

Aesthetic Talk

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Aesthetic Talk

Clinical pearls from nearly twenty years in aesthetic medicine

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Aesthetic Talk: Clinical pearls from nearly twenty years in aesthetic medicine

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*For every patient who trusted me with their face,
and for every practitioner who asked the question
nobody else in the room dared to ask.*

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Important notice

This book is intended for use by qualified medical and healthcare professionals only. The information it contains is provided for educational purposes and does not constitute medical advice. The techniques, products and protocols described should be performed only by appropriately trained and licensed practitioners, acting within their scope of practice and in accordance with the laws and regulations of their own jurisdiction.

Medical knowledge evolves rapidly. Every effort has been made to ensure the accuracy of the information at the time of publication, but the reader is responsible for verifying current guidelines, product licensing, manufacturer instructions and regulatory requirements before applying any technique described here.

The author and publisher expressly disclaim any liability for adverse outcomes, complications, loss or damage arising from the application of the information in this book. Aesthetic medicine carries inherent risk. Practitioners must obtain appropriate training, indemnity insurance and informed consent before performing any procedure.

This book does not replace formal training, mentorship or clinical supervision. The author strongly recommends hands-on training from qualified instructors before attempting any new technique.

A note on the numbers

Every dose, volume, injection depth, needle angle and anatomical measurement given in these pages is an approximate working range for an average adult, offered to illustrate relationships and principles — not a prescription to be applied to a specific patient.

Human anatomy varies substantially between individuals and the figures quoted will not hold true for every face. Depths in particular are given to teach the relative position of structures. The practitioner remains wholly responsible for assessing the patient in front of them, and for verifying current product, dosing and safety information from the manufacturer and from up-to-date clinical literature before treating.

A note on the patients

This is a work of clinical memoir. Every patient, colleague and clinical encounter described for narrative purposes is a composite, or has been substantially altered: names, nationalities, ages, timelines, settings and clinical details have all been changed in order to protect confidentiality.

No individual is identifiable, and any resemblance to a specific person is unintended. The clinical lessons are real; the people, as presented, are not.

A note on terminology

Throughout this book the term *patient* is used in preference to *client*. Aesthetic medicine, whatever its elective nature, is medicine.

The terms *filler* and *dermal filler* refer to injectable hyaluronic acid-based products unless otherwise specified. *Toxin* refers to botulinum toxin type A. Brand names are used where clinically relevant and do not constitute an endorsement. All product use should comply with current licensing and prescribing regulations in your jurisdiction.

PREFACE

The Accidental Aesthetician

My name is Dr. Ahmed Haq, and I became a Harley Street aesthetic doctor almost entirely by accident. I did not grow up dreaming about lips. I was trained in surgery.

Real surgery. Long nights, bad coffee, NHS corridors, people shouting across theatres, and consultants who could destroy your confidence with a single eyebrow movement. I graduated from the Royal College of Surgeons in Dublin and came to London for the West London surgical rotation. Eventually I ended up doing cardiothoracic surgery at the Royal Brompton.

To be clear, I was a junior surgeon — not some legendary heart surgeon saving lives while jazz music played dramatically in the background. Mostly I was learning, assisting, surviving, and trying not to embarrass myself in theatre. But surgery teaches you things that never leave you. Precision. Patience. Respect for tissue. And most importantly: humility. Because the human body has a very efficient way of punishing arrogance. Somewhere in the middle of all that, I had the opportunity to spend time at Johns Hopkins in Baltimore researching cardiac stem cells. That place changed the way I viewed medicine forever.

Johns Hopkins was operating on another level entirely. They had operating theatres dedicated purely to research. Not patients. Research. And inside those rooms were third-year medical students suturing bowel with better technique than many senior surgeons I had seen back in the UK. They were taught properly from the beginning — how to hold instruments, how to handle tissue, where to place tension, how to close bowel without crushing it with forceps like you were trying to repair garden hose piping.

Technique was treated like religion there. That experience stayed with me because it taught me something important: talent is overrated. Repetition, observation, and proper teaching matter far more. Then life intervened.

I was diagnosed with Hodgkin's lymphoma. One day you think your future is mapped out for the next thirty years. The next, you are sitting in a consultant's office being told that your own cells are trying to kill you.

The first time was, as these things go, contained. Six months of combination chemotherapy — the route the professor looking after me wanted to take — and then out the other side.

Contained is not the same as small. Cardiothoracic surgery is not a career you conduct between cycles of chemotherapy, and by the time I finished, the path had closed behind me. I would like to record, for the sake of accuracy, that I did not greatly miss the shouting, the tension, or the consultants who could dismantle you with a single movement of one eyebrow.

I got through it, and I never went back to surgery. I started doing aesthetics instead, and within a couple of years the weekends were covering a month of NHS salary. I kept picking up locums in A&E regardless, partly because I did not entirely believe the new life was real, and partly because I wanted to stay in touch with medicine that had blood in it.

Five years later it came back, and that settled the argument. I left A&E as well.

