objectives of treatment for the maxilla, mandible, maxillary dentition, and mandibular dentition in all three planes of space?
A: Specific objectives of treatment for Lauris are as follows:

Maxilla
AP: Maintain.
Vertical: Maintain.
Transverse: Maintain.

Mandible
AP: Maintain.
Vertical: Maintain.
Transverse: Maintain.

Maxillary dentition
AP: Maintain molars. Maintain incisor position.
Vertical: Maintain molars. Maintain incisor position.
Intermolar width: Maintain.

Mandibular dentition
AP: Protract molars. Retract canines. Upright incisors.
Vertical: Maintain molars. Maintain incisor position.
Intermolar/intercanine width: Maintain intermolar and intercanine widths.

Q: What was the outcome using this approach?
A: Progress and final records are shown in Figures 1.60 and 1.61. Note in Figure 1.60 how Lauris' first premolars have been extracted and his mandibular anterior teeth are being retracted, reciprocally, out of crossbite.Figure 1.61 illustrates Lauris at deband. Your treatment goals have been achieved. Lauris has a wonderful smile and bite, but his mandibular incisors have uprighted (superimposition, Figure 1.61f). Mandibular incisor crown retractions of this magnitude can result in reciprocal movement of mandibular incisor root apices forward through the alveolar process bone, although this does not appear to have happened here (Figure 1.61d). Extraction of lower premolars was an appropriate treatment for Lauris.

As the foregoing examples illustrate, much thought should go into considering whether you treat a patient nonextraction, with extraction of anterior teeth, with some combination of anterior and posterior teeth, or with some combination

Figure 1.60 Progress records of Lauris.

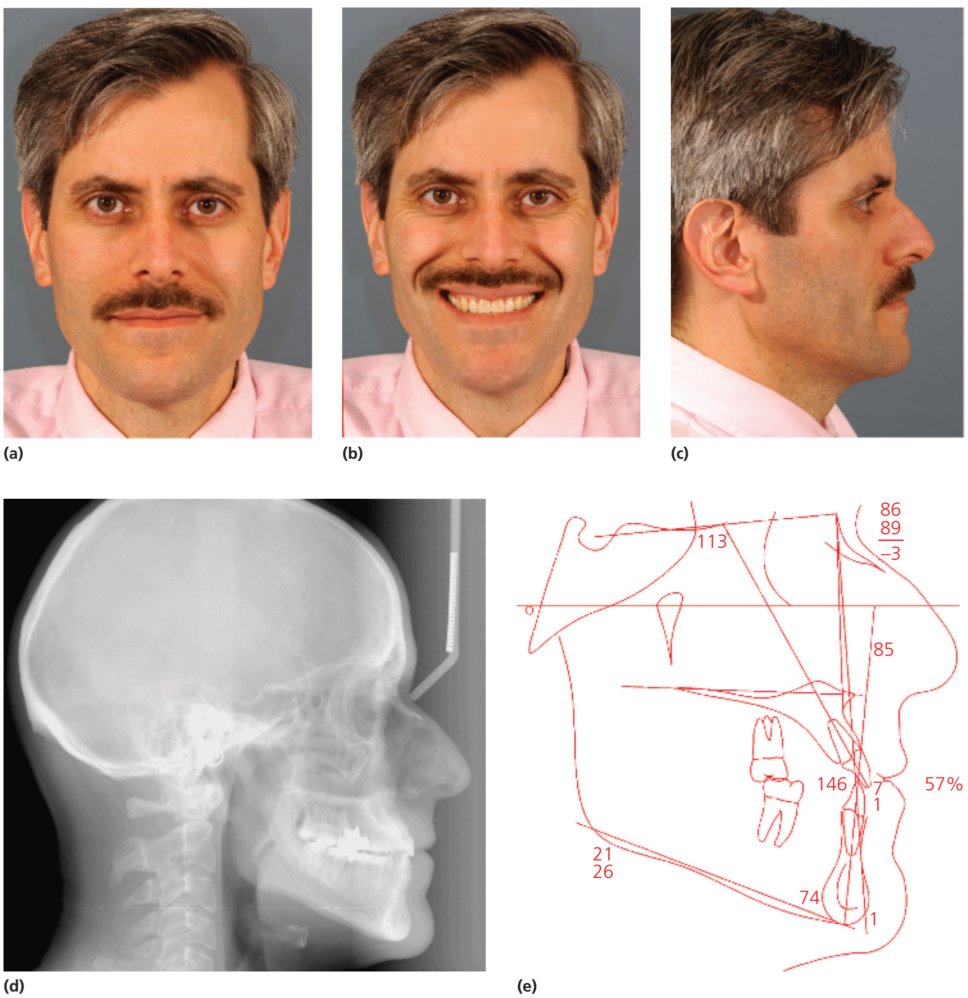

Figure 1.61 Deband records of Lauris.

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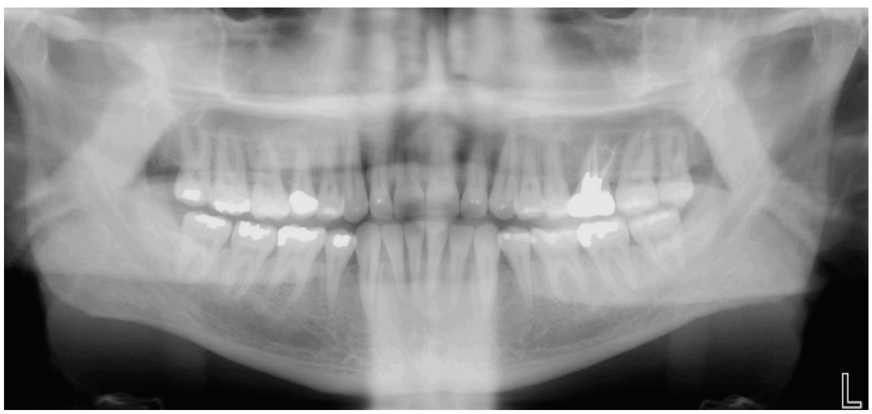

(g)

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Figure 1.61 (Continued)

of posterior teeth. You should always force yourself to consider the costs/benefits of all options.

