6Ways to Design a Face

Corrective Jaw Surgery to Optimize Bite, Airway, and Facial Balance

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6 WAYS TO DESIGN A FACE

Corrective Jaw Surgery to Optimize Bite, Airway, and Facial Balance

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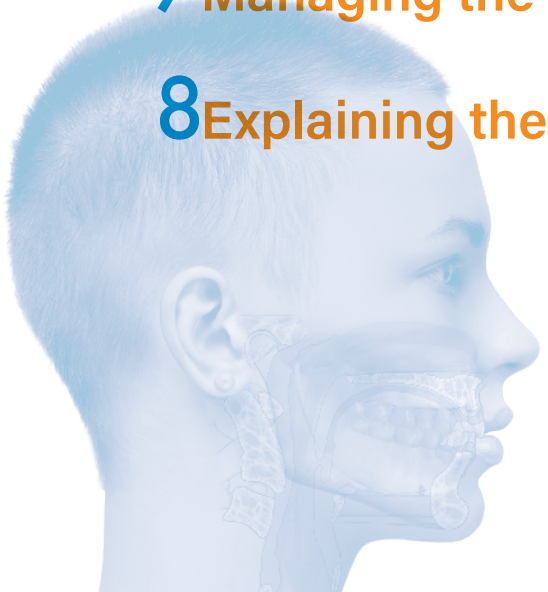
contents



foreword

Paul Coceancig is not the first dentist to correlate small jaws in adolescents with bad bites and crooked teeth, but his ideas about how to treat this condition—which seems to be rampant in Western society—depart significantly from established views.

Based on his extensive professional training and experience, and to some extent on the abysmal failure of his own orthodontic treatment as a teenager and again during dental school, Coceancig rejects the basic tenets of mainstream orthodontics, with its focus on the occlusion and its reliance on tooth extraction to make the teeth fit in the jaws. The long-term ramifications of this treatment approach—a disproportionate face, a collapsing tongue, and a compromised airway—are widespread and devastating to the overall health of patients.

Coceancig presents and argues very convincingly in favor of an alternative, holistic approach to correcting bad bites and crooked teeth that he has developed over many years, based on the premise that having 32 functional teeth is the natural and ideal state of every adult human. In his view, abnormal states of facial profile and bad bite fall on a spectrum, yet they all have exactly the same etiology—a small mandible. Furthermore, the most efficient way to permanently change a small jaw into one that is more proportionate to the rest of the face is through corrective jaw surgery, using simple distraction techniques that Coceancig has refined over many years with excellent results, as demonstrated by the dozens of well-documented cases he presents throughout his book.

By adopting Coceancig's philosophy and surgical protocol, oral and maxillofacial surgeons can reverse the cosmetic effects and correct for the orthodontic consequences of the small jaw in their adolescent patients. In addition, a variation of this surgery is highly effective in reversing the destructive consequences of a constricted airway on older people who have already developed obstructive sleep apnea (OSA) as a result of their small mandibles. By curing people of breathing difficulties, such as snoring, or of the risk or presence of OSA through corrective jaw surgery, the oral and maxillofacial surgeon is also helping to prevent a range of other diseases that are commonly associated with OSA.

Arun K. Garg, DMD
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INTRODUCTION

For over a decade my wife has been persistently asking me to write a book about corrective facial skeletal surgery. I resisted for a while, thinking the process to be too big, too elaborate, and too broad. After all, the writing process is very introspective, which for me is very isolating, and requires significant time and dedicated sustained concentration. As my wife can attest, to successfully write a book comes at the sacrifice of children, parents, friends, referrers, business associates, and patients.

Over the course of my career up to this point, certain commonly held philosophies, tenets, and basic ideas concerning what is a face, what is abnormality, and what is considered corrective have all needed alteration. These questions and their evolving answers have shaped my thinking and conceptualization of the necessary solutions.

I suppose, in formulating a basis for what follows in this book, I first need to introduce who I am, where I came from, and what made me.

MY STORY

I began dentistry when I was 17 at the University of Sydney in Australia. The Australian profession was then, as it is now, extremely conservative. The professors all hailed from the Commonwealth, so we students became an ordered, intellectualized, and socially privileged group focused on the community benefit of uniformly applied oral health care. Australian style.

At 23, and newly graduated as a "dental surgeon," I took a job at the local public dental hospital, and while my classmates were entering private practice and buying new cars and homes, I learned to extract teeth and started my fellowship studies with the Royal Australasian College of Dental Surgeons, which opened the door to specialist training positions in oral surgery. This led me to meet Professor John Edgar deBurgh Norman AO, Associate Professor Geoffrey McKellar, and Drs Alf Coren and Peter Vickers, independently brilliant people who would take me into their hospitals and show me orthognathic surgery. I held a retractor and listened while they talked; they advised me to forget about extracting teeth and formed my intellectual connection to orthognathic surgery. Their confidence and mentorship supported an offer for me to enroll in a 5-year specialty oral and maxillofacial surgery (OMS) program across the Tasman Sea in New Zealand. I was the only applicant.

So at 25 I flew to Otago University in Dunedin, where I was enrolled in a shortened medical degree program and also started a combined 3-year specialty degree in oral surgery. I was transferred to the Christchurch Medical School the following year, and with my new girlfriend (now wife) beside me, over the next 4 years I continued to study undergraduate medicine part-time and extracted a lot of teeth, fixed broken jaws, and learned the Kiwi public hospital version of surgically working alongside orthodontists. New Zealand style.

I was 29 when I eventually graduated from both programs. I was a dental surgeon, I was a medical doctor, and now I was a Kiwi version of an oral and maxillofacial surgeon. I was also now married to a Kiwi and had a first-born Kiwi daughter.

I wanted to return to Australia, so we packed up and headed home. After 2 years of certifying my New Zealand

medical degree in Australia, I knew I needed just a couple more years of dedicated jaw surgery mentorship. Not wanting to pursue further training in the United Kingdom, luckily I received an offer from the Singapore General Hospital to work in a private-public capacity under Dr Raymond Peck Hong Lian, a British-trained maxillofacial surgeon. He was to become my final teacher. After almost 2 years of constant operating in this vestige of England surrounded by Asia, I came to practically learn everything there was then to know about corrective jaw surgery. Singapore style.

Sixteen years after starting my training in dentistry, I had completed my Australian specialty qualification in maxillofacial surgery (the FRACDS-OMS) and opened my private specialty office shortly thereafter in Newcastle, Australia. Mostly I extracted diseased teeth for dentists and perfectly good teeth for orthodontists. As it is for most maxillofacial surgeons worldwide, the majority of those teeth were crowded premolars and impacted third molars. Slowly, as confidence grew among my referring orthodontists, a small trickle of corrective jaw surgery cases came through my doors too.

Today my private practice is almost entirely derived from corrective jaw surgery. I now rarely extract teeth.

REFERRAL MODEL FOR CORRECTIVE JAW SURGERY

Because orthognathic surgery is rare, and because it is basically used only following failed specialist orthodontics, the general professional dental view of orthognathic surgery is mostly negative. There is little dental understanding of what corrective jaw surgery is. Rather than seeing orthognathic surgery as being therapeutic, medical, necessary, or something of high reliability or functionality, a referral for orthognathic surgery is often discouraged by the dentist as an unnecessary and highly risky extreme. This pervasive dental view that reduces all forms of jaw surgery to a limited role of only removing teeth means that orthognathic surgery is extremely rare. In all practicality, and regardless of training, most oral and maxillofacial surgeons are reduced only to the role of an oral surgeon.

Few private surgeons practically or routinely offer corrective jaw surgery procedures because orthognathic surgery is seen as secondary to orthodontics. The teeth are always corrected first, followed reluctantly by the jaws.

In all of my training, I was taught that orthognathic surgery was based upon a pragmatic premise of performing to what

ner than seeing orthognathic surgery as being therapeutic, medical, necessary, or something of high reliability or functionality, a referral for orthognathic surgery is often discouraged by the dentist as an unnecessary and highly risky extreme.

an orthodontist wanted. There was simply no other source of referral. And to an enormous degree, this repeat stream of action-reaction generated a master-servant relationship. Orthodontists literally fed oral surgery practices, albeit with dental extractions and mostly impacted third molars, and to a much smaller degree with remedial orthognathic surgery when extraction-based orthodontics simply didn't work. To run a successful surgical business, I had to fundamentally believe in the orthodontic interpretation of everything to do with impacted and crowded teeth and in the primacy of orthodontists being the first to treat, examine, and interpret.

The major problem with this model, however, is that it traditionally ignores the face and the airway.

MY FIRST ORTHODONTIC EXPERIENCE

When I was 13 years old, my impacted canine tooth erupted extremely high behind my upper lip, and my mother recognized that it would never spontaneously be normal. In 1957, her 14-year-old sister had had an impacted and badly erupted canine removed on the advice of her orthodontist, and at 76 years old my Aunty Pam still complains bitterly of its effects on the symmetry and attractiveness of her face. As a surgical nurse trained at Sydney Hospital in Macquarie Street, my mother resolved in 1982 that her sister's fate would not also befall me, so she took me to a highly recommended Macquarie Street orthodontic specialist in Sydney.

He advised against my mother's proposal that expanding my maxilla would allow enough room to easily fit all my crooked teeth. He also said it was fanciful to believe that stimulating my maxilla to expand would somehow correct the underbite I was developing. He believed her proposal very controversial, impractical, and unfeasible. He further explained that I had a very flat middle face, that my

cheekbones were naturally small, and that extracting the canine would indeed be a terrible idea and make the whole appearance of my face worse. I remember feeling very ugly.

My father was very skeptical about removing four perfectly good premolar teeth in order to orthodontically bring down a single impacted canine tooth. It was at this point that my mother asked if there was any way to surgically bring my maxilla forward. She suggested that this would improve my facial proportions and possibly make my nasal breathing better.

You see it was no coincidence that on this same famous Sydney street worked my allergist, who every month would give me a needle against my dust mite allergy. Further down in another building was my respiratory specialist who treated my chronic asthma. Next door to that was the ENT surgeon who had removed my tonsils when I was 4 years old. And of course there was also my pediatrician who tried to help me grow normally despite my early recurrent croup, ongoing throat infections, stuffy nose, bad bite, crooked teeth, low energy, chronic snoring, and everything else.

The blank look, slight facial twitch, and total quiet of the orthodontist spoke everything in his mind about that horrible suggestion. His considered reply, confident and calm and practiced, was to explain that my mandible was too big, and when I was old enough I could have it broken surgically to bring it backward. He commented that this operation was horrible and full of risks; it sounded dreadful. It was obvious that extracting some premolars was the infinitely lesser of two evils. He said it would give me a perfect smile. He had dental models that explained the logic of it all. A lateral cephalometric radiograph explained his mathematics. He gave examples. He gave prices. He was a highly regarded dental specialist.

My father wanted another opinion, but my mother wanted to go ahead. In the end they said it was my decision, and my 13-year-old brain certainly didn't want my jaw broken, and I certainly didn't like my underbite or the look of my impacted canine, and I certainly didn't want to look like my flat-faced aunty either. So I decided it was best to remove my premolars. Two visits, two needles, and two teeth on each side. They put them in a jar for me to take home. My mother cried.

My orthodontic appliances were placed a month later, and when they were eventually taken off 2 years later when I was 15, it was not the pretty smile result my parents or I had expected. Even my schoolteachers expressed dismay at the cosmetic result. Four on the floor, braces in 24. All my school friends had had it too. We all had the same straight-teeth, flat-face look. And so too did every other school kid ydney train network. One treatment fitted all. The famous Sydney smile was everywhere. I hadn't escaped my aunty's fate.

The orthodontic retainer was very hard to wear. My teeth moved; they became crooked again. The positive overbite the orthodontist had struggled to gain by pulling my mandibular anterior teeth backward and maxillary anterior teeth forward gradually, relentlessly became edge-to-edge. Eventually I had a reverse bite again. My troublesome canine popped out of alignment. I saw my orthodontist every 6 months for follow-up, and every time he just told me to keep wearing my retainer. It was unbearable.

When I finished school and started university, I started experiencing jaw pain. After fabricating my own bite splint, which turned out to be useless, I visited my orthodontist, telling him that I was now a dental student. Essentially I was asking him as a mentor now to help me put all the random problems together, to help me fix them, to fix me. He smiled, said he was proud of me, said I must be stressed from all the study, and promptly referred me to an oral surgeon down the street to have my third molars removed.

When I turned up, the oral surgeon simply filled out a Sydney Hospital booking form. When I asked why I had to get my third molars removed, he told me that my parents had paid for braces and the orthodontist had asked, and of course because of the tooth crowding that came from not wearing my retainer. He wasn't interested in my jaw pain, saying it would clear up after the third molars were removed anyway. The fact that he was too busy and important to look at me for all the 5 minutes I was in his office made me start to doubt these people. All of them. The dentists who taught me. The dentists who treated me. The science that surrounded everything to do with how faces and bites developed and how they developed together. No one ever really explained anything to me, either as a patient or as a dental student. It was all a complete and illogical mystery to me. Why did everyone need braces for crowded teeth? Why did every kid I know have to have their premolars and third molars removed? Why did everyone need their tonsils out? Why did everyone have allergies, asthma, stress, and jaw pain? Here I was surrounded by books and people and institutions that should have been able to explain it all logically and coherently and scientifically, but they didn't. I kept wondering how I could have all of these unrelatable separate diseases affecting me? It was like one diagnosis per doctor. Damn was I unlucky.

I decided not to get my third molars removed, and they erupted normally (and I still have them). My jaw pain resolved by simply not chewing anything, my nasal

allergies cleared up when I moved out of my parent's home and into a series of new student houses, and coincidentally, I discovered a love for lap swimming, which also saw my allergic rhinitis, atopic eczema, and chronic asthma all miraculously clear up. It seemed all I had to do was escape Macquarie Street and my mum's insistence on the perfection of a Macquarie Street medical mind.

MY SECOND ORTHODONTIC EXPERIENCE

After moving to New Zealand and talking with many surgeons about what jaw surgery really was, I was still too afraid of it for myself. There was just so little known about it, and the jaw surgeons I worked with in training were mostly operating on syndromal kids and car accident victims. Besides, pushing my mandible backward simply to get a better bite seemed the opposite of what I needed, and my surgical mentors agreed. I was convinced it would surely choke me too, wouldn't it? No one seemed to agree with me on the potential breathing issues, but nonetheless I thought it was my maxilla that needed to be brought forward, which supported my mother's original, though very radical suggestion some 12 years previously. In the meantime I had orthodontic appliances put back on to see if I could achieve at least a stable bite and a smile I could live with.