The second time was a different order of thing entirely: radiotherapy, and then a stem cell transplant. I ended up in the Intensive Treatment Unit, circling the drain with critically low potassium. I remember lying there, machines beeping, fully aware of exactly what those numbers on the monitor meant, thinking: *Well, this is an inconvenient time to die.*

I didn't die. I survived, mostly out of stubbornness.

And then the strangest thing happened. When my hair finally grew back, it came back pitch black. I had been greying before the cancer. The chemotherapy had essentially given me a forced reboot, and apparently, my stem cells decided I needed a younger aesthetic. It was a dark, cosmic joke. I nearly died, but I came out of it with better hair.

But survival is only the first part of the problem. The second part is figuring out what the hell you are supposed to do with the life you just fought to keep.

I couldn't go back to the grueling physical demands of cardiothoracic surgery. My body wouldn't take it.

Two years later, the collateral damage of the illness would quietly dismantle my marriage, leaving me navigating a divorce and trying to figure out how to be a father to my son while my entire professional identity was in ruins.

There is one more thing about leaving, and I have never seen it written down anywhere, so I will write it down here.

Aesthetics paid. Not immediately, but soon, and then very well, and a doctor who has spent his entire adult life on a salary scale is not remotely prepared for what that does to him. I was still doing A&E shifts at the time, alongside people who were better clinicians than me, working harder than me, on a fraction of it. Then I would walk out to the car park.

You cannot drive a Porsche into an NHS car park. I mean that literally — you can, and I did, and I would not recommend it. Nobody says anything.

That is the point. The disapproval is ambient rather than spoken, and it is not really about the car. It is about a suspicion that runs through the whole of British medicine: that this is a vocation rather than a job, that a proper doctor is one who is being slightly ruined by it, and that anybody visibly doing well has quietly stopped being a proper doctor.

I felt it, and I felt it for years. It is remarkably difficult to look a colleague in the eye when they are worrying about a mortgage and you are not.

I have made my peace with it, and my peace is not the comfortable version.

The comfortable version is that earning well allowed me to practise with integrity — that I could turn patients away and refuse work I did not believe in because I did not need the money. It sounds good. I do not entirely believe it, and I have watched enough of this profession to know why.

Beyond a certain point, money does not remove the pressure to say yes. It creates it. A large lease on a fashionable street, staff to pay, equipment on finance, and a standard of living that has to be maintained will push a practitioner towards yes far harder than a modest practice ever will.

Some of the most over-treated faces I have seen were produced by very successful clinics, and they were over-treated for a reason that had nothing to do with clinical judgement. The incentive does not disappear as you earn more. Past a point, it inverts.

What actually matters is smaller and much less flattering. It is whether you need *this* patient, this week. A practitioner who requires the four hundred pounds sitting in front of him to cover something due on Friday will find it harder to say no, and that has nothing to do with being rich. It has to do with

the distance between what you earn and what you are committed to spending.

The freest practitioners I know are not the wealthiest. They are the ones with low overheads and no appetite for a life that has to be fed.

So the lesson is not earn more. It is owe less. Keep the gap wide enough that any individual patient can walk out of your clinic untreated without it mattering to you, and protect that gap more carefully than you protect your income. That is available to a practitioner in a rented room in a market town and unavailable to plenty of people on Harley Street, and any patient reading this should understand that the car in the car park tells them nothing whatsoever about the judgement of the person driving it.

In the interests of full disclosure, having just told you the car means nothing: I drive a Porsche. I have driven one more or less continuously for years, and I would like it on the record that I treat them appallingly. Every one I have owned has gone past a hundred thousand miles before I replaced it.

School runs, motorway slogs, supermarket car parks, more airport runs than I care to count. If you ever want to identify mine in a car park, look for the worst tyres.

I mention it because it makes the point better than the point did. It is not a trophy, it is a workhorse that happens to be German, and the only thing it reliably tells you about its driver is that he has an inefficient attitude to depreciation.

Nobody ever became a better physician by being frightened about money. And nobody ever became one by having a great deal of it, either. We simply have a national habit of pretending the first thing is a virtue. I should say what the gap was actually for, in my case, because it was not the car.

I was a single father. The clinic was busy — busier than it had any right to be, for reasons I will come to in a later chapter — and I ran it part-time anyway. School drop-off in the morning.

Pick-up in the afternoon. I did not take calls in the evenings, and I was reliably useless to anybody who tried. The practice was organised, quite deliberately, around a nine-year-old's timetable rather than the other way round.

There is a version of this book in which that is a footnote about work-life balance. It is not a footnote. It is the answer to the question of what any of this was for.

I have had a reasonably eventful career. I have injected on aircraft, presented at Olympia, been shouted at by consultants, been threatened by lawyers, been very frightened twice about my own mortality and once about my professional survival. If you asked me which part of it I would keep, it would not be any of that.

It would be the ordinary afternoons: the school gate, the walk home, the conversation about nothing in particular.

That is what the gap between earning and spending buys. Not a car. Time that belongs to you, in a career that will otherwise take all of it.