Now, back to our first patient, Monica (Figure 1.40) to finish with Steps 8, 9, and 10.

Q: What are the benefits/costs of nonextraction, anterior teeth extraction, and posterior teeth extraction combinations for Monica?

A: *Nonextraction*: It has clear benefits to her profile as her nasolabial angle is obtuse, and we do not wish to reduce her maxillary lip support, which could open this angle up more. Further, she has only mild crowding in both arches, her mandibular anterior periodontium is robust, her mandibular incisors are only slightly proclined, and she is unilaterally Class II by only a few millimeters (at the right canines). The cost of nonextraction treatment will be increased mandibular incisor proclination.

Extraction of anterior teeth: Extraction of a mandibular incisor would not be reasonable since she has such mild mandibular anterior crowding. Further, her maxillary incisors appear to be of normal mesiodistal width—so extraction of a mandibular incisor would result in a maxillary excess anterior Bolton discrepancy with excess incisor overjet and excess canine OJ at the end of treatment.

Extraction of posterior teeth: An argument could be made for extraction of a maxillary right premolar to move the maxillary right canine into Class I occlusion. The cost of doing so would be sacrifice of a permanent tooth.

Q: After weighing all nonextraction and extraction options, what treatment options do you recommend for Monica?

A: *Nonextraction:* banding/bonding arches (except for the maxillary lateral incisor), leveling/aligning arches, opening space for the maxillary right lateral incisor, placing the patient on a temporary bite plate (to open her bite and permit her maxillary right lateral incisor to swing forward out of crossbite), bonding the maxillary right lateral incisor, correcting her anterior crossbite, correcting her right Class II relationship with Class II elastics or other Class II force systems, and finishing.

Extraction of maxillary right first premolar: banding/bonding arches (except for her maxillary lateral incisor), extraction of her right first premolar, retraction of her maxillary right canine into a Class I relationship, bonding her maxillary right lateral incisor, placing her on a temporary bite plate, correction of anterior crossbite, leveling/aligning of arches, closure of remaining maxillary space, and finishing.

Remember, now that you have completed Steps 1–10, you must choose your final (recommended) treatment plan and develop your specific objectives of treatment. During your case presentation with Monica, you present the two treatment options. You determine that Monica will be a compliant patient. Patient compliance (e.g., elastic wear) will be an asset with either treatment approach.

Q: What is your recommended treatment plan for Monica?

A: Considering her compliance potential, Monica's final plan was nonextraction with Class II elastic wear on her right.

Q: What are the specific objectives of treatment for the maxilla, mandible, maxillary dentition, and mandibular dentition in all three planes of space?

A: Specific objectives of treatment for Monica are as follows:

Maxilla
- AP: Maintain.
- Vertical: Maintain.
- Transverse: Maintain.

Mandible
- AP: Maintain.
- Vertical: Maintain.
- Transverse: Maintain.

Maxillary dentition
- AP: Maintain left molar/canine. Retract right molar/canine. Procline incisors.
- Vertical: Maintain molars.
- Intermolar width: Maintain.

Mandibular dentition
- AP: Maintain left molar/canine. Maintain right molar/protract right canine.
- Vertical: Level curve of Spee with premolar extrusion and incisor intrusion.
- Intermolar/intercanine width: Maintain intermolar and intercanine widths.

Q: What was the outcome of this treatment approach? How was Monica's deep bite corrected?

A: Progress and final records for Monica are shown in Figures 1.62 and 1.63. Intraoral photographs made before debanding (Figure 1.62) show the maxillary right lateral incisor has been stepped up to balance her right and left maxillary lateral incisor gingival margins in preparation of composite veneers. Final images are shown in Figure 1.63. Her smile has improved dramatically, her crossbite has been corrected, her OB is nearly ideal, and her right Class II canine relationship is now Class I. As illustrated by her initial to posttreatment superimposition (Figure 1.63f), her deep bite was opened by mandibular molar eruption (which rotated her mandible down and back), very mild mandibular incisor proclination, and maxillary incisor proclination (the slight maxillary incisor eruption would tend to deepen her bite).

Q: In summary, what are the 10 steps you should follow to develop treatment plans for patients in this text and in your practice?.

A:
1. Obtain a thorough history and establish the patient's CC.
2. Examine the patient clinically and with appropriate records.
3. Exhaustively list diagnostic findings/problems and make your diagnosis.
4. Define your treatment goals.
5. Focus on solving the *primary* problem in each dimension (plus other major problems).
6. Consider surgical options.
7. Establish unknowns you face—strive to reduce significant unknowns *before* presenting final treatment plan to the patient and before doing anything irreversible.
8. Consider benefits/costs of treating nonextraction.
9. Consider benefits/costs of treating with extraction of anterior teeth or posterior teeth.
10. Weigh benefits/costs of all viable treatment options and establish the recommended plan and treatment objectives.

Remember, at every patient visit, examine the patient, force yourself to review your *primary problems*, and act to address these problems. Over time, the major problems should diminish and your attention can be drawn to smaller and smaller problems—until you finish treatment.