In 1995 my new orthodontist was adamant that the science and predictability of maxillary surgery was still a long way off. The SARME (surgically assisted rapid maxillary expansion) operation was just getting a foothold in the United States, Professor Maurice Mommaerts in Belgium was still 5 years away from developing his bone-borne palatal expansion device, jaw distraction technology was just starting (and badly), routine jaw correction surgery was just beginning a radical renaissance via Bill Arnett in the United States, and custom titanium plates for midfacial surgery wouldn't be developed in France for another 20 years.

My new orthodontist was convinced he could "grow" my small maxilla with a slower treatment cycle and edgewise brackets. I decided to believe him, and I endured another 2 years of orthodontic appliances, which didn't manage to grow anything of course. Silly me. I still had a flat midface, a too-big mandible, and a weird smile. And I snored. But I did have straight front teeth in a barely normal positive overbite.

SELF-REFLECTION AND FRUSTRATION

What this repeat orthodontic experience did for me was point out that I could not explain my own face to myself. I could not form a rational argument with an orthodontist. I could not see how all of my component layers and the interrelated parts fitted together three dimensionally. I could not see how my teeth sat in my dental bone, or how the dental bone sat in my jawbones, or how my jawbones sat in my face. I could hardly dissect myself. I could not see where my symmetry or proportionality or bite issues began or ended. And if I couldn't see them or simply describe them, how could I direct myself to seek the treatment that I needed or understand or critically examine the orthodontic advice I was receiving? And how could I seek to describe anyone's bite or jaw problems or aim to surgically treat what was evidentially a complex interrelational set of anatomical issues involving many medical and dental and cosmetic themes?

In the 1980s and 1990s there was very little known about how anyone could dynamically see inside a person. There were radiographs of course, but these produced flat 2D views. What I wanted to know was how to construct a whole face, how teeth sat three dimensionally in the pattern of the midface, and how that related to all the structures inside it and outside of it.

Having maintained a schoolboy interest in optics and physics and mathematics, I wanted to build a stereoscopic device to create 3D radiographic images so that I could demonstrate to my teachers my ruminative concepts on volumetric facial radiology. Eventually I made a simple 3D radiographic model of a face. With it I could effectively demonstrate the acquisition and diagnostic simplicity of volumetric imaging. This thing seemed real. It shimmered just in front of the viewer and showed everything in perfect fidelity and accuracy; behind to front, side to side. It was very dramatic when I first saw it, and for everyone since that has seen it too.

After contacting a German engineering scientist who had written on a similar concept decades earlier, soon enough I had a couple of German employees of Siemens visit me in New Zealand to see my setup. I used my physical model as a visual means of explaining the mental image of the future of maxillofacial surgery that I had. I explained that digitizing a series of plain radiographs from a rotating x-ray machine, assigning numerical values to the grayness of the pixels in the image, and then cross-referencing the values to adjacent images obtained in a circle and traveling around

an object would enable a suitably powerful computer algorithm to build up the pixels (now voxels) into a 3D space. The Germans were developing this same idea for use in cardiothoracic and arterial imaging using the 1984 work of Feldkamp, Davis, and Kress. While I had no idea how to develop an algorithm to accurately 3D fix the gray value points, I was adamant that the technology, if developed, would be extremely useful in dentistry and for maxillofacial surgery in particular.

I was sure that a dentist could have a unit in the office no bigger than an orthopantomogram machine and three dimensionally image things as fine as a tooth's root canal system or see entire dental arches and bites.

For me, all I wanted was a simply acquired means of explaining that the face, teeth, jaws, and everything else were part of one complex 3D object. I wanted to be able to see into, expand, revolve, and better imagine facial growth disorders and the corrective jaw surgery steps needed to manage them. Once I had that, I could then describe the symmetry and proportions of the skeleton and dentition of a face. And then I could scan many people and compare them, and see patterns, and maybe move on from there. But I never heard from the German Siemens scientists again.

Many years later, I ran into my old boss, Leslie Snape, at a conference. He told me he still has my invention sitting on a bench in a closed room somewhere in the bowels of Christchurch Hospital. He calls it the original cone beam. I just laugh. I call it a Kiwi version of cone beam. If it can't be made with pantyhose and sheep-fencing wire, it's not worth calling it a practical invention. (That's a Kiwi joke.)

PUTTING IT ALL TOGETHER

Today of course, we have software applications much more sophisticated than cone beam that can accurately duplicate the entire head, segmentalize it, and separate the component parts. It's upon this digital version of the patient that we can replicate real-life surgery and the volume changes affecting tongues, airways, faces, temporomandibular joints, and bites. One of the best things about digital imagery is that you can start explaining complex things to your patients. It means that I can reduce a compound anatomical narrative to the common language of the visual medium. I still need a certain kind of intellectual ability on the part of my patients, but increasingly the patients who do independently find me are naturally skilled in broad research and innate logic.

Over the years my jaw surgery practice has naturally divided itself between two broad arms.

st is that I am providing some form of remedial surgical treatment, usually well after orthodontics has come and gone, and usually only in adults who are deliberately seeking my direct care. The majority of these people snore or have health or lifestyle issues related in some way to their ease of breathing. These people find me because they researched their personal symptoms, asked themselves logical questions, and sought a means to explain everything that has and is happening in their lives as one set of interrelated health issues. These people are generally free of an orthodontist referral.

In effect, I offer two ends of a stick. One is a simple end where I use a simple operation to prevent bigger problems, and the other is a complex end where I treat really big problems using really big operations.

The second arm usually involves young adolescents who are accompanied by parents, who have first brought their child to an orthodontist, usually for an overbite correction. These patients usually have an orthodontist's referral. They are usually the hardest to treat, firstly because they did not independently seek me, secondly because they require a complex and seemingly contrived explanation they do not really want to hear, and thirdly because parents naturally see jaw correction surgery as incredibly invasive.

The ironic thing is that IMDO (intermolar mandibular distraction osteogenesis) is the simplest surgical operation that I offer. It is even simpler than third molar removal, and it usually helps avoid the third molar surgery that is part and parcel of normal orthodontics for overbite correction (not to mention it helps avoid everything else too). But the greatest benefit of IMDO in this second practice arm is that it prevents these adolescent patients from becoming patients in the first practice arm—the adults who come to me for snoring or other problems that lead to jaw surgery remediation through remedial BIMAX (advancement of both jaws).

In effect, I offer two ends of a stick. One is a simple end where I use a simple operation to prevent bigger problems, and the other is a complex end where I treat really big problems using really big operations. Any medical enterprise has two ends like this. At one end are the treatments of the disease after it has occurred. At the other end is the research and the development and the application

of treatments that prevent that disease from occurring in the first place.

Why do parents who have children with small jaws and big overbites persist with a belief that orthodontics alone fixes everything? If someone asks me what the true cost is of treating someone with a small jaw, then that total cost must include tonsillectomies, dentistry, oral surgery, orthodontics, TMJ therapy, rhinoplasties, chin implants, sleep studies, CPAP (continuous positive airway pressure) therapy, and finally remedial jaw surgery. But I can only collate these costs if I tie all those things together as one linked or total series. So this question of whether adolescent dental crowding, or bad bites, or even small jaws has any other consequence apart from braces is obviously a very pertinent one. Is there really a link to adult obstructive sleep apnea (OSA)? Will the adult eventually insist on cosmetic intervention? Are impacted third molars inevitable or can they be prevented? Is there any way of correcting a bad bite before it starts? Is there any way to prevent snoring or OSA from ever developing?

There were a number of events that occurred in my life that set me on the intellectual and professional pathway that I now lead. At some point it occurred to me that a narrow dental arch was more a case of a narrow nasal airway. At some point it occurred to me that a small jaw and an obstructing tongue were part of the same condition. At some point it occurred to me that dental crowding and teeth in adolescents presaged the development of OSA in late adulthood. At some point it occurred to me that everything that I was taught in becoming a dentist was not the sum of everything I could know and that it could be built upon.

This book is in effect a chronicle of those ideas and their assimilation into a complex philosophy and then a practical set of new operations and new treatments. My six ways to design a face include IMDO, GenioPaully, custom BIMAX, SuperBIMAX, custom PEEK, and SARME. I am not the inventor of any of these things. The appearance of their originality is a silk screen, behind which lies an indescribably complex history and the serial and compounded efforts and stories of millions of people.

It is unimaginable to me that any person would willingly submit themselves to any of these operations, however simple or complex. Although I have always acted gently and hopefully painlessly, and with compassion and with precision, it is another level completely to trust themselves to be the first to an operation never before performed, let alone believed. So I'd like to thank the patients whose stories illustrate this book. If there is an inventor, it was the individual child who would suggest to me that their condition was curable.

If only I would become as imaginative as their own mind in describing their own condition, and a match to their inspirational hope.

1

REIMAGINING ORTHODONTICS AND ORAL SURGERY

History should never be ignored, but it always is. In every clinical discipline, fundamental premises or truths exist to drive the culture, practice, and scientific enterprise of that specific clinical group. History establishes how one thought or practice began and of course leads to another and the next. Through history we see why we do things a certain way today. It is in history that we see how our forebears interpreted the same observations we still see, and how their older philosophies of care underpin our modern clinical practices and wider community expectation.

Not all clinical disciplines are necessarily in agreement with each other. Chiropractors, osteopaths, and naturopaths undoubtedly disagree with physicians over many things. But no matter their underpinnings (respective of philosophical or scientific premise), almost all clinical groups were initially founded by a single person (or guru or saint). And yet we pretend that because we live in today, that it specifically equates to modernness or betterness, and what was in the past—which is by default old or ancient—has no part to play in how we think or practice today.

Our modern disciplines have evolved to be rooted only in new science, eternal debate, and collective polite argument. We can only accuse the other distant craft group of quackery. As proper scientific doctors we simply cannot believe that deception and magic and nonscience and esotericism can exist in ourselves. But is there really any true separation of modern practice from older practice? Are our modern treatments really so different to our founding fathers' so many centuries or decades or years ago? Are we really doing so much better? Consider Fig 1-1.

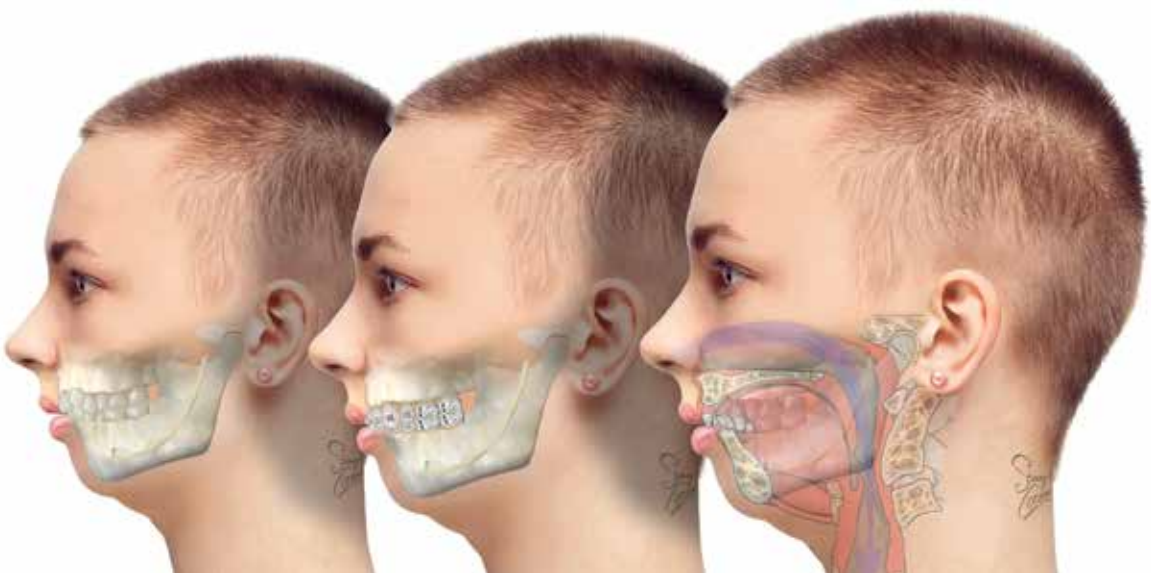

FIG 1-1 Traditional orthodontics alone with dental extractions and removal of impacted third molars will result in straight anterior teeth, but this century-old, well-established, and universally accepted treatment ignores the why and how of tooth crowding, as well as how the airways, face, tongue, jaws, and teeth are all indivisibly interrelated.

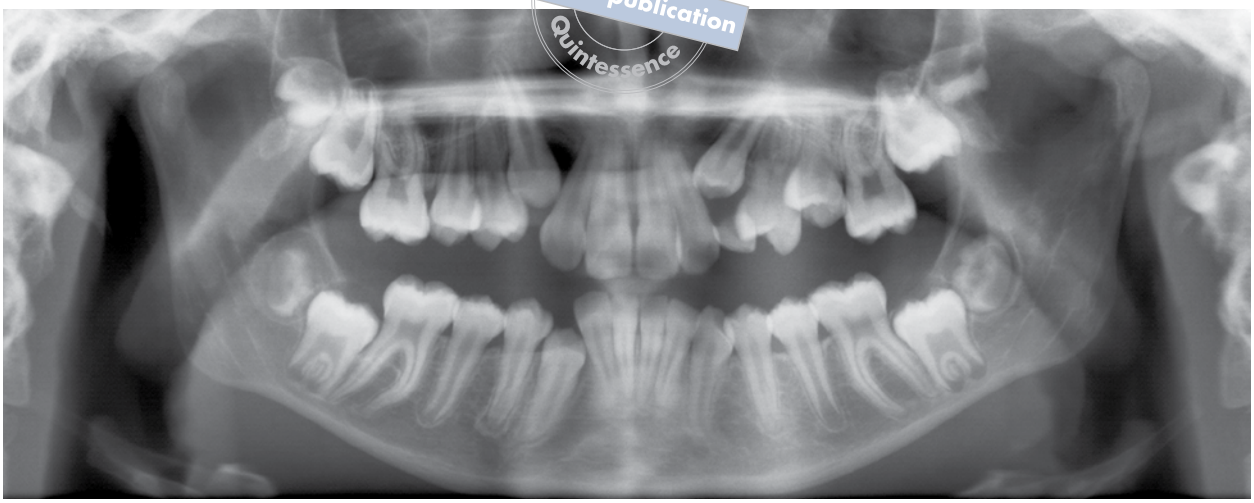

FIG 1-2 Severe dental crowding in a 12-year-old as seen on a panoramic radiograph. In a "normal" 12- to 13-year-old, without considering the third molars, the 28 permanent teeth should all erupt into a Class I occlusion, and there should be no retained primary teeth. Multiple impacted canines and second molars and impending impacted third molars can be orthodontically seen as a dental state of "late eruptive development" or of "premolar or third molar redundancy" or simply as "too many teeth." By contrast, the maxillofacial surgeon's view is to see that there is a normal developing complement of 32 adult teeth, there is a primary smallness of the jaws, there are codeveloping facial and airway problems, and all of it can only formally be evaluated by total-head 3D head analysis, or via volumetric medical CT imaging.