Someone mentioned aesthetics. At the time, aesthetics felt completely alien to me. Nobody in my family had Botox. None of my friends had filler. I did not even properly understand the industry. To me, it seemed like a strange parallel universe populated by glossy brochures and unnaturally smooth foreheads.

But I needed to survive. So I did what any confused doctor with mild desperation and access to the internet would do. I built a website. Years earlier, at boarding school in America, I had been forced to learn HTML coding — a class I absolutely hated at the time.

Suddenly it became useful. Funny how life works like that. The things you resent often become the things that rescue you later. I built a basic webpage overnight, wrote some painfully average marketing copy, and put it on Google Ads the next morning. Then the phone started ringing. There was just one problem. I had no clinic. No insurance. No real plan. And almost no practical experience. So naturally, I told everybody I was fully booked for the next two months. Then I hung up the phone and panicked. I found the cheapest accredited course I could afford.

It taught me approximately three things: how to inject Botox into the frown, how to place filler into the nasolabial fold, and how to cautiously inject about 0.2 millilitres into a lip while looking terrified. But the course gave me one genuinely valuable piece of advice: Do not lose momentum.

So I started injecting almost immediately.

I could not afford a proper clinic, so I began doing house calls across London. Which somehow escalated into treating patients in Chelsea, then flying abroad, then eventually treating people on private planes heading into Dubai and across the Middle East. None of this was planned.

The strange thing is that aesthetic medicine back then was simple. Incredibly simple compared to now. One filler. A few Botox indications. A

couple of lips. Maybe a nasolabial fold if you were feeling adventurous. That was the industry.

Then patients started asking for more. Cheeks. Jawlines. Temples. Tear troughs. Noses. And honestly, most of us learned in real time. Not through endless expensive courses. Not through social media reels filmed under surgical lighting while someone says “absolute game changer” every fourteen seconds. We learned through repetition.

Observation. Mistakes. Thousands and thousands of faces. In 2010, I performed one of the first non-surgical rhinoplasty demonstrations at a major aesthetic conference. Standing there in front of consultant ENT surgeons and professors discussing injectable rhinoplasty felt surreal at the time. Half the audience was fascinated.

The other half looked like they wanted security called immediately. But that was aesthetics then. We were building the field while simultaneously arguing about whether it should even exist. And if I am honest, the lips humbled me more than anything else. Even after doing several lip patients a day, every day, for years, I still felt nervous.

Lips are dynamic, unforgiving, emotional structures. Tiny differences completely change a face. Back then my lip technique was embarrassingly simple: inject, apply gauze, compress, pray. You would release the gauze after two minutes and hope the outcome looked elegant instead of catastrophic. People now imagine aesthetic medicine as glamorous.

But the truth is most injectors spend years quietly developing instinct through repetition and anxiety. Which is why I remain sceptical of overly theatrical cadaveric teaching courses. We are not open surgeons dissecting tissue planes in preserved anatomy labs. Injecting a living face is completely different.

Blood vessels move. Tissue responds. The patient breathes. The anatomy is alive.

Over the years, I watched colleagues complicate their lip technique. More entry points. More product. More drama. And more disasters. Lips that looked like they had been inflated rather than enhanced. Patients who came back unhappy, swollen, and asymmetric. My technique stayed simple.

Inject. Massage. Hold gauze for a few minutes. And the results were consistently beautiful. Not because I was more talented. Because I was less complicated. The simplest technique is often the best technique. Least trauma.

Maximum result. Minimum opportunity for error. That lesson took me years to fully trust. Now I teach it on day one.

The Dunning-Kruger Problem — Why the Most Dangerous Injector in the Room Is the One Who Just Finished Their First Course *Let me tell you about the most dangerous injector I have ever met. I had known him for years. I saw him every morning in the mirror.*

Early in my aesthetic career — I had done the courses, I had the certificates on the wall, I felt entirely competent — I was injecting a bottom lip. Simple case. A little volume, a little definition. I had done it before and it had gone fine, which is precisely the problem, because “it went fine before” is the sentence that precedes most disasters in this field.

I watched the lip balloon in front of me. Not swell — balloon. And then, over the next few seconds, it went blue. A deep, dusky, wrong blue that I had read about in exactly one paragraph of one manual and had never expected to see on a person, in my chair, because of my needle.

I had put the product where I thought it should go. The anatomy underneath had other ideas. In that moment I understood, with a clarity that no course had ever given me, that I did not actually know where my needle was. I knew where I intended it to be. Those are not the same thing, and the distance between them is where people get hurt.

The lip recovered. I was lucky, and the patient was gracious, and I have thought about that blue lip almost every working day since. It was the best thing that happened to my career, because it knocked me off a peak I did not know I was standing on.

There is a psychological phenomenon called the Dunning-Kruger Effect. It was described in 1999 by two psychologists at Cornell University who noticed something deeply uncomfortable about human nature: the less competent a person is, the more confident they tend to be. And the more competent a person becomes, the more acutely they feel the weight of what they still do not know.