Finally, *at every patient visit do everything you can for the patient*. Even if you and your staff are very busy, do not procras-

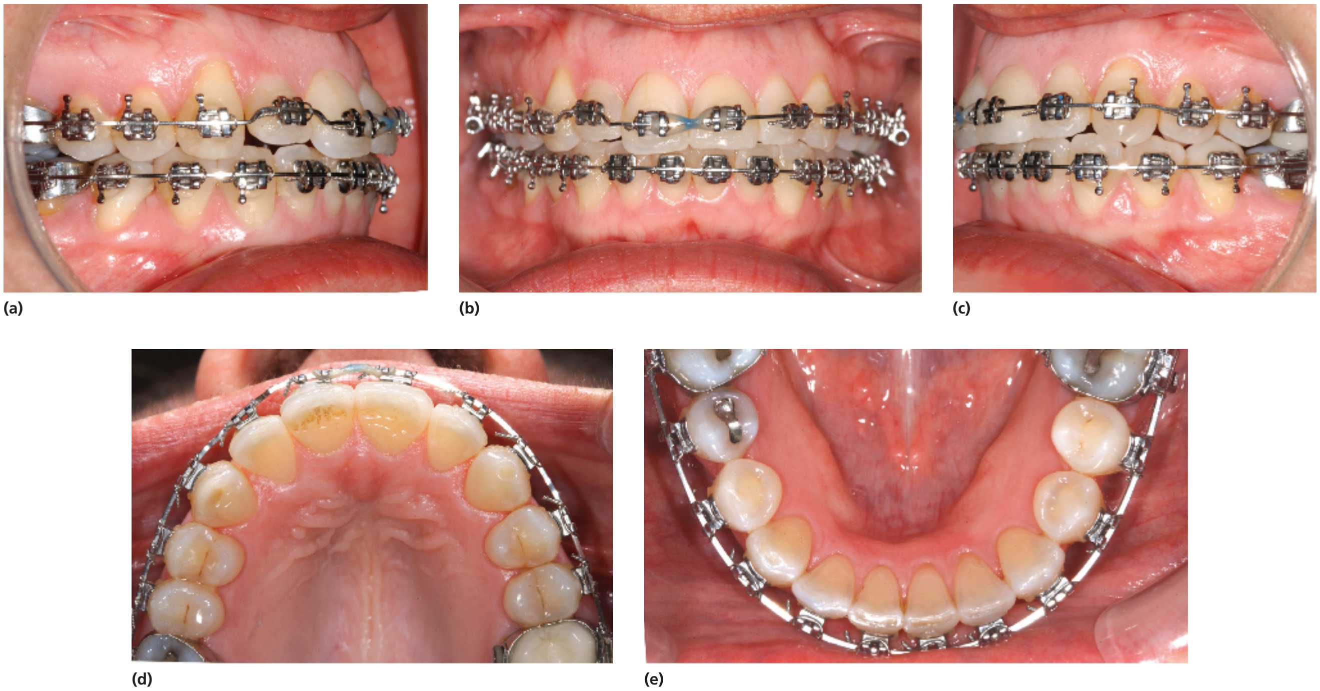


Figure 1.62 Progress records of Monica.