ORTHODONTIC DIAGNOSIS

Orthodontics was largely introduced by Edward Angle in 1895. He described the classification of "malocclusion"—a Latinization he invented to describe a "bad bite"—as Class I, Class II (division 1 or 2), or Class III. Orthodontists today still use the Angle classification of malocclusion to "diagnose" orthodontic cases, adding words such as "mild" or "severe" to further describe these classes followed by other descriptors such as "redundant" or "impacted" teeth or incisor relationship or jaw-profile association.

In the context of a purely dental clinical examination, a 12-year-old child with prominent maxillary anterior teeth may be described as a "severe Class II bilateral molar malocclusion, with significant incisor overjet, deep incisor overbite, and moderate mandibular incisor crowding, with impacted maxillary canines and impending third molar impactions" (see Fig 1-2).

Further dental analysis can be given with lateral cephalometry (Fig 1-3). The measurements of angles and proportions of certain points and positions of the facial bones and skull base began in the late 19th century, alongside the science of anthropometry, which eventually incorporated radiographic imaging in the early 20th century. The language of modern cephalometry is orthodontically oriented. It has its own culture bred from a hundred different voices with racially separated data sets identifying what is "normal" and "abnormal." It exists only for orthodontists and today is rarely used by jaw correction surgeons.

Dental orthopantomogram (OPG; Fig 1-2) analysis, also known as *panoramic radiography*, allows a better assessment of the teeth and mandible, but it is a highly distorted view. Even used together, panoramic radiography and lateral cephalometry are still poor descriptors of jaw and facial volumes and proportionality, and they are completely inadequate in assessing both airways and faces (Fig 1-4).

Yet the extrapolation from dental radiographic analysis to dental clinical pathway persists because it is formalized. Parents believe it to be true, and of course dental professionals believe it to be true. We all collectively and repeatably participate. The orthodontic waiting room is full of kids with braces and elastics. Our society is convinced that braces and dental extractions and orthodontics are all noninvasive and essentially benign and normal processes. But what we potentially ignore is the obvious fact that the 12-year-old sitting in front of us now will be an adult later on (Fig 1-5). And all of the dental analysis that is geared toward directing an adolescent orthodontic process, a process centered only on the negative esthetic of big anterior teeth, intrinsically ignores the biggest issue of them all—the ongoing lifetime effects, which started at birth and will end in old age, of what caused the bad bites and dental crowding and permanent tooth impactions to occur in the first place: the small mandible.

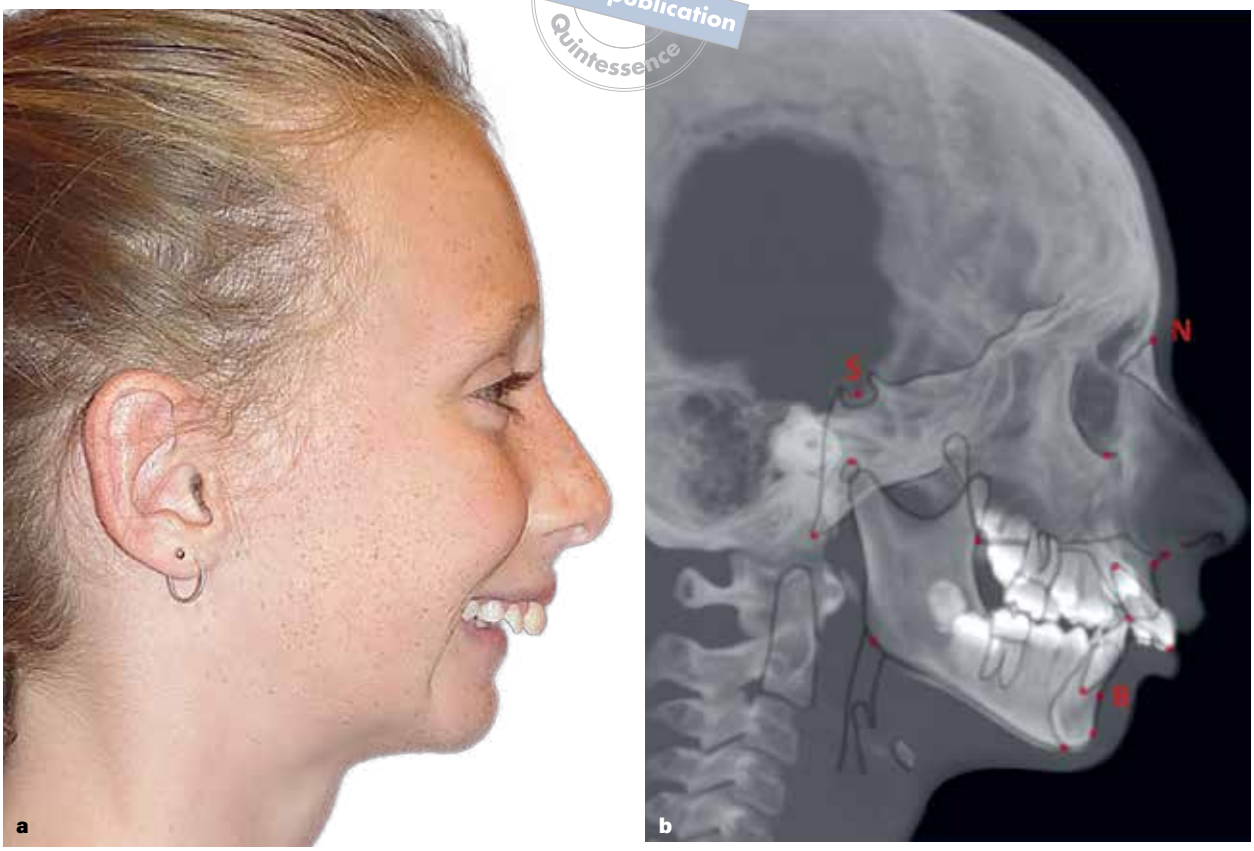

FIG 1-3 *(a and b)* An assessment of the profile of the same 12-year-old child with prominent maxillary anterior teeth may now include an orthodontic description of the jaw base. The cephalometric analysis evolves the orthodontic diagnosis as "severe Class II bilateral molar malocclusion, with significant incisor overjet, deep incisor overbite, and moderate mandibular incisor crowding, with impacted maxillary canines and impending second and third molar impactions, on a platform of anterior maxillary excess shown by excessive SNA angle and mild SNB angle deficiency." While the airway can be imagined on a lateral cephalogram, it is often poorly seen. Inherent postures such as neck uprightness and unnatural head position coupled with the awake state as well as transient states such as breath holding, swallowing, jaw posturing, general movement, and lip pursing, not to mention the general anxiety of the child entering the x-ray machine, all affect how the soft tissues tense and move as well as how reliably they can be interpreted or referenced. In my view, which is supported by meta-analysis,[1] the best measurement to determine whether a mandible truly lengthens due to an interventional orthodontic or surgical mandibular treatment is to compare the SNB angle on serial lateral cephalometry. Measuring changes in the SNB angle is relatively free of the confounder of growth and is not affected by genioplasty or GenioPaully. An accurate SNB measurement requires that the child does not posture the jaw forward—a notoriously difficult thing, as natural jaw joint position on lateral cephalometry is extremely difficult to confirm. The full treatment for this case is presented in chapter 9, along with our method of validating her surgical outcomes.

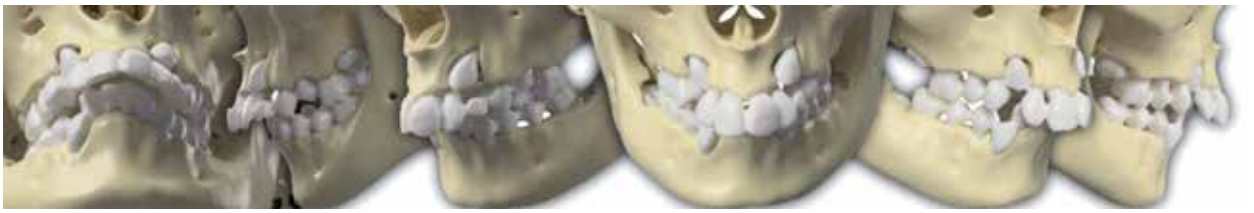

FIG 1-4 A better way than lateral cephalometry is to assess the interplay of teeth and bone as volumes in three dimensions. But this hard tissue 3D view does not see the soft tissues, and it does not visualize the airway or those anatomical structures that influence it. The competition of the dental perspective, which sees excessive and crowded teeth that together form a malocclusion, versus the medical perspective, which sees small jaw volumes and compromised airways and altered facial form, catches the parent between complex philosophical arguments. The crystal clear alternatives are divided as *(1)* traditional camouflage orthodontics based on dental extractions (including third molars) and *(2)* early interventional surgery through the intermolar mandibular distraction osteogenesis (IMDO) protocol. The winner or loser of that choice can only ever be the child and future adult.

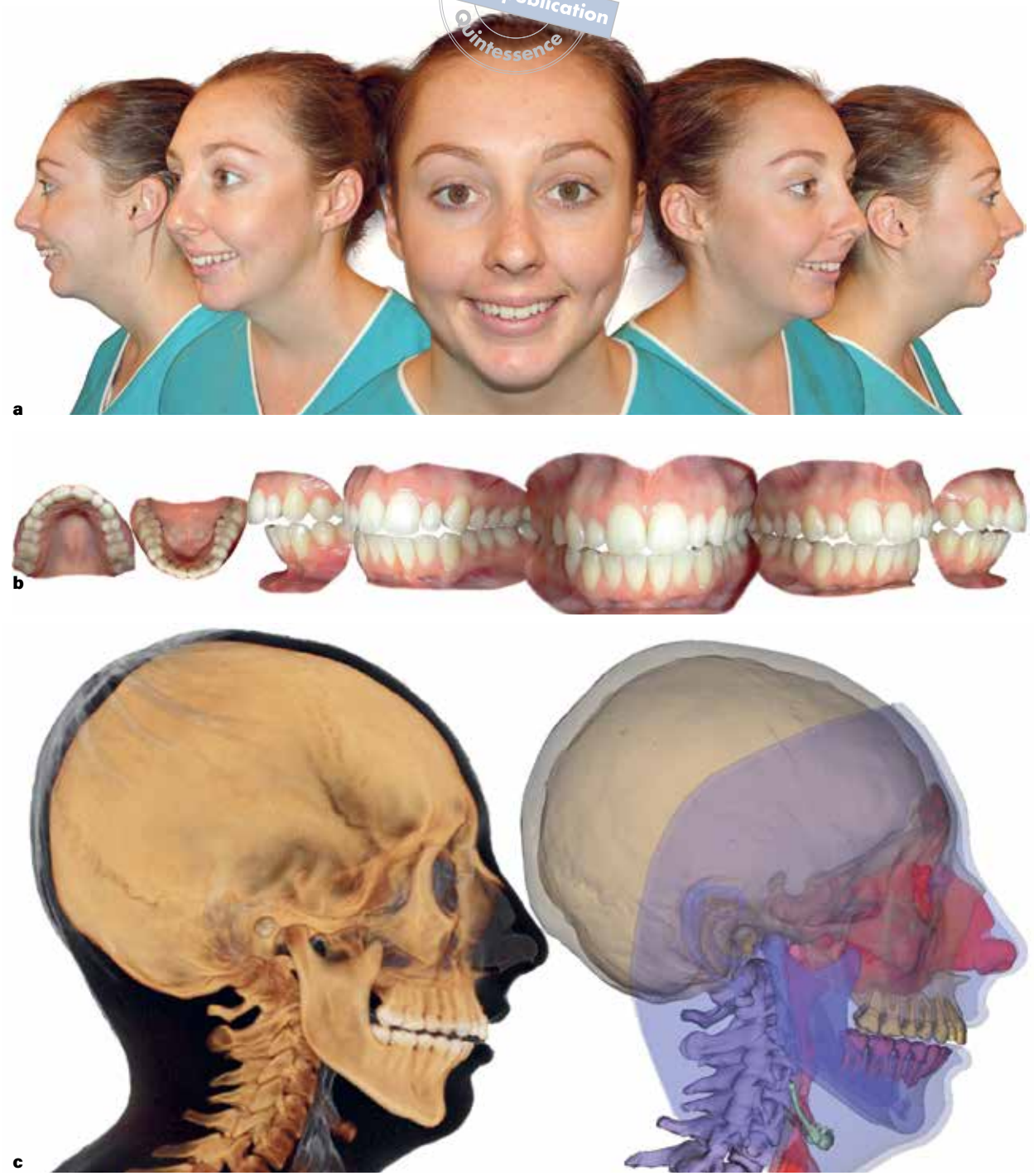

FIG 1-5 This patient had been treated with orthodontic bite splints and expert nonextraction camouflage orthodontics since the age of 12 years for a significant dental overjet and severe dental crowding. *(a)* At age 20 years, after having her third molars removed, she presented with complaints of significant breathing issues, general exercise intolerance, and a chronic forward head posture that she found impossible to correct by "standing up straight." While she had not yet developed full obstructive sleep apnea (OSA), she knew she snored, and she knew her small mandible had a big part to play in the history of her issues. For this patient, remedial jaw surgery coupled with repeat orthodontics offered a significant chance of permanent cure for her airway, bite, and posture problems. *(b)* With her jaw seated anatomically in its natural joint positions, her teeth only met at the back of her mouth, with a significant anterior open bite. The forward head posture and poor chin-neck contour are significant esthetic concerns. *(c)* Digitally derived lateral cephalometry with medical computed tomography (CT) or dental CBCT has significantly evolved from the traditional plain x-ray machine. Segmentation of color rendering allows for clear visualization of teeth, jaws, airway, and cervical posture. Such rendering not only improves diagnostic performance for the dentist but also helps the patient understand the complex interlinks of airway, bite, skeleton, and face. In this case, even with extreme forward head positioning, forward collapse of the cervical spine is still pushing into the back of the tongue, significantly reducing airway lumen and causing pronounced airway obstruction during sleep and during erect exercise.