The Dunning-Kruger curve. The peak of confidence comes early, on almost no competence — which is precisely where the most dangerous injector stands.

In most fields, this is an interesting academic observation. In aesthetics, it is a clinical hazard. The curve tells the story better than any paragraph. You begin knowing nothing.

Confidence is low. Then you do a beginner course. You learn the basics. You inject a few lips. The results are fine. The patients are happy. And suddenly, you are standing on the Peak of Mount Stupid — the highest point of confidence relative to actual competence that you will ever reach in your career.

At the peak, you feel invincible. You post your results on Instagram. You book more patients. You start offering treatments you have not been taught. You tell yourself that the advanced anatomy is for surgeons, not injectors. You believe that a good eye and a steady hand are sufficient.

They are not. The fall from the peak is inevitable. It is triggered by the first complication you do not understand. The first result that looks wrong and you cannot explain why. The first patient who comes back with a lump and you have no idea what caused it.

The first time you inject a tear trough and the patient looks worse, not better. The first time a lip goes blue in front of you.

That is the Valley of Despair. Most practitioners hit the valley somewhere between six months and two years into their practice. The good ones stay there for a while, sit with the discomfort, and start reading. They go back to the anatomy.

They take advanced courses. They watch their results with a more critical eye. They realise that what they thought was a good result was actually a mediocre result, and that they had simply not yet developed the eye to see the difference. That is the Slope of Enlightenment.

The bad ones never reach the valley at all. They stay on the peak indefinitely, protected by the fact that most patients do not know enough to tell them the result is wrong. They accumulate a practice built on patients who are grateful for anything, and they mistake gratitude for excellence.

The Plateau of Sustainability — the place where genuine competence and genuine confidence finally meet — is earned slowly. It looks like quiet certainty. It looks like a practitioner who can explain exactly what they did and why, who can predict a complication before it happens, who can look at a face and know immediately which layer has failed and what to do about it.

It does not look like a hundred Instagram posts. If you are reading this book, you are probably in one of two places on the curve. You are either on the peak — fresh from a beginner course, full of enthusiasm, and slightly unnerved by how much of this book you did not know existed.

Or you are in the valley — experienced enough to have seen your own limitations, humbled enough to go looking for answers. Both are good places to be.

The peak is uncomfortable once you realise where you are. But the realisation itself is progress. The moment you understand that a weekend course does not make you a facial anatomy expert is the moment you start becoming one. The valley is painful.

But it is the only road to the plateau. This book is written for the practitioner who has decided to climb. Not the one who is satisfied with the view from the peak, but the one who looked down and felt the vertigo of realising how far there is still to go.

That vertigo is not a weakness. It is the beginning of clinical intelligence. Welcome to the slope.

The frameworks that follow in this book — the three cells of the skin, the three layers of injection, the sequencing of treatment by stage — are set out formally, with the evidence, in three companion papers published in the Harley Street Institute journal, *Aesthetic Intelligence*. The book teaches them the way I actually think about them. The papers defend them the way a journal requires. If you want the references, go there. If you want the reasoning, stay here.

Introduction: Leave Your Textbooks at the Door Let me tell you what this book is not. It is not a surgical anatomy atlas. It is not a dermatology reference. It is not a plastic surgery manual. If you came here looking for the precise course of the angular artery, the histological subtypes of collagen fibres, or a diagram of the facial nerve branches, I have bad news for you.

Close this book. Go find a library. That information exists in abundance, and it will not help you inject a face. This book was written from the point of view of injection and action. That is the only point of view that matters when you are standing in front of a patient with a syringe in your hand.

Here is what I want you to do before you read another word: take everything you think you know about facial anatomy — the arteries, the veins, their courses, their depths, the surgical planes, the named structures that surgeons spend years memorising — and put it in a box. Not forever. Just for now. Because the problem with coming into aesthetic medicine loaded with surgical anatomy is that you start thinking like a surgeon. And surgeons, for all their considerable talent, are not injectors. A surgeon lifts.

A surgeon cuts. A surgeon removes. Their entire framework is built around accessing structures, moving them, tightening them, excising them. The results, when the surgeon is good, can be extraordinary. But they are also, very often, not natural. Because when you treat only the laxity — when you pull the skin tight without addressing the volume loss underneath it — you get a face that looks operated on.

Tight in the wrong places. Hollow in others. The light hits it differently. The shadows are wrong. You can always tell. Dermatology has its own blind spot. The dermatologist looks at the surface.

The disease. The texture. The pigmentation. The skin itself. And they are brilliant at it. But the skin is not the whole story. The skin is the last layer.

It is the result of everything happening underneath it. What I am asking you to do in this book is think differently. Think in layers. Think in depth.

Think in the way that an injector thinks. We start at the surface — the epidermis, the dermis — because that is where we can see and touch. Then we go deeper.

The subcutaneous layer. The fat compartments. The SMAS. The bone. Each layer has its own biology, its own ageing process, its own set of problems, and its own set of solutions. And the injector's job is to understand all of them, because the injector is the only specialist who can address all of them without cutting a single thing. That is not a small thing.