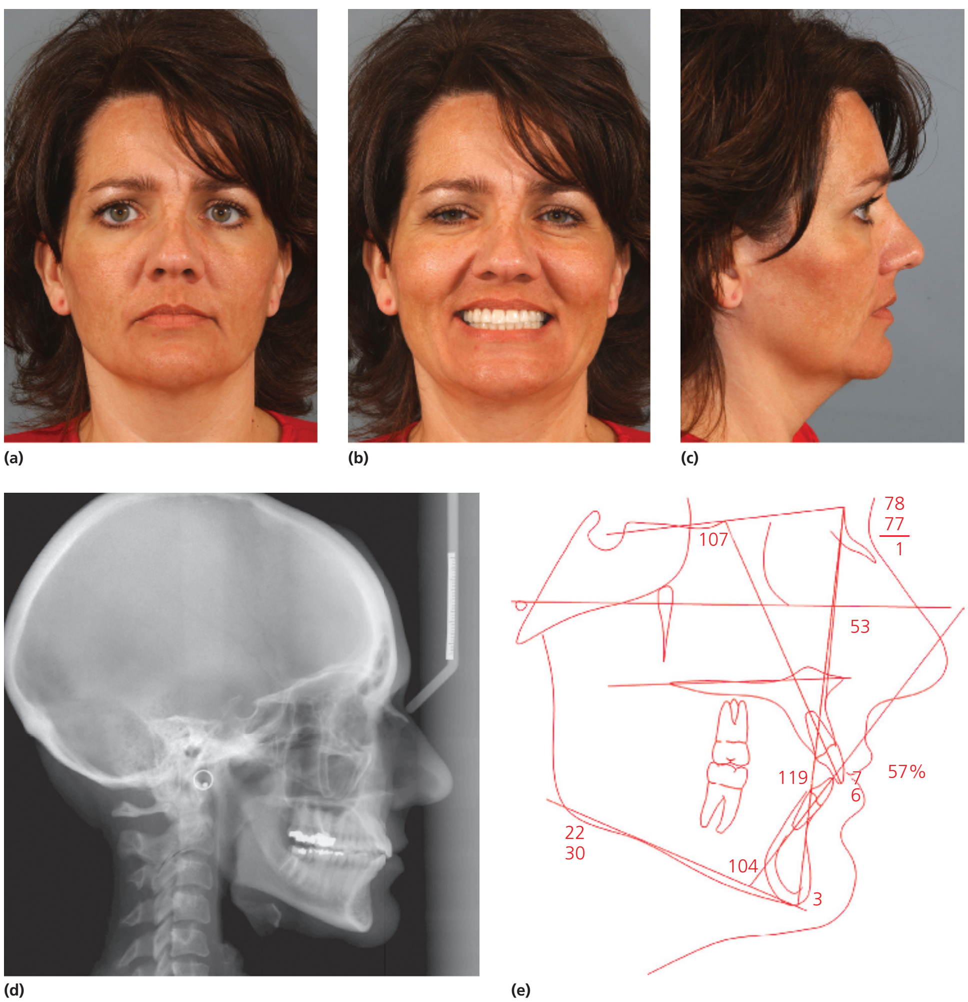
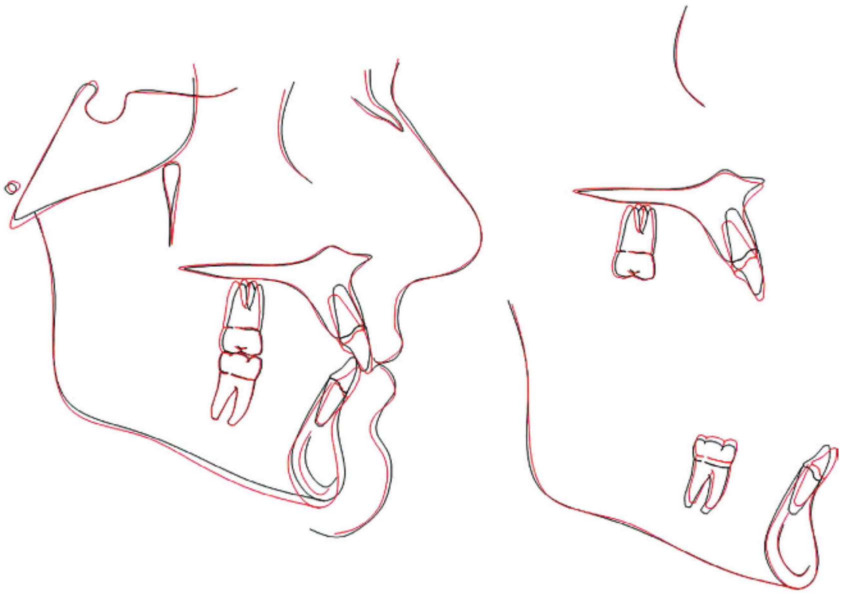
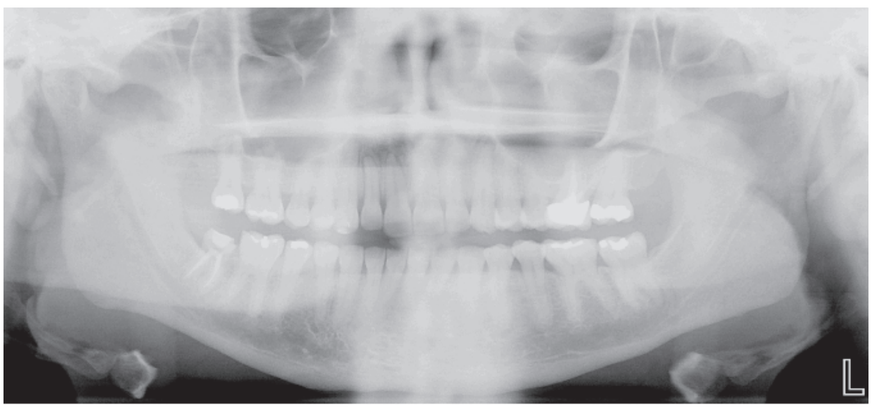


Figure 1.63 Deband records of Monica.



(f)



(g)

(h)

(i)

(j)

(k)

(l)

Figure 1.63 (Continued)

tinate until the next appointment to get as much done for that patient as possible. As a doctor you must follow the golden rule: Do for the patient what you would want done for yourself.

References

1 English JD, Do-Quang T-D, Salas-Lopez AM. Orthodontic records and case evaluation. In: English JD, Peltomaki T, Pham-Litschel K, editors. *Mosby's orthodontic review*. St. Louis: Mosby Elsevier; 2009. p. 39.

2 Proffit WR, Ackerman JL. Orthodontic diagnosis: the development of a problem list. In: Proffit WR, Fields HW, editors. *Contemporary Orthodontics*. 2nd ed. St. Louis: Mosby; 1993. p. 148.

3 Wylie WL, Johnson EL. Rapid evaluation of facial dysplasia in the vertical plane. *Angle Orthod.* 1952;**22**:165–81.

4 Vig RG, Brundo GC. The kinetics of anterior tooth display. *J Prosthet Dent.* 1978;**39**:502–5.

5 Kurth JR, Kokich VG. Open gingival embrasures after orthodontic treatment in adults: prevalence and etiology. *Am J Orthod Dentofac Orthop.* 2001;**120**:116–23.

6 Fridrich KL, Casko JS. Genioplasty strategies for anterior facial vertical dysplasias. *Int J Adult Orthodon Orthognath Surg.* 1997;**12**:35–41.

7 Bjork A. Facial growth in man, studied with the aid of metallic implants. *Acta Odontol Scand.* 1955;**13**:9–34.

8 Bjork A, Skieller V. Normal and abnormal growth of the mandible: a synthesis of longitudinal cephalometric implant studies over a period of 25 years. *Eur J Orthod.* 1983;**5**:1–46.

9 Schudy FF. Vertical growth versus anteroposterior growth as related to function and treatment. *Angle Orthod.* 1964;**34**:75–93.

10 Schudy FF. The rotation of the mandible resulting from growth: its implications in orthodontic treatment. *Angle Orthod.* 1965;**35**:36–50.