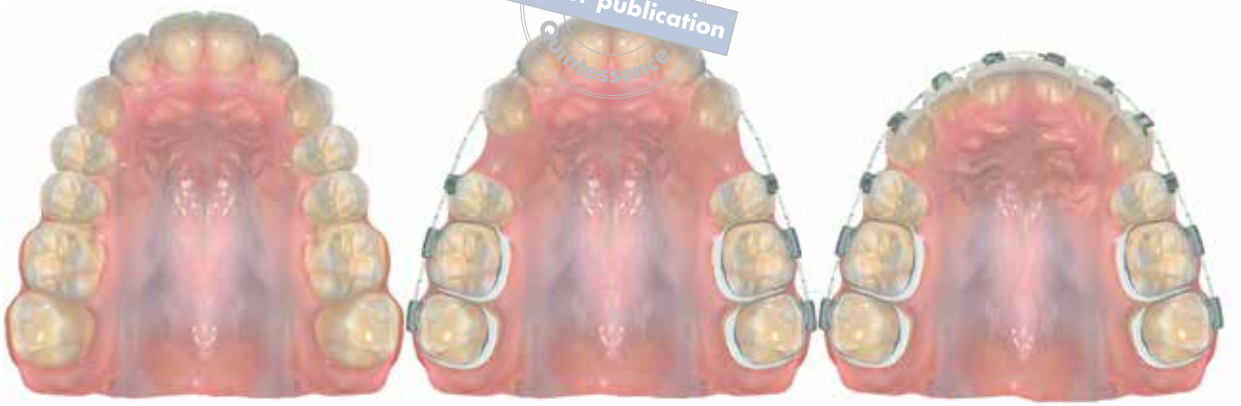

FIG 1-6 Extracting premolars to create dental space for orthodontic decrowding, or retraction of prominent anterior teeth, is the basis for all camouflage orthodontics used for the treatment of Class II, division 1 malocculsion.

DENTAL EXTRACTIONS AND TRADITIONAL ORTHODONTICS

Almost all humans have the genetic potential to develop and keep 32 adult teeth. But almost all adolescents have dental crowding as their adult dentition erupts, giving the visual illusion that there are an excessive number of adult teeth. And almost all adolescents with dental crowding will develop impactions of the third molars; this near-universal impaction in people with crowded teeth gives an illusion that third molars are redundant or evolutionarily unnecessary to modern human existence. Because we have people in modern society whose craft it is to treat this human commonality of dental crowding, if a child with crowded teeth has a mother wanting to treat that crowding or crookedness with classical orthodontics, then braces combined with dental extractions of any of those 32 adult teeth through oral surgery is inevitable.

Crowded or overly prominent anterior teeth, or any bad bite (whether Class I, II, or III) for that matter, usually results in premolar extractions (Fig 1-6). While this is considered "minor oral surgery" because it is performed in-office by the dentist, it is by no means "minor" to the individual losing these teeth. Combine this with third molar removal, and a given orthodontic patient is losing a minimum of six to eight permanent teeth. Even if "nonextraction" treatment is selected in clinical orthodontics (Fig 1-7), third molar removal is still almost universally required. So if an adolescent has dental crowding, or a malocclusion, or dental impactions, or all of these together, then dental extractions carry an almost 100% certainty in late adolescence or early adulthood.

From a total of 32 teeth, and following classical orthodontic treatment for dental crowding, the end result will almost never be 32 teeth. The end result will be 28, 26, or 24 teeth. If you lose four premolars and four third molars, these lost eight "back" teeth represent a certain loss of occlusal table and dental volume, not to mention upward of ~33% loss in total dental mass (Fig 1-8). These lost teeth will also not develop associated alveolar bone or gingiva to support them, and they will not support the face that surrounds them.

Non-jaw surgeons may empiricize that corrective jaw surgery is invasive. But what equal professional rationalism exists to explain or accept that permanently removing one-third of a child's dental mass through a combination of classical orthodontics and traditional oral surgery is not considered invasive? In my view, traditional orthodontics inherently relies upon invasiveness and is quite the opposite of conservative.

Nevertheless, this competition between ideas of conservatism, easiness, or invasiveness only considers the teeth. Are there other things we are ignoring by insisting on focusing and diminishing our diagnosis or treatments to the teeth alone? In demonizing the other professional, it's easy to ignore what we do not know or see or talk about, or worse, what we refuse to acknowledge.

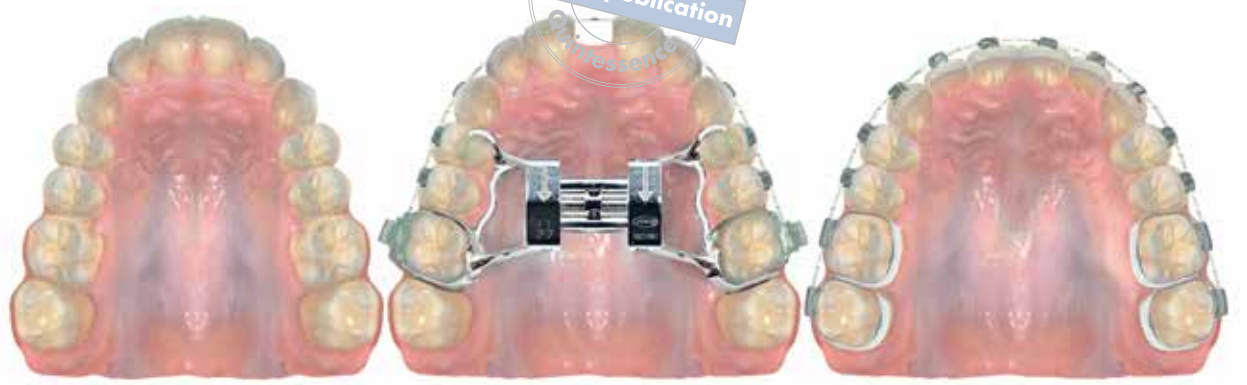

FIG 1-7A For Class II, division 1 malocculusion, not extracting premolars requires expansion and then orthodontic retraction of prominent anterior teeth as the created midline gap is closed. This backward retraction further potentiates the chance of maxillary third molar impactions. How this combination or pattern of orthodontics ultimately leads to other facial and airway effects is described in chapter 12.

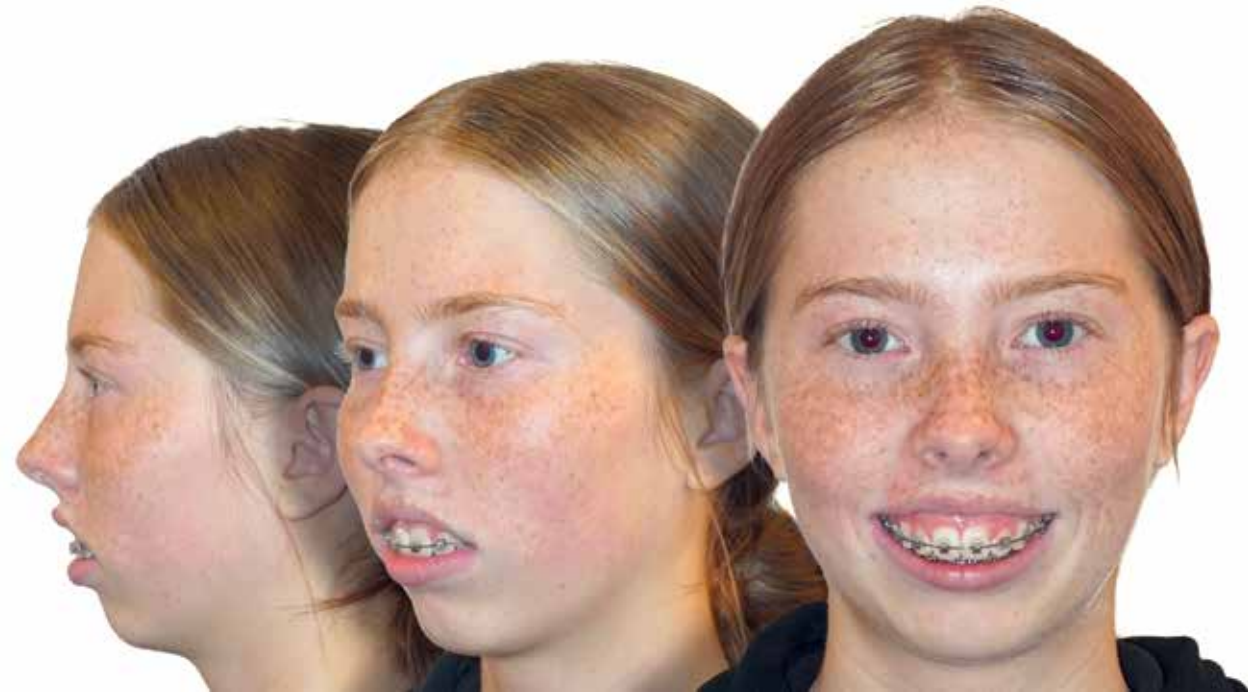

FIG 1-7B The process of expansion and retraction of prominent anterior teeth produces an elongation and vertically excessive "gumminess" to the anterior teeth and accentuates the original lip incompetence associated with the small mandible. This pattern of camouflage orthodontics is a leading cosmetic drive and preamble to later remedial BIMAX surgery. This patient's treatment is explained in Fig 15-14.

NONDENTAL CONSEQUENCES OF TRADITIONAL LEAD-ORTHODONTICS

Lead-orthodontics is what I call treatment where orthodontics comes first. The orthodontist is the first to assess the bad bite and the dental crowding. Everything that follows is as a consequence of the orthodontic event that preceded it, or of the orthodontic assessment, or of the orthodontic diagnosis. The entire conversation revolves around the orthodontic management of teeth (Figs 1-9 and 1-10).

As a maxillofacial surgeon, I clinically see three broad age groups of people. The first group are adolescents (10–21 years old) with a full complement of crowded adult teeth and a bad bite looking to keep everything and in a perfect face, forever. The second group are adults (22–49 years old) unhappy with the gummy smiles and cosmetic facial disproportions they attribute to camouflage orthodontics. And finally there is the middle-aged group (50+ years) with a history of orthodontics and dental extractions who now snore and have thick necks and uncontrollable

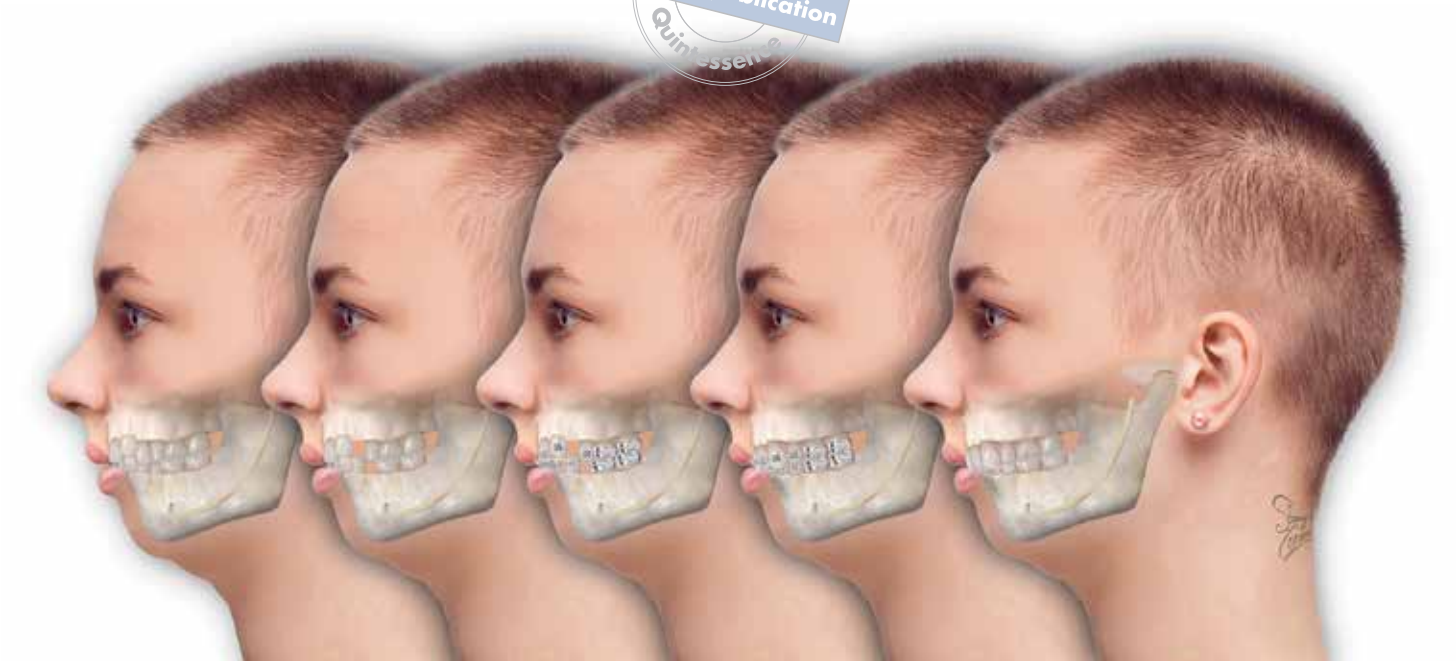

FIG 1-8 Extraction-based orthodontics of Class I dental crowding in adolescence. Four premolar extractions are a staple of almost all orthodontic practice. The third molar impactions that almost inevitably co-arise will eventually lead to a consideration of their extraction, premised on an idea of them being redundant or unnecessary. But removing eight teeth leads to a ~33% reduction in dental mass. In assessing only the bite, there is no assessment of the face or future adult airway. Forward planning toward how the adult face will look—after the orthodontics is completed—is entirely ignored.