That is actually extraordinary. Think about what that means in practice. When a patient comes to me, I am not looking at their nasolabial folds in isolation. I am not looking at their lips and thinking only about the lips. I am looking at the entire face as a system.

I am asking: what is the primary driver of ageing here? Is it volume loss? Is it skeletal resorption? Is it skin laxity? Is it all three? And then I am asking: what is the right age group for what I am about to do?

Because the patient who needs a facelift should not be sitting in my chair getting filler. And the patient who needs filler should not be sitting in a surgeon's chair getting pulled. My job is to optimise.

Symmetry, balance, proportion. If I am improving someone's lips, why am I not also addressing the perioral lines? If I am treating the nasolabial folds, why am I not thinking about the cheek volume that is causing them? If I am rejuvenating the lower face, why am I leaving the upper eyelids looking like they belong to a different person?

This is the thing that drives me absolutely insane about the way aesthetics is often practised. A patient will come in, get a full face of filler — cheeks, jawline, the works — and walk out with lips that look like they belong on a dried apricot.

Nobody thought about the lips. Nobody thought about the periocular area. Nobody thought about the whole. They treated the parts they were asked about and sent the patient home. That is not optimisation.

That is a transaction. This book is about optimisation. It is about looking at the face the way a composer looks at an orchestra — not instrument by instrument, but as a whole, understanding how each section affects every other section, knowing when to bring something in and when to leave it alone.

You will not find surgical anatomy here. You will find injection anatomy — the anatomy of what we can reach, what we can change, and what we should leave alone. You will find clinical thinking.

You will find honest opinions. You will find the things that the textbooks do not tell you, because the textbooks were written by people who have never stood in front of a patient at nine o'clock on a Tuesday morning and had to make a decision.

Read this book the way it was written. With your preconceptions set aside, your mind open, and your ego checked at the door. The face is waiting.

“You cannot fix what you cannot see. And you cannot see what you do not understand.”

“A craftsman knows his tools. A master knows when not to use them.”

“Every face is a different problem. Every problem has a layer.”



PART I

The Architecture of the Skin

“If you do not understand the canvas, you have no business holding the brush.”

CHAPTER 1

The Three-Cell Theory — Understanding Skin at the Source

Aesthetic medicine has an obsession with products. We talk endlessly about the newest filler, the strongest toxin, the most advanced biostimulator. We memorize rheology charts and debate G-prime until we are blue in the face.

But products do not heal tissue. Products do not build collagen. Products do not make skin look younger.

Cells do.

If you want to understand aesthetic medicine, you have to stop looking at the syringe and start looking at the cells. Everything we do — every injection, every peel, every laser, every thread — is simply a method of manipulating cellular behaviour. We are either trying to wake a cell up, calm a cell down, or convince a cell to build something it stopped building ten years ago.

If you do not understand the cells you are targeting, you are just a technician following a recipe.

There are three cells that dictate the aesthetic quality of the human face. If you master how these three cells behave, how they communicate, and how they fail, you will understand ninety percent of non-surgical aesthetics.

I call this the Three-Cell Theory.

1. The Fibroblast (The Architect)

The fibroblast lives in the dermis. It is the architect and the builder of the skin. Its job is to produce collagen, elastin, and hyaluronic acid — the structural proteins and hydration molecules that make skin firm, bouncy, and resilient.

When we are young, fibroblasts are highly active. They constantly repair and rebuild the dermal matrix. But as we age, they become lazy. They go to sleep. They stop producing collagen and start producing enzymes (metalloproteinases) that actually break collagen down.

Almost every anti-ageing treatment we perform — from microneedling to polynucleotides, from chemical peels to radiofrequency — has one primary goal: to wake the fibroblast up and force it back to work.

2. The Keratinocyte (The Bricklayer)

The keratinocyte lives in the epidermis. It is the bricklayer of the skin. Its job is to form a tough, waterproof barrier that protects the body from the outside world.

Keratinocytes are born at the bottom of the epidermis and slowly migrate to the surface, where they die and flake off. In youthful skin, this turnover process takes about 28 days. The surface is constantly refreshed, reflecting light evenly and giving the skin a “glow.”

As we age, this turnover slows down drastically. It can take up to 45 or 60 days. The dead keratinocytes pile up on the surface like unraked leaves. The skin becomes dull, rough, and thickened.

When we use retinoids, chemical peels, or exfoliants, our goal is to accelerate the keratinocyte turnover rate, forcing the skin to shed its dead layers and reveal the fresh, light-reflecting cells beneath.

3. The Melanocyte (The Painter)

The melanocyte lives at the junction between the epidermis and the dermis. It is the painter of the skin. Its job is to produce melanin — the pigment that gives skin its colour and protects it from UV radiation.

Melanocytes are highly reactive and easily traumatised. When they are exposed to UV light, hormonal changes, or physical trauma (like picking a spot or a harsh laser treatment), they panic. And when a melanocyte panics, it throws paint everywhere. This results in hyperpigmentation, melasma, and uneven skin tone.