11 Skieller V, Bjork A, Linde-Hansen T. Prediction of mandibular growth rotation evaluated from a longitudinal implant sample. *Am J Orthod.* 1984;**86**:359–70.

12 Miller S, Kerr WJ. A new look at mandibular growth—a preliminary report. *Eur J Orthod.* 1992;**14**:95–8.

13 Spady M, Buschang PH, Demirjian A, LaPalme L. Mandibular rotation and angular remodeling during childhood and adolescence. *Am J Hum Biol.* 1992;**4**:683–9.

14 Karlsen AT. Craniofacial growth differences between low and high MP-SN angle males: a longitudinal study. *Angle Orthod.* 1995;**65**:341–50.

15 Chung CH, Wong WW. Craniofacial growth in untreated skeletal Class II subjects: a longitudinal study. *Am J Orthod Dentofacial Orthop.* 2002;**122**:619–26.

16 Bishara SE, Jakobsen JR. Longitudinal changes in three normal facial types. *Am J Orthod.* 1985;**88**:466–502.

17 Jacob HB, Buschang PH. Vertical craniofacial growth changes in French-Canadians between 10 and 15 years of age. *Am J Orthod Dentofacial Orthop.* 2011;**139**: 797–805.

18 Zankl A, Eberle L, Molinari L, Schinzel A. Growth charts for nose length, nasal protrusion, and philtrum length from birth to 97 years. *Am J Med Genet.* 2002;**111**:388–91.

19 Fundalej P. Long-term changes of the upper lip position relative to the incisor edge. *Am J Orthod Dentofacial Orthop.* 2008;**135**:204–9.

20 Burstone, C. Lip posture and its significance in treatment planning. *Am J Orthod.* 1967;**53**:262–84.

21 Vig PS, Cohen AM. Vertical growth of the lips a serial cephalometric study. *Am J Orthod.* 1979;**75**:405–15.

22 Sarver DM, Ackerman MB. Dynamic smile visualization and quantification: Part 1. Evolution of the concept and dynamic records for smile capture. *Am J Orthod Dentofacial Orthop*. 2003;**124**:4–12.

23 Mammandras AH. Linear changes of the maxillary and mandibular lips. *Am J Orthod Dentofacial Orthop.* 1988;**94**:405–10.

24 Sarver DM. The importance of incisor positioning in the esthetic smile: the smile arc. *Am J Orthod Dentofacial Orthop.* 2001;**120**:98–111.

25 Hulsey CM. An esthetic evaluation of lip-teeth relationships present in the smile. *Am J Orthod.* 1970;**57**:132–44.

26 Mackley RJ. An evaluation of smiles before and after orthodontic treatment. *Angle Orthod.* 1993;**63**:183–9; discussion 190.

27 Ackerman JL, Ackerman MB, Brensinger CM, Landis JR. A morphometric analysis of the posed smile. *Clin Orthod Res.* 1998;**1**:2–11.

28 Kokich VO, Kokich VG, Kiyak HA. Perceptions of dental professionals and laypersons to altered dental esthetics: asymmetric and symmetric situations. *Am J Orthod Dentofacial Orthop.* 2006;**130**:141–51.

29 Benson KJ, Laskin DM. Upper lip asymmetry in adults during smiling. *J Oral Maxillofac Surg.* 2001;**59**:396–8.

30 Kokich VO Jr, Kiyak HA, Shapiro PA. Comparing the perception of dentists and lay people to altered dental esthetics. *J Esthet Dent.* 1999:**11**;311–24.

31 Polo M. Botulinum toxin type A in the treatment of excessive gingival display. *Am J Orthod Dentofacial Orthop.* 2005;**127**:214–8.

32 Niamtu J 3rd. Botox injections for gummy smiles. *Am J Orthod Dentofacial Orthop.* 2008;**133**:782–3; author reply 783-4.

33 Scott CR, Goonewardene MS, Murray K. Influence of lips on the perception of malocclusion. *Am J Orthod Dentofacial Orthop.* 2006;**130**:152–62.

34 Janson G, Branco NC, Fernandes TM, Sathler R, Garib D, Lauris JR. Influence of orthodontic treatment, midline position, buccal corridor and smile arc on smile attractiveness. *Angle Orthod.* 2011;**81**:153–61.

35 Pinho S, Ciriaco C, Faber J, Lenza MA. Impact of dental asymmetries on the perception of smile esthetics. *Am J Orthod Dentofacial Orthop.* 2007;**132**:748–53.

36 Roden-Johnson D, Gallerano R, English J. The effects of buccal corridor spaces and arch form on smile esthetics. *Am J Orthod Dentofacial Orthop.* 2005;**127**:343–50.

37 Moore T, Southard KA, Casko JS, Qian F, Southard TE. Buccal corridors and smile esthetics. *Am J Orthod Dentofacial Orthop.* 2005;**127**:208–13.

38 Jornung J, Fardal O. Perceptions of patients' smiles: a comparison of patients' and dentists' opinions. *J Am Dent Assoc.* 2007;**138**:1544–53.

39 Dunn WJ, Murchison DF, Broome JC. Esthetics: patients' perceptions of dental attractiveness. *J Prosthodont.* 1996;**5**:166–71.

40 Correa BD, Viera Bittencourt MA, Machado AW. Influence of maxillary canine gingival margin asymmetries on the perception of smile esthetics among orthodontists and laypersons. *Am J Orthod Dentofacial Orthop.* 2014;**145**:55–63.