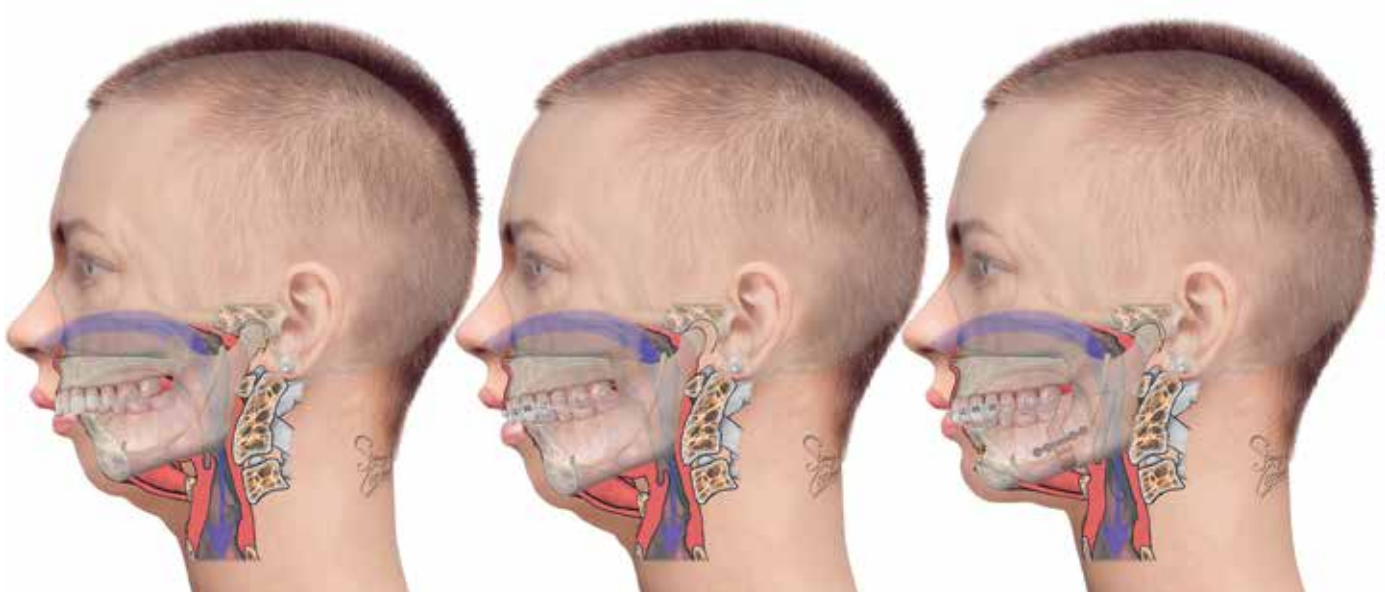

FIG 1-9 In treating without orthodontic extractions, traditional lead-orthodontics for Class II, division 1 malocclusion aims to attempt some retraction of the maxillary anterior teeth. A vertical gumminess results, with parted lips, accentuated by the lack of chin often associated with anterior mandibular hypoplasia (AMHypo). Orthodontic retraction (Class II) elastics also train the patient to hold the mandible forward while awake. After the impacted third molars are removed, the lip incompetence and chronic forward jaw posturing can be relieved with a small advancement bilateral sagittal split osteotomy (BSSO) into a Class I bite, with upward sliding genioplasty to improve lip seal. This basic form of jaw correction surgery is always and only done under the primary direction and recommendation of the treating orthodontist, and almost always with current orthodontics in place in order to help "settle the bite." Because the geniohyoid is not greatly stretched or advanced by either the sliding genioplasty or the small BSSO, there is only a small advancement of the airway—certainly not to a degree that would permanently overcome the total effect of glossoptosis or future risk of developing OSA.

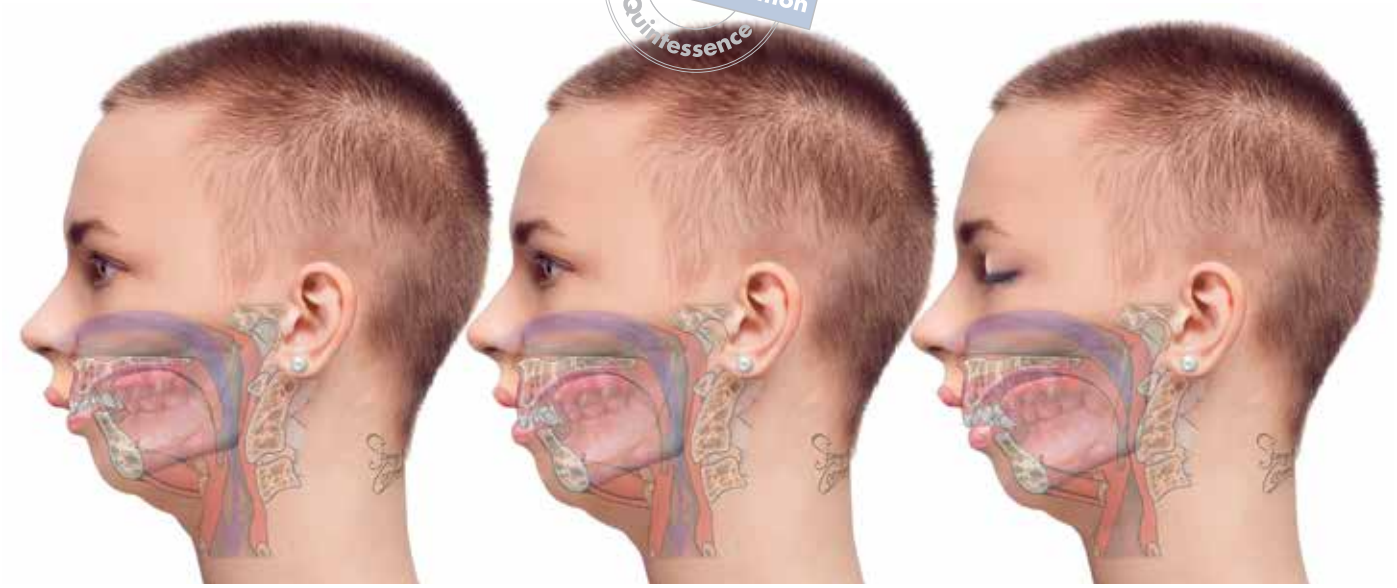

FIG 1-10 By their constraining effect and pull-back on the maxilla, all jaw splints, including MyoBrace, TwinBlock, Herbst, and Frankel, will partially "correct" the maxillary anterior tooth prominence of Class II malocclusion, but this effect is entirely by restricting growth of the maxilla, by pulling the maxillary anterior teeth backward and downward, and by positioning the mandible forward during waking hours. As a Cochrane review demonstrates,[1] there is no proof that jaw splints will grow a child's small mandible or act any differently to any other form of camouflage orthodontics. MyoBrace offers no scientific support for their marketing and therapeutic claims that they "grow the small mandible." At night, the supine child, adolescent, or adult will fall asleep, and as conscious tone is lost, the tongue and jaw will completely relax backward to obstruct the airway, thus reversing any daytime influence of such presumed tongue training and awake forward jaw posturing. The eventual effect of all camouflage nonextraction orthodontic therapies for Class II is therefore the same—to shrink the maxilla, produce a gummy smile, and help train the adolescent to hold the mandible forward, but only when awake.

For every person with a bad bite, there are three combined, interwoven, inseparable treatment considerations: occlusion, airway, face.

weight gain. In reality these three groups are really the same people, with the same anatomical conditions, just at different stages of their lives.

This book explains how each of these groups can be managed with one overlying simple clinical philosophy, and that is to include a surgical treatment from the start. The jaw surgeon has never *not* been part of the equation of the treatment of bad bites. Likewise, it is not possible to ignore or delete orthodontics from that equation either. But the relationship between the two sides must be reinterpreted to correctly solve it. As I will come to elaborate in this book, orthodontics and oral surgery go hand in hand. They developed together from the start. Whether I operate on jaws to remove third molars or operate on jaws to fundamentally fix jaw size, the operations are fundamentally the same, and they are performed almost the same way, and with a similar collaborative orthodontic effort. All that changes is primacy.

Maxillofacial surgeons are the absolute and only experts in facial disease and of facial abnormality and skeletal facial issues, all of which are associated with bad bites, crowded teeth, airways, and of course the face. The maxillofacial surgeon still needs orthodontics, but the relationship has changed. The roles have swapped.

For every person with a bad bite, there are three combined, interwoven, inseparable treatment considerations: occlusion, airway, face.

OUR ILLUSTRATIONS

In order to properly compare all the treatment types and ideas of anatomical derangement that lead to malocclusion, and in order to make meaningful assessments of the effects of treatments on facial proportions and airways and across ages, this book uses a single model—an imaginary female as she transitions through life. In the 7th century Pythagoras described six ages of man: infancy (0–6 years),

FIG 1-11 Seeing a person through the ages. In this case at 24 months, 17 years, and 65 years, respectively. Proportionally, the small mandible is eternal. Understanding why small mandibles are so common, and their link to dental crowding, bad bites, and airway obstruction, is a primary subject of this book.

adolescence (7–21 years), adulthood (22–49 years), middle age (50–62 years), old age (63–79 years), and advanced age (80+ years). This book illustrates the model at three of these ages (Fig 1-11): infancy (around age 2 years), adolescence (around age 17 years), and old age (around age 65 years). Throughout the book you will see only her relaxed facial profile with teeth together, both awake and asleep.

This model was built by merging data from real CT scans, precise anatomical segmentations of hard and soft tissues, and a lifetime of study involving many thousands of people to determine the interlinks between facial profile, airway, jaws, and occlusion. Peppered among these illustrations are real examples of actual patients and procedures that best illustrate my concepts that underlie both facial surgical and orthodontic diagnosis, as well as the comparative therapeutic and cosmetic benefits of this treatment or that.

For instance, the illustrations explaining the mechanism of thumb sucking were directly derived from the volumetric scans of a real infant who had developed anterior open bite. The images were donated by a Sydney-based radiology house who had examined an intubated child in a Sydney-based pediatric-care ICU. He had developed life-threatening suppurant pansinusitis following advice to his mother to actively cease his thumb sucking habit.

CT scans of infants are profoundly rare. But with this single image set came our first proof of the importance of nasal breathing to developing a normal healthy state of the nasal sinuses. With it too came an understanding of the developmental compromise committed upon the maxilla and sinuses by the small mandible and glossoptosis, as well as the contribution that normal nasal breathing gave to normal midfacial development. It also pointed out to us that dental advice that demonizes thumb sucking in order to prevent the development of anterior open bite was harmful in that it deleted the "why" the child was thumb sucking in the first place.

Another illustration base focused on a single midadolescent female and the effect that IMDO had upon correcting her Class II malocclusion and in normalizing her small mandible. It was through this patient that we precisely studied the effects of tongue muscle pull, particularly the geniohyoid muscle, and its relief upon glossoptosis. These CT scans were painstakingly rendered in 3D to accurately demonstrate the multiple dental, facial, and airway issues associated with anterior mandibular hypoplasia (AMHypo).

There is very little that differentiates one human from another. The tiny genetic differences that distinguish a Finn from a native Peruvian would account for less than 0.0001% of the entire human genome. Yet it is in the expression

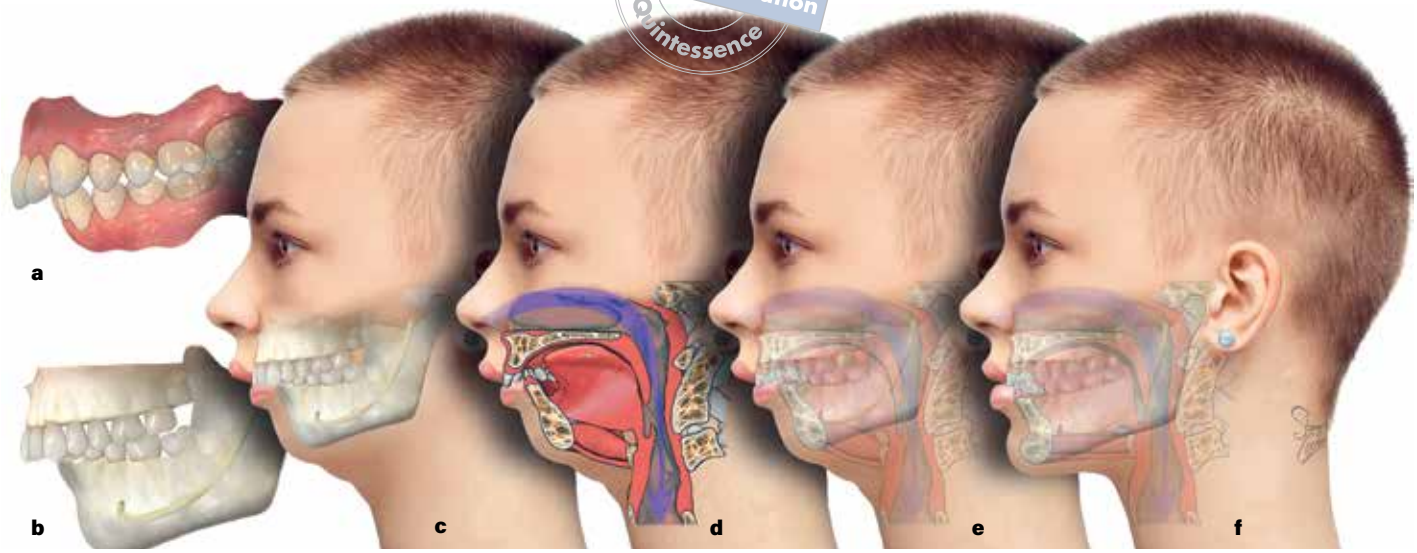

FIG 1-12 How various anatomical layers are interpreted. *(a)* View of how a dentist, parent, or orthodontist may see the relationship of maxillary and mandibular teeth in the mouth, or within an intraoral photograph. The patient may hold the mandible forward, altering the true bite relationship, but this visual oral examination is blind to jaw joint position. *(b)* A radiograph such as an OPG or even a lateral cephalogram gives little obvious further elaboration upon the visual beyond an examination of tooth roots, or at best the mandibular outline. There is no true elaboration on jaw joints, neutral jaw joint seating, or the overlying face itself. *(c)* The skeletal, dental, and facial relationships can be married, but this view still ignores the internal. *(d)* Cross-sectional views help understand toned (awake) tongue muscle contraction and airway patency. *(e)* Fusion of all pretreatment elements allows us to see the combined awake and erect relationships of the tongue, jaws, teeth, occlusion, airway, jaw joints, and facial effects that combine as a result of the small mandible. *(f)* The directed surgical treatment of part *c* to fundamentally correct the mandibular volume demonstrates a comparative means of seeing how dental impactions, malocclusion, airway collapse, tongue contraction, and facial profile may change toward an ideal anatomical state.

of this tiny genetic difference that we examine all that is different between ourselves, or groups of ourselves. In studying faces and jaws and bites, what we are looking for is a common fundamental link that gives the human commonality for all the various patterns of dental crowding and the connected link to inner airways and overlying facial form. And it is through these same illustrations that we can examine the effects of bespoke facial design that augments the therapeutic facial surgical intervention—six ways.