Treating the melanocyte is the hardest job in aesthetic medicine. You cannot force it. You cannot aggressively attack it. You have to negotiate with it. You have to calm it down using tyrosinase inhibitors, gentle peels, and strict sun

protection.

The Ecosystem

These three cells do not work in isolation. They communicate constantly. If the keratinocyte barrier is compromised, UV light penetrates deeper, triggering the melanocyte to overproduce pigment. If the fibroblast stops producing collagen, the dermal matrix collapses, and the epidermis above it wrinkles, no matter how fast the keratinocytes are turning over.

You cannot treat one without considering the others.

When a patient sits in your chair complaining of “looking tired,” they are rarely complaining about one specific thing. They are complaining about the combined failure of these three cells.

Their fibroblasts have stopped building (laxity and fine lines).

Their keratinocytes have stopped shedding (dullness and rough texture).

Their melanocytes have panicked (uneven pigmentation).

Your job is not to sell them a syringe of filler. Your job is to diagnose which cell is failing and design a treatment plan to fix it.

Once you view the face through the lens of the Three-Cell Theory, aesthetic medicine stops being a menu of treatments and becomes a logical, biological science.

Why This Matters in the Consultation Room

Here is where the theory becomes practical. When a patient sits down in your chair and tells you they want to “look fresher,” most injectors immediately start thinking about which syringe to pick up. Botulinum toxin for the forehead. A bit of filler under the eyes. Maybe some lip volume.

But if you have absorbed the Three-Cell Theory, your brain works differently. You look at the face and you ask: which cell is the primary driver of what I see?

If the skin is dull, rough, and congested with uneven tone, the keratinocyte is the problem. No amount of filler will fix that. You need to accelerate turnover. Retinoids. A peel. Consistent SPF. The patient does not need a needle yet. They need a prescription.

If the skin is sagging, creased, and lacking the bounce it had at thirty, the fibroblast is the problem. You can fill the hollows, yes. But if you do not

address the underlying collagen deficit, you are inflating a deflating tyre. You need to wake the fibroblast. Polynucleotides. Radiofrequency. Microneedling. Biostimulators. The filler is the finishing touch, not the foundation.

If the skin has patches of brown, uneven pigmentation, and post-inflammatory marks, the melanocyte is the problem. And the melanocyte is the most dangerous cell to treat aggressively. Go too hard, too fast, and you will make the pigmentation worse. You need to negotiate. Slow, consistent tyrosinase inhibition. Gentle chemical exfoliation. Strict, non-negotiable sun protection. Patience.

This is the clinical application of the Three-Cell Theory. It is not academic. It is the difference between a practitioner who gets results and a practitioner who gets complaints.

The Hierarchy of Treatment

There is also a logical hierarchy here that most practitioners get backwards.

Most practitioners treat the surface first. They see a wrinkle and they fill it. They see dullness and they reach for a skin booster. They see a fold and they inject it.

The correct hierarchy is: fix the cell behaviour first, then refine the architecture.

A patient with severely compromised keratinocyte function — thick, dull, congested skin — will not absorb a skin booster efficiently. The product will sit in the wrong plane. The result will be mediocre.

But if you spend six to eight weeks on a proper retinoid and SPF protocol first, the skin transforms. The keratinocytes are turning over properly. The barrier is intact. Now when you inject a skin booster, it integrates beautifully. The result is dramatically better.

The same logic applies to the fibroblast. A patient who has been on a good polynucleotide protocol for three months will respond to filler differently than a patient who has never done any collagen stimulation. Their dermal matrix is healthier. The filler integrates better. It lasts longer. The result looks more natural.

Fix the biology first. Then refine the architecture.

That is the Three-Cell Theory in practice. And that is where we begin.

(The formal version of this model — the fibroblast, the melanocyte, the keratinocyte, and the treatments that act on each — is set out with the evidence in the first of three companion papers in Aesthetic Intelligence, the Harley Street Institute journal. The book gives you how I think about it; the paper gives you the references. Together with the papers on injection depth and on sequencing, they carry one idea between them, and you will meet it on nearly every page from here:

anatomical ageing is not a treatment target. We are not rebuilding the face you used to have. We are looking after the one you have now.)

CHAPTER 2

The Three-Layer Theory — Injection Anatomy

If the Three-Cell Theory tells you *what* is happening to the skin, the Three-Layer Theory tells you *where* to put your needle to fix it.

Choosing the tool

	Needle	Cannula
Plane it suits	Dermis (perpendicular); periosteum (on bone)	Subcutaneous fat, along the plane
Typical gauge	27–30G, short	22–25G, 50–70 mm
Best for	Precise, small-volume deposits at a fixed point	Volume and blending across a wide area from one entry
Movement	In and out at one point	Travels; one entry serves several vectors
Vascular risk	Higher — sharp tip can enter a vessel	Lower — blunt tip tends to push vessels aside
Trade-off	Precision, at the cost of more entry points	Coverage and safety, at the cost of fine precision

A cannula is not automatically safer in every plane — it is safer in the plane it was designed for. The tool follows the layer, not the fashion.