41 Dixon AD, Hoyte DA, Ronning O. *Fundamentals of craniofacial growth*. Boca Raton: CRC Press, 1997.

42 Enlow DH, Hans MG. *Essentials of facial growth*. 2nd ed. Ann Arbor: Needham Press; 2008.

43 Hartsfield JK Jr, Morford LA, Otero LM. In: Bourzgui F, editor. Genetic factors affecting facial growth, Orthodontics—basic aspects and clinical considerations. Rijeka, Croatia: InTech. [cited 2014 Aug 11]. Available from: http://www.intechopen.com/books/orthodontics-basic-aspects-and-clinical-considerations/genetic-factors-affecting-facial-growth. pp. 125–52.

44 Duterloo HS, Planché PG. *Handbook of cephalometric superimposition*. Hanover Park: Quintessence; 2011.

45 Bastir M, Rosas A, O'Higgins P. Craniofacial levels and the morphologic maturation of the human skull. *J Anat.* 2006;**209**:637–54.

46 Bjork A. Cranial base development. *Am J Orthod.* 1955;**41**:198–225.

47 Buschang PH, Santos-Pinto A. Condylar growth and glenoid fossa displacement during childhood and adolescence. *Am J Orthod Dentofacial Orthop.* 1998;**113**:437–42.

48 Bjork A, Skieller V. Growth of the maxilla in three dimensions as revealed radiographically by the implant method. *Br J Orthod.* 1977;**4**:53–64.

49 Baumrind S, Ben-Bassat Y, Bravo LA, Curry S, Korn EL. Partitioning the components of maxillary tooth displacement by the comparison of data from three cephalometric superimpositions. *Angle Orthod.* 1996;**66**:111–24.

50 Ranly DM. *A synopsis of craniofacial growth*. 2nd ed. Norwalk: Appleton and Lange; 1988.

51 Buschang PH. Craniofacial growth and development. In: English, JD, Peltomaki T, Litschel K, editors. *Mosby's orthodontic review*. St. Louis: Elsevier Mosby; 2009. pp. 1–12.

52 Tanner JM, Whitehouse RH, Takaishi M. Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children, 1965. *Arch Dis Child.* 1966;**41**:454–71.

53 Malina RM, Bouchard C, Beunen G. Human growth: selected aspects of current research on well-nourished children. *Ann Rev Anthropol.* 1988;**17**:187–219.

54 Mellion ZJ, Behrents, RG, Johnston LE Jr. The pattern of facial skeletal growth and its relationship to various common indexes of maturation. *Am J Orthod Dentofacial Orthop.* 2013;**143**:845–54.

55 Bambha JK, Van Natta P. Longitudinal study of facial growth in relation to skeletal maturation during adolescence. *Am J Orthod.* 1963;**49**:481–93.

56 Hunter CJ. The correlation of facial growth with body height and skeletal maturation at adolescence. *Angle Orthod.* 1966;**36**:44–54.

57 Mitani H. Contributions of the posterior cranial base and mandibular condyles to facial depth and height during puberty. *Angle Orthod.* 1973;**43**:337–43.

58 Buschang PH, Jacob HB, Demirjian A. Female adolescent craniofacial growth spurts: real or fiction? *Eur J Orthod.* 2013;**35**:819–25.

59 Nanda RS. The rates of growth of several facial components measured from serial cephalometric roentgenograms. *Am J Orthod.* 1955;**41**:658–73.

60 Buschang PH, Tanguay R, Demirjian A, LaPalme L, Goldstein H. Modeling longitudinal mandibular growth: percentiles for gnathion from 6 to 15 years of age in girls. *Am J Orthod Dentofacial Orthop.* 1989;**95**:60–6.

61 van der Beek MC, Hoeksma JB, Prahl-Andersen B. Vertical facial growth and statural growth in girls: a longitudinal comparison. *Eur J Orthod.* 1996;**18**:549–55.

62 Buschang PH, Martins J. Childhood and adolescent changes of skeletal relationships. *Angle Orthod.* 1998;**68**:199–206; discussion 207-8.

63 Chvatal BA, Behrents RG, Ceen RF, Buschang PH. Development and testing of multilevel models for longitudinal craniofacial growth prediction. *Am J Orthod Dentofacial Orthop.* 2005;**128**:45–56.

64 Bishara SE, Peterson LC, Bishara EC. Changes in facial dimensions and relationships between the ages of 5 and 25 years. *Am J Orthod.* 1984;**85**:238–52.

65 van der Beek MC, Hoeksma JB, Prahl-Andersen B. Vertical facial growth: a longitudinal study from 7 to 14 years of age. *Eur J Orthod.* 1991;**13**:202-8.

66 Houston WJ. Relationships between skeletal maturity estimated from hand-wrist radiographs and the timing of the adolescent growth spurt. *Eur J Orthod.* 1980;**2**:81–93.

67 Hagg U, Taranger J. Maturation indicators and the pubertal growth spurt. *Am J Orthod.* 1982;**82**:299–309.

68 Flores-Mir C, Nebbe B, Major PW. Use of skeletal maturation based on hand-wrist radiographic analysis as a predictor of facial growth: a systematic review. *Angle Orthod.* 2004;**74**:118–24.

69 Hunter WS, Baumrind S, Popovich F, Jorgensen G. Forecasting the timing of peak mandibular growth in males by using skeletal age. *Am J Orthop Dentofacial Orthod.* 2007;**131**:327–33.

70 Gabriel DB, Southard KA, Qian F, Marshall SD, Franciscus RG, Southard TE. Cervical vertebrae maturation method: poor reproducibility. *Am J Orthod Dentofacial Orthop.* 2009;**136**:478.e1–7; discussion 478-80.