ORTHODONTIC CLASSIFICATIONS OF MALOCCLUSION

Malocclusion is a dental term, and it relates to the teeth alone. The Angle classification was invented before we had radiographs or CT scans or even a modern understanding of functional facial anatomy. As such, the Angle classification of malocclusion is outdated and, frankly, not useful to a description of orthognathics. More importantly, this classification is exclusionary in that it does not define why the condition of malocclusion exists. By only looking at teeth, the Angle classification inherently excludes any understanding of the volume effects of jaws, particularly their effect on facial profiles, the postural effects on the tongue, and the overall lifetime effects of the compromised inner airway.

In my worldview, there are six methods for looking at malocclusion in profile (Fig 1-12). First, you can look at the teeth and gingiva and their intra-arch relationship. Second, you can look at how the malocclusion state relates to the underlying local dental skeleton. Third, you can look at how the dental skeleton and bite relate to the profile of the face. Fourth, you can look at how the whole facial skeleton relates to the inner airway. Fifth, you can look at the way the occlusion, skeleton, face, and airway combine simultaneously. And finally, you can look at the way that a given therapy affects all four anatomical states in terms of dynamic posture (asleep vs awake, smiling vs relaxed, supine vs erect).

Considering the different orthodontic classifications for malocclusion, this chapter presents 1:1 illustrations (see Figs 1-13 to 1-16) to demonstrate that all forms of common malocclusion are derived from the same basic common smallness of the mandible, or what I call *anterior mandibular hypoplasia* (AMHypo). Illustrations are made with a chin button (normogenia) or without a chin button (agenia),

but note that almost all people with AMHypo also have some form of agenia. You will notice that those who have a chin button have a subtly greater pull on the geniohyoid muscle, with slightly greater patency of the airway behind the epiglottis (the so-called C3ERPO point, or 3rd **C**ervical, upper **E**piglottic, **R**etropharyngeal **O**bstruction point), and slightly more defined chin-neck contour. This may indicate a view that the natural chin button has an evolutionary function in stretching the geniohyoid to help overcome glossoptosis, but this is not my opinion. I honestly do not know why some humans have chin buttons, except that they look good. My therapeutic opinion is that the surgical GenioPaully, which creates a chin button, does have these therapeutic airway effects, as well as a primary effect on the lower lip posture, lip competency, and general esthetic lines that are drawn.

> Which profile the patient displays, and which treatment philosophy you choose, will define everything that will follow. The only fundamental differences are whether there is agenia or not, AMHypo or not, and maxillary hypoplasia or not.

All illustrations have a full complement of 32 teeth—in various patterns of eruption or crowding or alignment or development or impaction. In each series of this set of four, part *a* shows a normal mandible, with or without a chin button. Parts *b* to *f* show AMHypo with and without agenia, respectively. In *b* to *f*, the small mandible proportions are exactly the same between each illustration, and only the pattern of dental crowding is different. All have impacted third molar development as well.

Parts *c* to *f* show AMHypo as well as relative smallness of the maxilla, known as *maxillary hypoplasia*, which is always associated with additional nasal airway issues further complicated by the small mandible.

Which profile the patient displays, and which treatment philosophy you choose, will define everything that will follow. The only fundamental differences are whether there is agenia or not, AMHypo or not, and maxillary hypoplasia or not. The decision to treat the malocclusion class is not based on numbers contrived through lateral cephalometry. It is based on rationalizing that 32 teeth are normal and must volumetrically assess the ideal positioning of these 32 teeth, a normally spaced airway, and a normally proportioned profile, all within patient-specific facial volumetric needs to accommodate all internal anatomy.

The questions then become: How do I fit everything in? How do I keep everything? What do I need to make bigger? What steps do I need to take to move from small to normal? How do I make this specific individual with all her myriad issues "normal"? And how do I reclassify everything to see the pattern of it all and at once?

A NEW ORTHOGNATHIC CLASSIFICATION

In order to merge all the different themes of treatment, I propose that there is a need for a different way to orthognathically classify the average profile of a face. By *average*, I mean 98% of us, excluding the 2% that fall into a battle of an infinite variety of subtle or imagined abnormality. This classification therefore is of facially normal variance based on the almost absolute commonality of AMHypo. Ours is not a discussion on the pathologic variances of a thousand other vague and rare conditions. Of course not every single person in every single instance will fall easily into this subclass of this or that, but the overwhelming majority will.

In my assessment, the current means of orthodontic malocclusion classification ignores everything outside of the bite anyway, including the "why" of how a particular and common malocclusion occurs. And if we don't know the common "why," then we will never know how to fix almost everything.

> AMHypo is so ubiquitous, and so common, that we see it as normal; and because it is normal, we don't see it at all.

In my classification (Figs 1-13 to 1-16), I still use the principles of Class I, II (including divisions 1 and 2), and III malocclusions that Angle first defined, but I also give an added viewpoint of how anterior open bite develops. In my mind, these abnormal states of facial profile and bad bite fall on a spectrum (*b* through *f*), but they all have exactly the same small mandible. The base condition is always AMHypo. In my mind, AMHypo is so ubiquitous, and so common, that we see it as normal; and because it is normal, we don't see it at all.

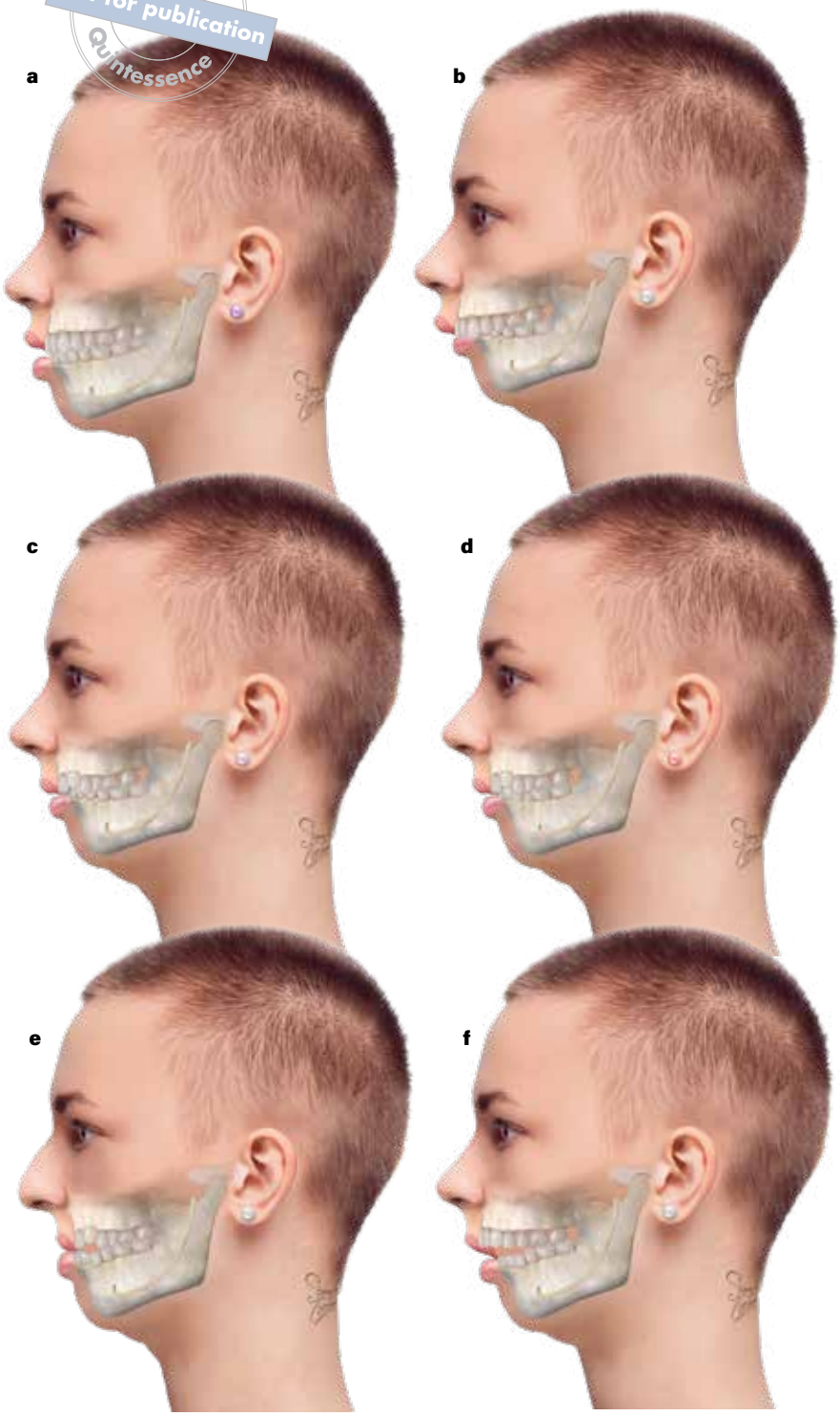

FIG 1-13 Orthognathic malocclusion series 1: Agenia, with and without AMHypo, with and without maxillary hypoplasia, looking only at the teeth and facial profile and jaw size. *(a)* Normally proportioned mandible and maxilla without a chin button (agenia). There is a full complement of 32 teeth in full natural occlusion. Without a natural chin button, the lips are slightly parted, and the molar and incisor occlusion is an Angle Class I without dental crowding. With the lips slightly parted, the anterior teeth are prominently seen, and the appearance is of anterior dental fullness. Orthodontically, this would be called maxillomandibular protrusion with Class I occlusion without crowding. *(b)* AMHypo with agenia with the maxillary arch drawn normally. The appearance is of prominent upper lip protrusion and prominent maxillary tooth display. There is significant dental overjet and deep incisor overbite. The mandibular teeth are not crowded, and the first molars are in a Class II relationship. Orthodontically this is classified as Angle Class II, division 1 malocclusion. Division 1 implies that the maxillary incisors are well forward of the mandibular incisors. *(c)* AMHypo with agenia with the collapse of the maxillary anterior teeth, creating crowding or retrusion of the maxillary incisors, which deepens the dental overbite but reduces the dental overjet. There is less mandibular dental crowding, but there are impacting third molars. The molar relationship is Class II. The orthodontic classification here is Angle Class II, division 2 malocclusion. *(d)* AMHypo with agenia leading to mandibular crowding. There is also maxillary crowding and impacted maxillary canines due to a secondary small maxilla. The molars are in a Class I relationship. This would be called Angle Class I malocclusion with severe dental crowding. *(e)* Severely small maxilla with extreme dental crowding. This is caused by a lack of pneumatization of the maxillary sinuses and is an extension of the state shown in *d*. Both are caused by open mouth breathing due to the inherent AMHypo. The extreme smallness of the maxilla means the maxillary anterior teeth lie in line with or behind the mandibular anterior teeth, and there is a negative Class III molar relationship. This "malocclusion" gives the illusion that the mandible is too big. The orthodontic classification is Class III malocclusion with severe dental crowding. *(f)* Here the smallness of the maxilla and the dental crowding are associated with the anterior teeth not meeting at all. This is called *anterior open bite*, and its genesis pathophysiologically is related to the inherent smallness of the mandible—AMHypo—and is almost always associated with an infantile habit of airway-compensating thumb sucking (see Fig 1-17).

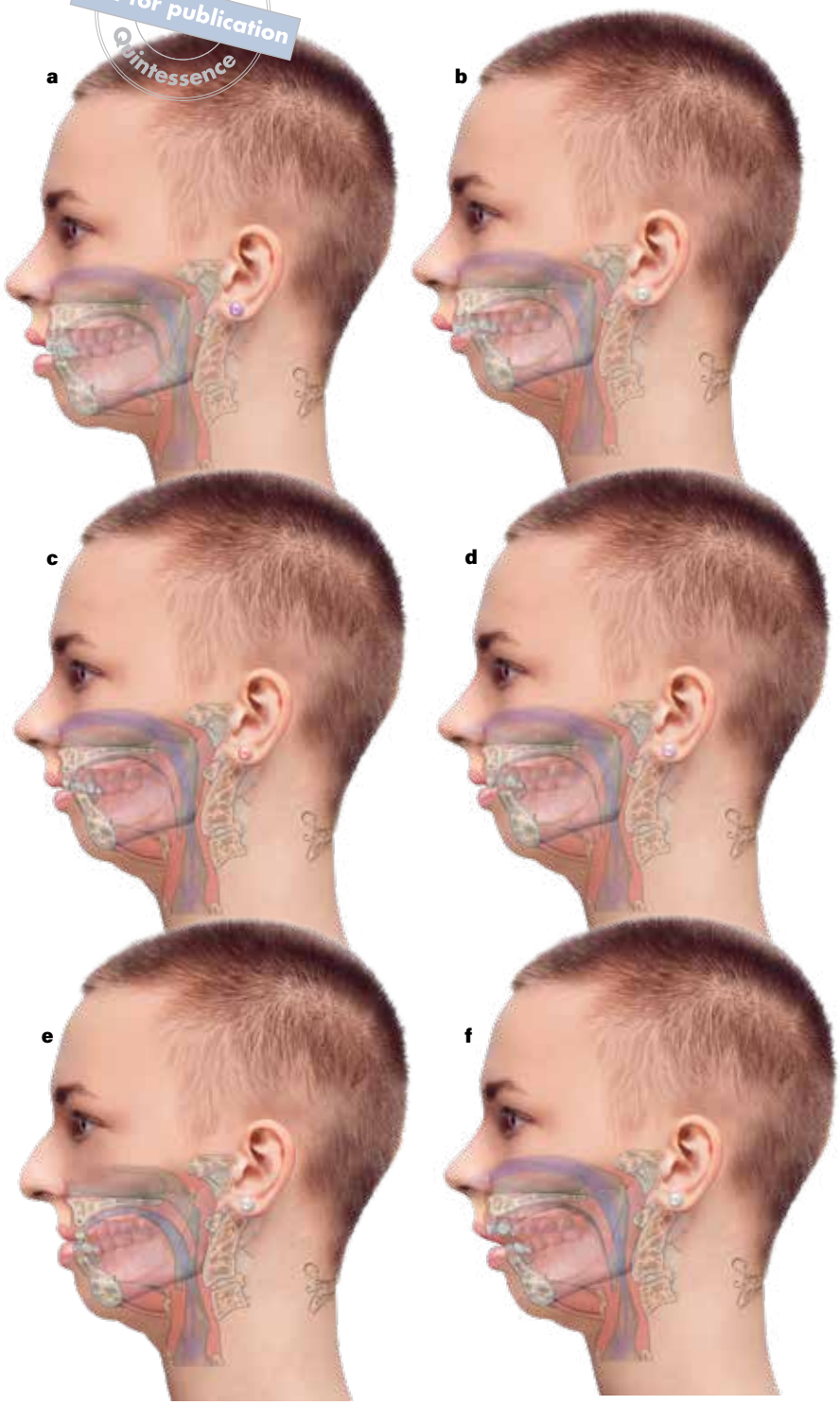

FIG 1-14 Orthognathic malocclusion series 2: Agenia, with and without AMHypo, with and without maxillary hypoplasia, looking at the teeth, tongue, airway, facial profile, and jaw size. By saying that 98% of all malocclusion is caused by AMHypo, our new orthognathic classification can rationalize simultaneously the airway and facial profile effects, in addition to malocclusion of dental crowding patterns. The only additional considerations are therefore *(1)* Is there a chin? *(2)* Is there an effect on maxillary development? *(3)* Is the condylar anatomy normal? *(a)* Normally proportioned mandible and maxilla without a chin button (agenia). *(b)* AMHypo with agenia with the maxillary arch drawn normally. *(c)* AMHypo with agenia with the collapse of the maxillary anterior teeth. *(d)* AMHypo with agenia leading to mandibular crowding, in addition to a secondary small maxilla leading to maxillary crowding and impacted maxillary canines. *(e)* Severely small maxilla with extreme dental crowding. Because there is chronic nasal blockage, innate glossoptosis, and agenia (short geniohyoid distance), this state poses the greatest lifetime anatomical risk of OSA. *(f)* Here the smallness of the anterior maxilla and the dental crowding on a primary base of AMHypo and agenia are associated with the anterior teeth not meeting at all.