The five layers of the face, from skin to bone. Every injection decision begins with knowing which of these planes the needle tip is in.

Here is something nobody tells you on a beginner course. Every kind of doctor has their own anatomy, and it is shaped entirely by what their tools let them see.

A surgeon has surgical anatomy. He opens the face up and looks at it. When an artery does not run where the textbook promised — and it often does not — he sees the real one, right there, and works around it. A radiologist has radiological anatomy. She images the thing first, then acts on the picture.

You have neither. You work blind. Your needle goes under the skin and you cannot see a single thing it is near. And here is the part that took me a blue lip to understand: it does not actually matter how many diagrams of the facial artery you have memorised, because you cannot know which version of that artery is under *this* patient's skin, on *this* side of *this* face, at the moment your needle is in it. The one thing you need to know is the one thing you cannot see.

So we give up trying to see the map, and we control the one thing we actually can: depth. Not where the artery runs — you will never know that in time — but which layer your needle tip is resting in. That you can control, every single time, and it is reproducible. This is the whole of it, and it is why I say it to every practitioner I train: **for an injector, anatomy is depth.** The only question that matters when the plunger is under your thumb is *what layer am I in, and with what tool.*

There are three such layers for filler. (Botox is a different game — it goes into muscle, and muscle is a different logic, so leave it out of this model entirely.) Each of the three is defined not just by its depth but by the tool that belongs in it. That pairing — layer and tool — is the model.

Layer 1 — The needle, in the skin (the canvas)

The dermis is the skin itself. It is tough, fibrous, and highly resistant to expansion.

When you inject into the dermis, you are not trying to change the shape of the face. You are trying to change the quality of the canvas. This is where you place skin boosters, polynucleotides, and very fine, soft fillers to hydrate the tissue and smooth out superficial lines (like smoker's lines or necklace lines on the neck).

Injecting into the dermis requires precision. The needle goes in shallow, almost parallel to the skin. You should see the grey shadow of the metal through

the tissue. If you inject too deep, you waste the product in the fat. If you inject too superficially, you create white bumps (blanching) that can take weeks to resolve.

Layer 2 — The cannula, in the subcutaneous (the cushion)

Immediately below the dermis lies the superficial subcutaneous fat. This is the cushion of the face. It is soft, vascular, and highly compliant.

When we age, we lose volume in this layer. The cushion deflates. The skin above it begins to sag, creating shadows, folds, and the classic “tired” appearance.

This is the primary layer for volumisation and contouring. This is where we place filler to soften the nasolabial folds, plump the cheeks, and restore the youthful convexity of the face.

Because this layer is highly vascular, it is also the most dangerous layer for needles. This is the domain of the blunt-tipped cannula. A cannula can glide through the soft fat, pushing vessels aside rather than piercing them. If you are volumising the midface in the subcutaneous layer with a sharp needle, you are playing Russian roulette with the patient’s facial artery.

Layer 3 — The needle, on the periosteum (the foundation)

Deep beneath the fat and the muscle lies the bone. The periosteum is the tough, fibrous membrane that covers the bone surface.

When we age, our facial skeleton literally shrinks. The eye sockets widen, the cheekbones recede, and the jawline loses its sharp angle. The foundation of the house begins to crumble.

To fix this, we must go deep. We take a sharp needle, pass straight through the skin, the fat, and the muscle, until we tap hard bone. We then deposit a small, highly cohesive (stiff) bolus of filler directly onto the periosteum.

This is structural work. We are not filling lines; we are rebuilding the skeleton. By placing product on the bone, we create a pillar of support that lifts all the soft tissues above it. This is how you project a weak chin, define a jawline, or lift a descending cheek without making the face look fat or puffy.

The layers do not care where you are on the face

Here is the elegant part, and the reason this model is worth more than a shelf of regional protocols. A filler behaves according to the *layer*, not the *location*. A cannula in the subcutaneous plane of the nose is the same manoeuvre as a cannula in the subcutaneous plane of the cheek, or the chin, or the temple. A needle to bone at the cheekbone follows the same logic as a needle to bone at the chin. You do not need a separate mental script for every region of the face. You need to decide which of the three layers the correction belongs in, and the tool, the product, and the technique all follow from that. Learn three layers, not fifty regions.

A word on the deep fat — deliberately left out

The anatomically minded reader will notice that between the subcutaneous cushion and the bone sit the deep fat compartments — the deep medial cheek fat, the SOOF, and the rest — and that I have not given them a layer. That is on purpose, and it is not an oversight, so let me be straight about it.