71 Nestman TS, Marshall SD, Qian F, Holton N, Franciscus RG, Southard TE. Cervical vertebrae maturation method morphologic criteria: poor reproducibility. *Am J Orthod Dentofacial Orthop.* 2011;**140**:182-8.

72 Baume RM, Buschang PH, Weinstein S. Stature, head height, and growth of the vertical face. *Am J Orthod.* 1983;**83**:477–84.

73 Tanner JM, Cameron N. Investigation of the mid-growth spurt in height, weight and limb circumferences in single-year velocity data from the London, 1966–67 growth survey. *Ann Hum Biol.* 1980;**7**:565–77.

74 Gasser T, Müller HG, Köhler W, Prader A, Largo R, Molinari L. An analysis of the mid-growth and adolescent spurts of height based on acceleration. *Ann Hum Biol.* 1985;**12**:129–48.

75 Molinari L, Largo RH, Prader A. Analysis of the growth spurt at age seven (mid-growth spurt). *Helv Paediatr Acta.* 1980;**35**:325–34.

76 Sheehy A, Gasser T, Molinari L, Largo RH. An analysis of variance of the pubertal and midgrowth spurts for length and width. *Ann Hum Biol.* 1999;**26**:309–31.

77 Buschang PH, Tanguay R, Demirjian A, LaPalme L, Turkewicz J. Mathematical models of longitudinal mandibular growth for children with normal and untreated Class II, division 1 malocclusion. *Eur J Orthod.* 1988;**10**:227–34.

78 Proffit WR, Fields HW Jr, Sarver DM, editors. *Contemporary orthodontics*. 4th ed. St. Louis: Mosby Elsevier Health Sciences; 2007.

79 Hartsfield, J.K., Jr. Genetics and orthodontics. In: Graber LW, Vanarsdall RL, Vig KWL, editors. *Orthodontics: current principles and techniques*. 5th ed. St. Louis: Elsevier Mosby; 2011. pp. 139–56.

80 Nielsen IL. Vertical malocclusions: etiology, development, diagnosis and some aspects of treatment. *Angle Orthod.* 1991;**61**:247–60.

81 Solow B, Kreiborg S. Soft-tissue stretching: a possible control factor in craniofacial morphogenesis. *Scand J Dent Res.* 1977;**85**:505–7.

82 Linder-Aronson S. Adenoids: their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. *Acta Otolaryngol Suppl.* 1970;**265**:1–132.

83 Linder-Aronson S Respiratory function in relation to facial morphology and the dentition. *Br J Orthod.* 1979;**6**:59–71.

84 Solow B, Siersbæk-Nielsen S, Greve E. Airway adequacy, head posture, and craniofacial morphology. *Am J Orthod.* 1984;**86**:214–23.

85 Behlfelt K, Linder-Aronson S, McWilliam J, Neander P, Laage-Hellman J. Craniofacial morphology in children with and without enlarged tonsils. *Eur J Orthod.* 1990;**12**:233–43.

86 Vig KW. Nasal obstruction and facial growth: the strength of evidence for clinical assumptions. *Am J Orthod Dentofacial Orthop.* 1998;**113**:603–11.

87 Peltomäki T. The effect of mode of breathing on craniofacial growth—revisited. *Eur J Orthod.* 2007;**29**:426–9.

88 Vogl C, Atchley WR, Cowley DE, Crenshaw P, Murray JD, Pomp D. The epigenetic influence of growth hormone on skeletal development. *Growth Dev Aging.* 1993;**57**:163–82.

89 Yamaguchi T, Maki K, Shibasaki Y. Growth hormone receptor gene variant and mandibular height in the normal Japanese population. *Am J Orthod Dentofacial Orthop.* 2001;**119**:650–3.

90 Zhou J, Lu Y, Gao XH, Chen YC, Lu JJ, Bai YX, et al. The growth hormone receptor gene is associated with mandibular height in a Chinese population. *J Dent Res.* 2005;**84**:1052–6.

91 Tomoyasu Y, Yamaguchi T, Tajima A, Nakajima T, Inoue I, Maki K. Further evidence for an association between mandibular height and the growth hormone receptor gene in a Japanese population. *Am J Orthod Dentofacial Orthop.* 2009;**136**:536–41.

92 van Spronsen PH. Long-face craniofacial morphology: cause or effect of weak masticatory musculature? *Semin Orthod.* 2010;**16**:99–117.

93 Buschang PH, Heider J, Carrillo R. The morphological characteristics, growth and etiology of the hyperdivergent phenotype. *Semin Orthod.* 2013;**19**:212–26.

94 Björk A. Variations in the growth pattern of the human mandible: longitudinal radiographic study by the implant method. *J Dent Res.* 1963;**42** Pt.2:400–11.

95 Björk A, Skieller V. Facial development and tooth eruption. An implant study at the age of puberty. *Am J Orthod*. 1972;**62**:339–83.

96 Odegaard J. Growth of the mandible studied with the aid of metal implant. *Am J Orthod.* 1970;**57**:145–57.

97 Odegaard J. Mandibular rotation studies with the aid of metal implants. *Am J Orthod.* 1970;**58**:448–54.

98 Lavergne J, Gasson N. Operational definitions of mandibular morphogenetic and positional rotations. *Scand J Dent Res.* 1977;**85**:185–92.

99 Sinclair PM, Little RM. Dentofacial maturation of untreated normals. *Am J Orthod.* 1985;**88**:146–56.

100 Lavergne J, Gasson N. Direction and intensity of mandibular rotation in the sagittal adjustment during growth of the jaws. *Scand J Dent Res.* 1977;**85**:193–6.