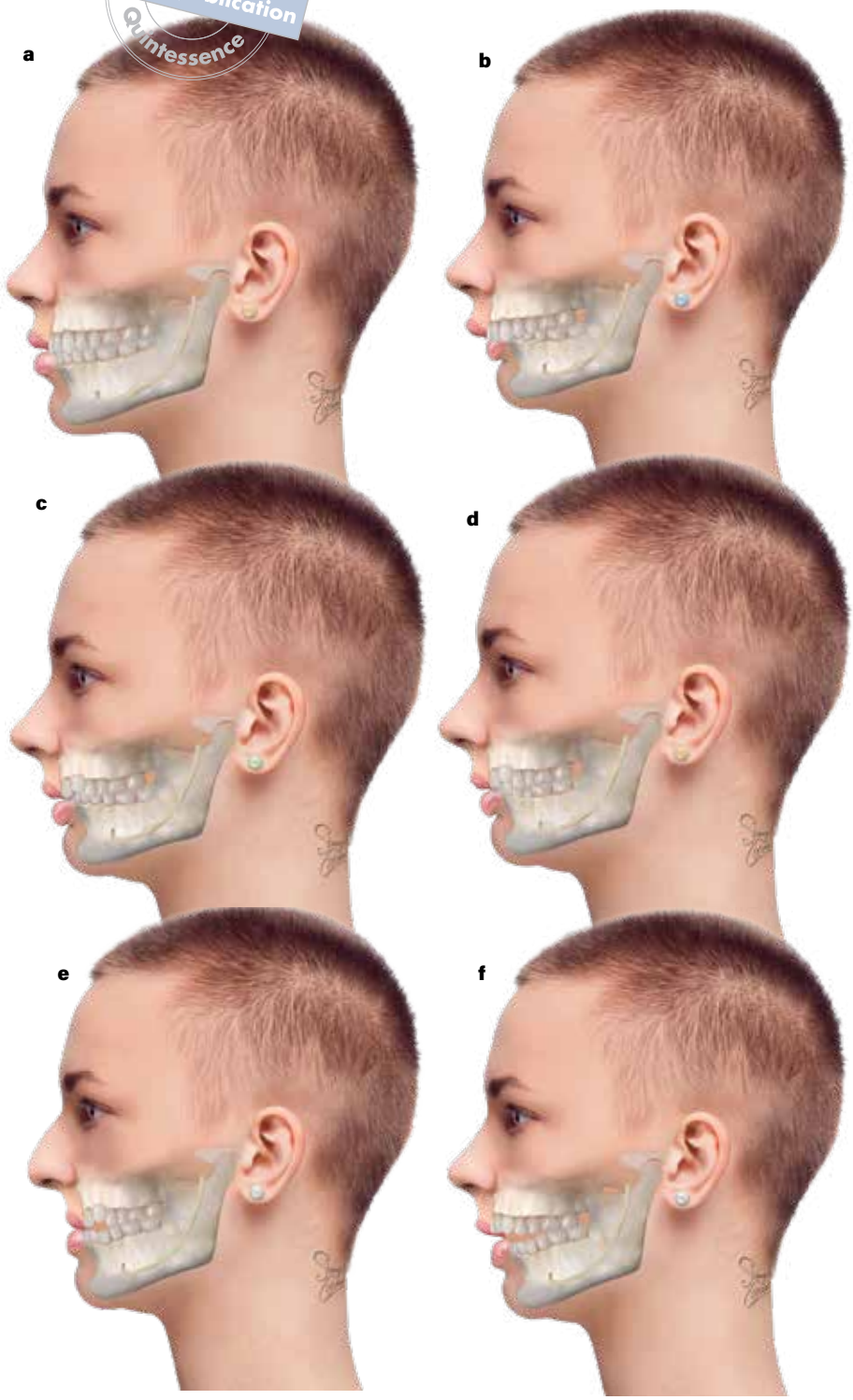

FIG 1-15 Orthognathic malocclusion series 3: Normogenia looking only at the teeth and facial profile and jaw size. *(a)* Normally proportioned mandible and maxilla with a chin button (normogenia). With a chin button, the lips are closed at rest, and a proportion of the maxillary front teeth are esthetically seen below the line of the relaxed upper lip. The appearance is of a full and complete smile. Orthodontically this would be called Class I normal occlusion without crowding. With a more defined jawline, this is still a very feminine profile but less neotenic (young looking). The geniohyoid is at full stretch, and there is good chin-neck contour. I call this "the California look." *(b)* AMHypo with normogenia with the maxillary arch drawn normally. The outward curl of the lips and the slight prominence of the maxillary anterior teeth normalizes nasal projection and nasal-lip balance, and the slight retrusion of the normal chin button is considered in combination very feminine and neotenic. I call this "the soft French look." *(c)* AMHypo with normogenia with the collapse of the maxillary anterior teeth. There is relative collapse of the upper lip relative to the base of the nose, giving a sense of overforward nasal size or projection (a common cause to seek cosmetic nasal tip reduction). *(d)* AMHypo with normogenia leading to mandibular crowding, in addition to maxillary crowding and impacted maxillary canines. There is a prominent lower lip curl because of the collapse of the upper lip. *(e)* AMHypo with normogenia with a severely small maxilla with extreme dental crowding, leading to buccal crossbite and a high arched palate. The illusion is that the mandible is too big, which is further accentuated by what is essentially still a normal chin button. I call this the "wicked witch of the west" look. *(f)* Here the smallness of the anterior maxilla and the dental crowding are associated with the anterior teeth not meeting at all. All anterior open bite is associated with infantile glossoptosis, which remains present throughout the lifetime of the individual and is pathologically expressed as OSA in adulthood.

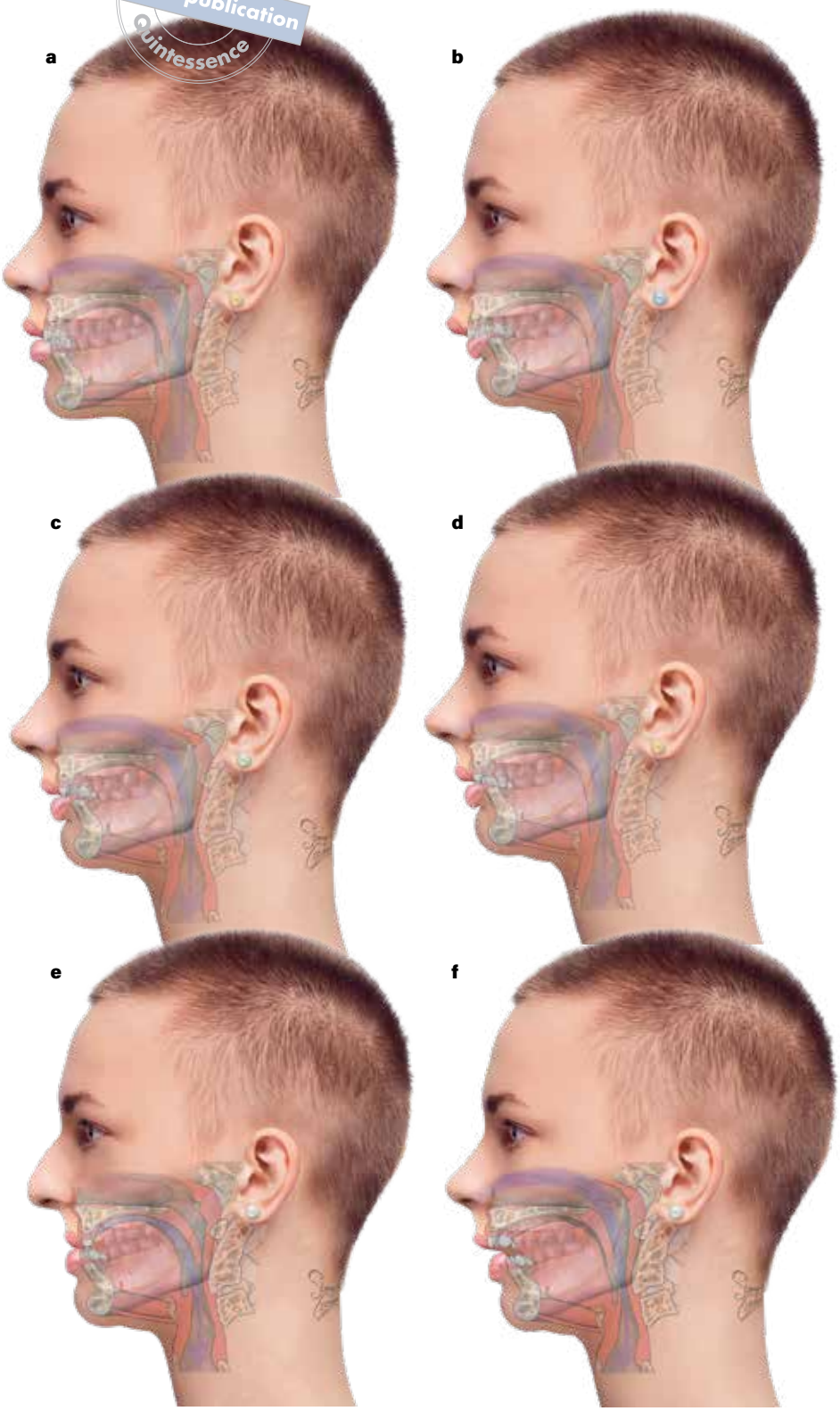

FIG 1-16 Orthognathic malocclusion series 4: Normogenia looking at the teeth, tongue, airway, facial profile, and jaw size. *(a)* Normally proportioned mandible and maxilla with a chin button (normogenia). This person is at least anatomical risk of lifetime development of OSA. *(b)* AMHypo with normogenia with the maxillary arch drawn normally. *(c)* AMHypo with normogenia with the collapse of the maxillary anterior teeth. *(d)* AMHypo with normogenia leading to mandibular crowding, in addition to maxillary crowding and impacted maxillary canines. *(e)* AMHypo with normogenia with a severely small maxilla and extreme dental crowding. *(f)* Here the smallness of the maxilla and the dental crowding are associated with the anterior teeth not meeting at all in anterior open bite. In all these cases *(b to f)*, the degree of glossoptosis and OSA risk is the same. Assessment of airway state in an erect, toned, and relaxed posture is a different assessment to being supine, relaxed, and in deep sleep, as will be explained in later chapters.

But to say AMHypo is abnormal, my first classification type is to describe the ideal. Thus I define the first state—Class I normal occlusion with 32 uncrowded teeth in a normal adult face. This is the anatomical and treatment ideal toward which we convert the AMHypo state, and from it relieve the patterns of airway, facial, and dental issues AMHypo causes. To accept this state is to believe that all of us should functionally develop 32 teeth—our teeth—as a normality of being our individual versions of human.

If you cannot accept this premise of my view of the ideal universal Class I occlusion founded on 32 teeth, then the rest of this book will be nonsense to you.

Pretreatment profile classification

My baseline dental assumption is that if there is nondevelopment of individual permanent teeth, it is because the teeth did not bud and therefore develop crowding or impaction. In my view, the lack of an individual random tooth is due to lack of local volume and not the result of specific genetic predetermination. A normal human complement of teeth is 32.

CLASS A

Class A describes a normal complement of 32 teeth without crowding in Class I orthodontic occlusion, with or without a chin button. There is no AMHypo. This may be called "Class I occlusion with no dental crowding and full dentition." This state is the gold-standard ideal outcome for all treatments that we apply to a face, jaws, and occlusion. There is no glossoptosis.

CLASS B

Class B describes the presence of AMHypo, with or without a chin button, with normal development of 32 teeth but with at least the mandibular third molars impacted. There is little remaining dental crowding, and the prominent anterior teeth are in an Angle Class II, division 1 pattern. There is normal maxillary development, though it is usually narrow posteriorly. This may be called "Class II malocclusion with prominent anterior teeth" or "Class II, division 1 malocclusion." There is inherent glossoptosis.

CLASS C

Class C also describes the presence of AMHypo, with or without a chin button, with normal development of 32 teeth but with all third molars impacted. There is significant dental crowding in the maxilla, such that the anterior teeth lean inward, reducing the natural dental overjet. The maxilla is not typically narrow, but it may be slightly shorter posteriorly. There is relatively little mandibular dental crowding. This may be called "Class II malocclusion with collapsed maxillary anterior teeth" or "Class II, division 2 malocclusion." There is glossoptosis.

CLASS D

Class D describes the presence of AMHypo, with or without a chin button, with normal development of 32 teeth. There is severe dental and incisor crowding in both jaws, but a Class I molar relationship is preserved. This may be called "Class I malocclusion with dental crowding." The third molars are usually all impacted. The maxilla is developmentally small, and there is glossoptosis.