101 Buschang PH, Gandini Jr. LG. Mandibular skeletal growth and modelling between 10 and 15 years of age. *Eur J Orthod.* 2002;**24**:69–79.

102 Mojdehi M, Buschang PH, English JD, Wolford LM. Postsurgical growth changes in the mandible of adolescents with vertical maxillary excess growth pattern. *Am J Orthod Dentofacial Orthop.* 2001;**119**:106–16.

103 Kim J, Nielsen IL. A longitudinal study of condylar growth and mandibular rotation in untreated subjects with Class II malocclusion. *Angle Orthod.* 2002;**72**:105–11.

104 Björk A. Prediction of mandibular growth rotation. *Am J Orthod.* 1969;**55**:585–99.

105 Baumrind S, Korn EL, West EE. Prediction of mandibular rotation: an empirical test of clinician performance. *Am J Orthod.* 1984;**86**:371–85.

106 Leslie LR, Southard TE, Southard KA, Casko JS, Jakobsen JR, Tolley EA, et al. Prediction of mandibular growth rotation: assessment of the Skieller, Björk, and Linde-Hansen method. *Am J Orthod Dentofacial Orthop.* 1998;**114**:659–67.

107 Nanda SK. Growth patterns in subjects with long and short faces. *Am J Orthod Dentofacial Orthop.* 1990;**98**:247–58.

108 Isaacson JR, Isaacson RJ, Speidel TM, Worms FW. Extreme variation in vertical facial growth and associated variation in skeletal and dental relations. *Angle Orthod.* 1971;**41**:219–29.

109 Kuitert R, Beckmann S, van Loenen M, Tuinzing B, Zentner A. Dentoalveolar compensation in subjects with vertical skeletal dysplasia. *Am J Orthod Dentofacial Orthop.* 2006;**129**:649–57.

110 Janson GR, Metaxas A, Woodside DG. Variation in maxillary and mandibular molar and incisor vertical dimension in 12-year-old subjects with excess, normal, and short lower anterior face height. *Am J Orthod Dentofacial Orthop.* 1994;**106**:409–18.

111 Liu SS, Buschang PH. How does tooth eruption relate to vertical mandibular growth displacement? *Am J Orthod Dentofacial Orthop.* 2011;**139**:745–51.

112 Alexander RG. The effects on tooth position and maxillofacial vertical growth during treatment of scoliosis with the Milwaukee brace. *Am J Orthod.* 1966;**52**:161–89.

113 Elder, JR, Tuenge RH, Cephalometric and histologic changes produced by extra-oral high-pull traction to the maxilla in Macaca mulatta. *Am J Orthod.* 1974;**66**:599–616.

114 Stöckli PW, Willert HG. Tissue reactions in the temporomandibular joint resulting from anterior displacement of the mandible in the monkey. *Am J Orthod.* 1971;**60**:142–55.

115 Woodside DG., Metaxas A., Altuna G. The influence of functional appliance therapy on glenoid fossa remodeling. *Am J Orthod Dentofacial Orthop.* 1987;**92**:181–98.

116 Voudouris JC, Woodside DG, Altuna G, Angelopoulos G, Bourque PJ, Lacouture CY. Condyle-fossa modifications and muscle interactions during Herbst treatment, Part 2. Results and conclusions. *Am J Orthod Dentofacial Orthop.* 2003;**124**:13–29.

117 Bishara SE, Cummins DM, Zaher AR. Treatment and posttreatment changes in patients with Class II, Division 1 malocclusion after extraction and nonextraction treatment. *Am J Orthod Dentofacial Orthop.* 1997;**111**:18–27.

118 Ritucci R, Nanda R. The effect of chin cup therapy on the growth and development of the cranial base and midface. *Am J Orthod Dentofacial Orthop.* 1986;**90**:475–83.

119 McNamara JA Jr, Bryan FA. Long-term mandibular adaptations to protrusive function: an experimental study in Macaca mulatta. *Am J Orthod Dentofacial Orthop.* 1987;**92**:98–108.
120 Pancherz H. The effects, limitations, and long-term dentofacial adaptations to treatment with the Herbst appliance. *Semin Orthod.* 1997;**3**:232–43.
121 Konik M, Pancherz H, Hansen K. The mechanism of Class II correction in late Herbst treatment. *Am J Orthod Dentofacial Orthop.* 1997;**112**:87–91.
122 Demisch A, Ingervall B, Thüer U. Mandibular displacement in Angle Class II, division 2 malocclusion. *Am J Orthod Dentofacial Orthop.* 1992;**102**:509–18.
123 Marshall WA, Tanner JM: Puberty. In: Falkner F, Tanner J, editors. *Human growth: a comprehensive treatise. Vol. 2, Postnatal growth: neurobiology*. 2nd ed. New York: Plenum Publishing, 1986. pp. 171–203.
124 Brandt S, Safirstein GR. Different extractions for different malocclusions. *Am J Orthod.* 1975;**68**:15–41.
125 Creekmore TD. Where teeth should be positioned in the face and jaws and how to get them there. *J Clin Orthod.* 1997;**31**:586–608.
126 Kim TK, Kim JT, Mah J, Yang WS, Baek SH. First or second premolar extraction effects on facial vertical dimension. *Angle Orthod.* 2005;**75**:177–82.
127 Hwang S, Chung CJ, Choi YJ, Huh JK, Kim KH. Changes in hyoid, tongue and pharyngeal airway after mandibular setback surgery by intraoral vertical ramus osteotomy. *Angle Orthod.* 2010;**80**:302–8.
128 Okeson JP. *Management of temporomandibular disorders and occlusion*. 5th ed. St. Louis: Mosby; 2003.