CLASS E

Class E describes the presence of AMHypo with normal development of 32 teeth but with almost universal impaction of the third molars, particularly in the maxilla. The teeth are severely crowded, and the very small relative size of the maxilla brings the incisor relationship into a reverse overjet (the maxillary anterior teeth lie behind the mandibular anterior teeth). The overwhelming etiologic cause is the primary AMHypo, leading to chronic open mouth breathing and severe pneumatic underdevelopment of the maxilla and the midfacial sinuses. The chronic nasal obstruction associated with the deformed and reduced upper nasal airways is often seen as secondary to the open mouth breathing. In my view, AMHypo has led to supine open mouth breathing, which has lead to chronic nasal obstruction. Chronic nasal obstruction does not primarily lead to open mouth breathing. Because the mandible appears larger than the maxilla, a visual association is made between the large mandible and the severe dental malocclusion state. The presence of a hooked or aquiline or proportionally large nose, or dropped Caucasian nasal tip, is always because of developmental hypoplasia or smallness of the underlying piriform, nasal spine, and maxilla and is primarily caused by the primary smallness of the mandible and inherent glossoptosis. Overall, this pattern may be called "Class III malocclusion," and it is linked to the orthodontic description of "adenoid facies" and is the entire pathophysiologic pathway leading to the "Hapsburg jaw." The glossoptosis is chronic, with profound nasal obstruction.

CLASS F

Class F describes the presence of AMHypo, with or without a chin button, with normal development of 32 teeth. The third molars may or may not be impacted. Dental crowding is present in the maxilla, but the maxillary anterior teeth only partially overlap, or they openly overlap the line of the

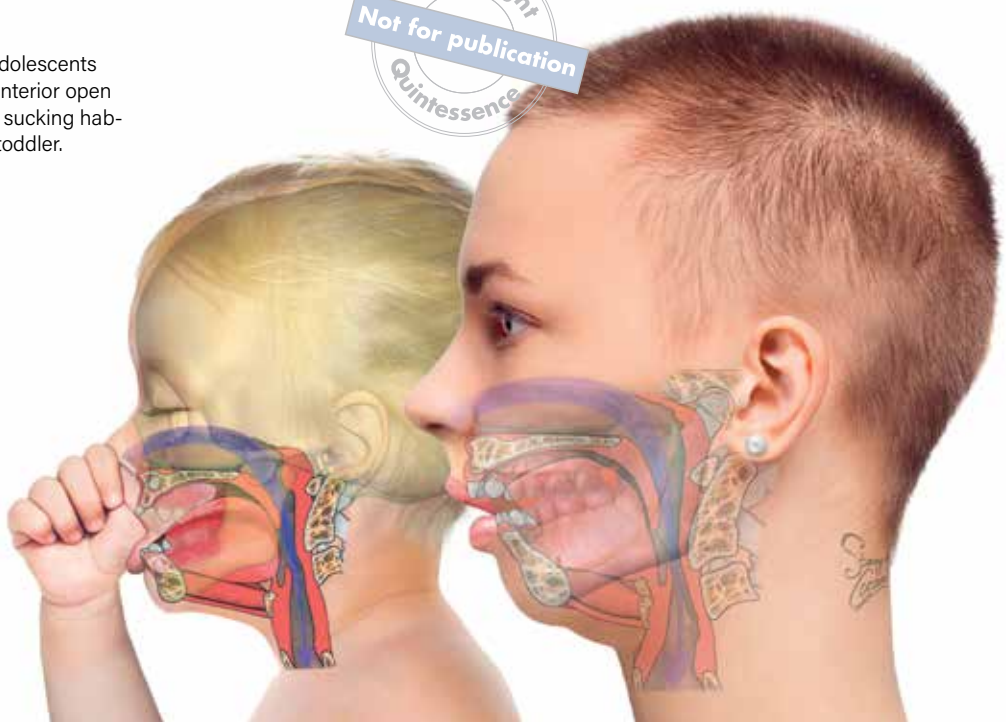

FIG 1-17 Most adolescents and adults with anterior open bite had a thumb sucking habit as an infant or toddler.

mandibular anterior teeth. This condition occurs because of chronic open mouth breathing, where there is over-eruption of the molars, which further props open the front of the mouth, leading to severe lip incompetency. Where the open mouth breathing has been closed because of adaptive thumb sucking in order to drive nasal breathing, there is worsening of the anterior dentoalveolar deformity in the maxilla. Overall the condition may be called "anterior open bite" and is often associated with tongue thrusting (see Fig 1-19), which helps close the oral lip seal and thus enables for normal nasal breathing while awake. Tongue thrusting does not cause the anterior open bite. Anterior open bite is overwhelmingly present only with AMHypo. The association of anterior open bite and glossoptosis is pathognomonic.

ANTERIOR OPEN BITE AND THUMB SUCKING

The adolescent presentation of prominent anterior teeth, a small mandible, tongue thrusting, and anterior open bite malocclusion is seen to be associated with an infant or childhood habit of thumb sucking (Fig 1-17). How infantile thumb sucking transitions into anterior open bite in the adolescent, and why the infant sucks their thumb in the first place, has been an eternally confused area of clinical philosophy and conjecture involving many craft and interest groups, and thumb sucking is an almost universally demonized childhood habit. In my view, thumb sucking is actually an adaptive response to glossoptosis and a life-saving habit for babies with abnormally small jaws (see Fig 1-19).

To understand anterior open bite is to also understand the interrelationship between concepts of obligate nasal breathing, tongue thrusting, glossoptosis, AMHypo, anterior oral seal, and thumb sucking. Neonates are naturally obligate nasal breathers, and to nasally breathe, the mouth and jaws must be closed and sealed. But in a neonate with a small mandible, and with innate glossoptosis, the baby may find it desperately difficult to nasally breathe while asleep and lying on their back (Fig 1-18).

Pierre Robin was the first person to describe the reasons behind the phenomenon of the "blue baby." He taught mothers and doctors how to nurse and lay these small-mandible babies prone and on their stomachs to help with breathing. Unfortunately, his insight and knowledge on the dangers of glossoptosis are barely remembered. After all, mothers today are taught to always place their babies on their back to sleep—"back is best" —to reduce the risk of sudden infant death syndrome. But for babies with a small mandible and glossoptosis, there is inherent tongue collapse that blocks normal breathing during supine sleep. Cue the thumb.

A distressed newborn, lying on their back and unable to nasally breathe with a closed mouth, will be reactively

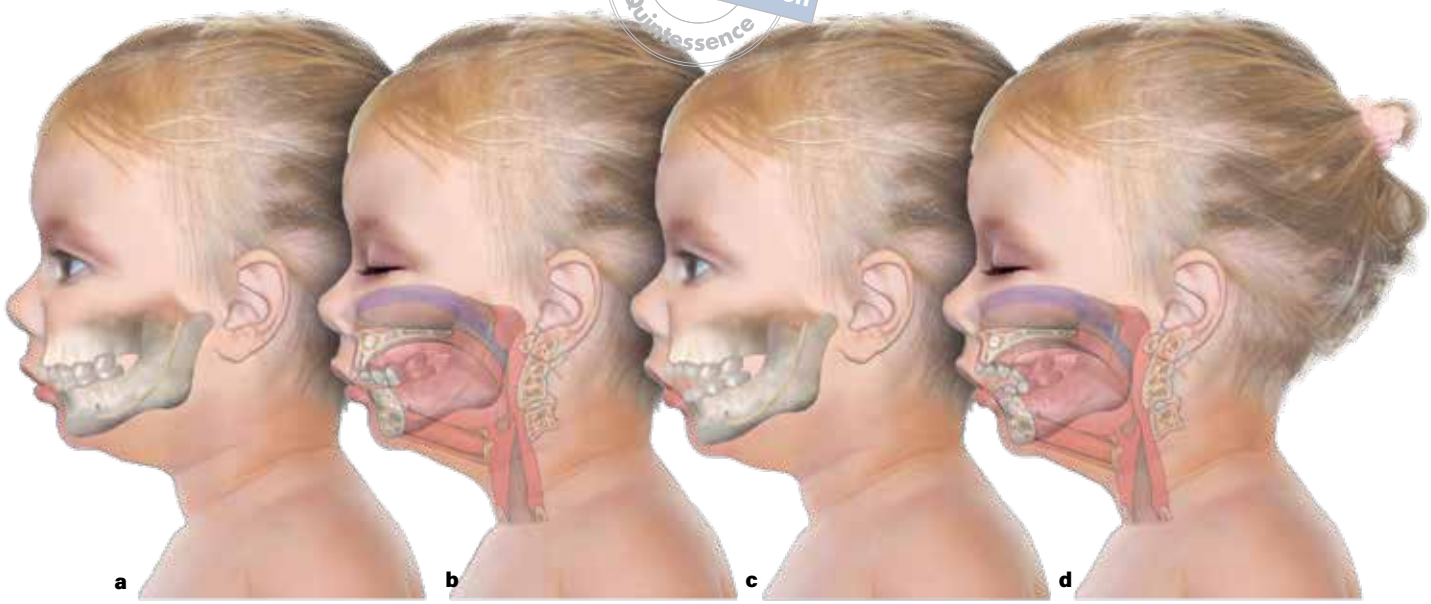

FIG 1-18 A young child with a prominent dental overjet *(a and b)* and a child with anterior open bite *(c and d)* have the same inherited small mandible (AMHypo). All that differs between them are the different postural habits that enable them to overcome glossoptosis and nighttime airway collapse. The child with the prominent overjet learns to sleep with their mouth open, lying on their front or side. The child with anterior open bite, on the other hand, learns to suck their thumb to help hold the mandible forward when they sleep in any position. Which way the child randomly selects will lead eventually to an adolescent orthodontic and orthognathic classification.

unsettled. They will thrash and cry, and the thumb waving in front of them becomes the soother. Not just for comfort like many believe, but because the simple act of sucking the thumb naturally closes the mouth, seals the lips, and holds the tongue and small mandible forward, thereby relieving the glossoptosis and permitting nasal breathing again (Fig 1-19).

Seen this way, then, thumb sucking is a functional and life-saving adaptation to a fundamentally abnormal anatomical condition. Eventually this thumb sucking becomes a functionally dependent behavior whereby normal nasal breathing, even during supine sleep, becomes possible and dependable and necessary (and terribly hard to break as an unconscious habit later in childhood).

Many dentists and speech pathologists recognize the association between thumb sucking and anterior open bite and small jaws and erroneously assume that the thumb sucking causes the small jaws. But it is actually the other way around. Adolescents and adults with anterior open bite and small jaws have always had an inherently small mandible, and the resulting glossoptosis made it difficult for them to breathe during sleep, hence the natural neonatal survival mechanism of thumb sucking. While almost all people who have anterior open bite sucked their thumbs as children, thumb sucking did not cause their small jaws.

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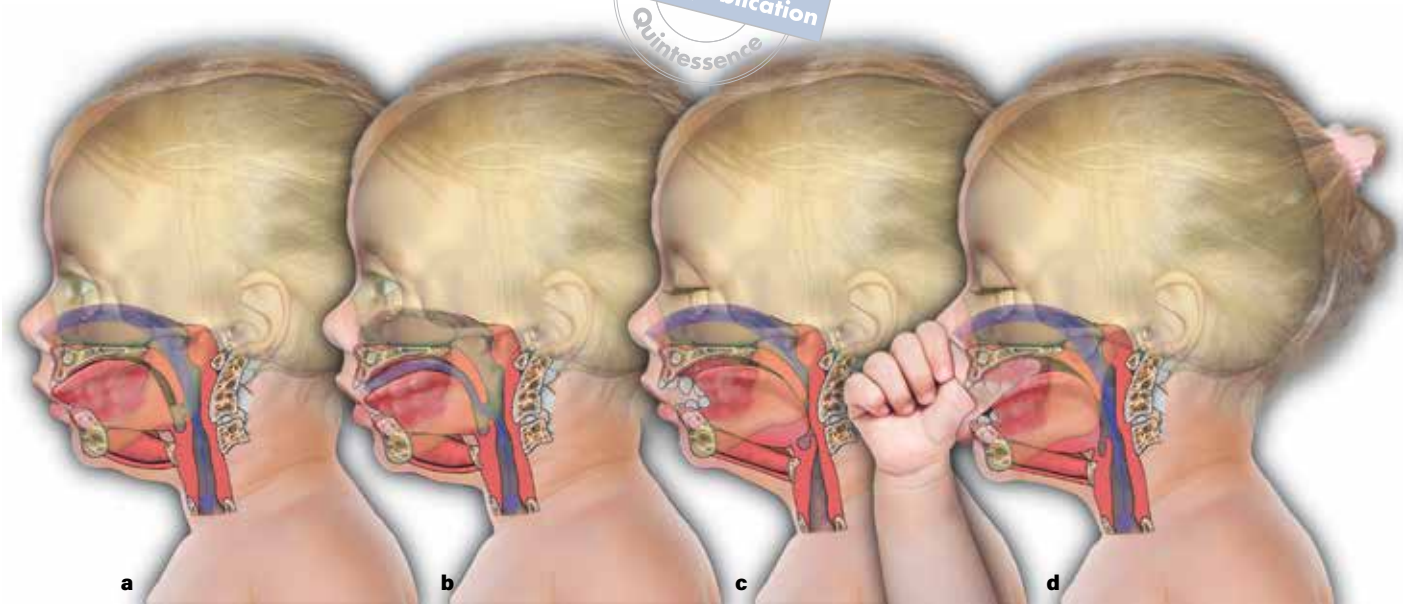

FIG 1-19 A common complaint of orthodontists treating anterior open bite in adolescents is persistent tongue thrusting that they say complicates successful orthodontic therapy. Tongue thrusting *(a)* as an adaptive position of the tongue has its origin in the infantile period and is only present during the awake state. By closing the hole between the anterior open bite and parted lips, it simultaneously brings the back of the tongue forward and restitutes normal nasal breathing. Tongue thrusting, as with general tongue tone, can only occur during fully awake or light sleep states. Without tongue thrusting, there is no innate oral seal, and obligate open mouth breathing occurs *(b)*. During deep sleep, with loss of tongue tone and under the influence of gravity with supine sleeping, the relaxed tongue collapses the retroglossal airway completely, which is called glossoptosis *(c)*. Lying on the side, along with the natural discovery of the thumb, enables the mandible to translate forward and thus opens the retroglossal airway. The second metatarsal joint of the thumb locks behind the incisor teeth, and the lips form a natural seal. The combination enables natural nasal breathing, and a simultaneous natural relief of both oral seal and of glossoptosis *(d)*. There is nothing unnatural about thumb sucking. Thumb sucking is a naturally adaptive measure to the primary state of AMHypo in order to overcome glossoptosis—and thus survive the neonatal and infantile period. It leads to normal midfacial development and normal sinus health. But it is demonized for the deformation of the anterior maxilla and is blamed as a cause for the development of the small jaw.

INDEX

Page numbers followed by "f" denote figures.