The deep fat is real, it ages, and there is a whole literature on volumising it. I am not going to argue that it does not matter. I am going to make a narrower and more useful point: it is not a *primary* target, and it is out of scope for this three-layer model. In nearly twenty years I have found that working the two ends of the stack — projection on the bone below, contour in the subcutaneous above — corrects the visible result in the enormous majority of aesthetic patients, without me ever needing to chase a deep fat pad. A small deposit on the periosteum changes the geometry of everything sitting on top of it; you rarely need to refill the pad itself. There are exceptions — a handful of faces where a deep deficit genuinely dominates — and those belong to more advanced or surgical hands. But as a default, the deep fat is a place I choose not to go, and this book is not going to teach you to go there. That is a longer argument, and I make it in full in the companion journal paper. Here, it is simply out of scope.

One refinement, because “out of scope” is easy to misread as “I do not know it is there.” Deep fat is best understood not as a fourth mandatory plane but as a **selective anatomical target sitting inside the structural category**. The three planes stay as they are — skin with a needle, subcutaneous contour with a cannula, structural projection on the periosteum when projection is genuinely required — and deep fat becomes an option *within* that third category, chosen on diagnosis, not on principle.

It is also worth saying that “deep fat” is not one homogeneous injectable layer. In the cheek alone the deep targets described include the medial and lateral SOOF, the deep medial cheek fat, the deeper preperiosteal spaces, and the buccal fat pad — which is deeper again and is not something I would ever treat as an ordinary filler compartment. So when someone says “I inject deep fat,” they have not actually told you anything. The useful sentence names **which compartment or space, and why.**

The Golden Rule of Layers

The most common mistake in aesthetic medicine — the reason why so many people walk around looking overfilled, unnatural, and bizarre — is a failure to respect the layers.

Injectors try to solve a Layer 3 problem (bone loss) by pumping massive amounts of filler into Layer 2 (the fat). They try to lift a sagging face by inflating the cushion instead of rebuilding the foundation. The result is the dreaded “pillow face” — heavy, distorted, and distinctly unnatural.

Conversely, injectors try to solve a Layer 1 problem (fine lines and poor skin quality) by injecting thick filler into Layer 2, hoping it will stretch the skin smooth. It doesn't. It just makes the patient look swollen.

The rule is absolute: - Treat the canvas in the dermis. - Treat the cushion in the fat. - Treat the foundation on the bone.

The nose is the clearest demonstration of the Three-Layer Theory in action. A patient comes in with a dorsal hump. You are not going to the epidermis. You are not going to the dermis. You are going to the subcutaneous layer — or, if the hump is severe, to the periosteum itself. You use a cannula in the subcutaneous plane to camouflage the hump by building the bridge above and below it. Or you use a needle on the periosteum to place a small bolus of high-G-prime filler at the radix, lifting the starting point of the nose so the hump reads as a gentle slope rather than a protrusion.

Same nose. Same complaint. Different layers. Different tools. Different results.

And there is a fourth move that most injectors forget entirely: the dermis itself. Once the structural work is done — the hump camouflaged, the radix lifted — tiny linear threads of soft filler injected intradermally along the dorsum can add beautiful finishing touches. Contour definition in the line.

A sharpness that the subcutaneous layer alone cannot deliver. It is the difference between a good nose job and a great one.

The scaffold is the cannula and the needle on bone. The finishing is the dermis.

The Three-Cell Theory tells you what the skin is made of and how to keep it healthy. The Three-Layer Theory tells you where to inject and why. Together, they are the complete anatomical framework for aesthetic medicine. Learn them, or put the syringe down.

And hold on to the sentence underneath both, because it is the spine of everything that follows in this book: *anatomical ageing is not a treatment target*. The face at sixty is not the face at twenty with volume removed, and your job is not to put every lost millilitre back. Your job is to manage the visible consequences of ageing — regenerate the envelope, support the contour, project the structure — in the right layer and, as we will see later, in the right order. The full argument is set out in the companion paper in *Aesthetic Intelligence*; the working version is what you are reading.

CHAPTER 3

The Factory Floor — The Deep Dermis and the Cell That Builds You

If we are going to understand the skin, we must start from the bottom and work our way up. You do not build a house by inspecting the roof tiles. You start with the foundation.

In the skin, that foundation is the deep reticular dermis.

If the superficial dermis is the factory floor where the delicate work happens, the deep dermis is the structural steel holding the entire building together. It is thick, it is tough, and it absolutely does not care about your expensive face creams. Nothing you rub on the surface of the skin is getting down here.

You can spend five hundred pounds on a miracle serum, and the deep dermis will not even register its existence.

This is the layer that gives the skin its mechanical strength and its ability to resist folding. When the reticular dermis is intact, thick, and healthy, the skin holds its shape against gravity and expression. When it weakens, everything above it starts collapsing in slow motion.

Here live the lines that stay even when you stop smiling: - Deep crow's feet that carve into the cheek - Accordion lines on the lower face - Forehead grooving that looks like it was cut with a chisel - Lower-face laxity and general structural failure This layer ages not because the patient smiled too much, but because the fibroblasts got bored, the Matrix Metalloproteinases (MMPs) got excited, and the collagen cross-linking stopped playing nicely. The reticular dermis does not whisper its decline. It announces it.

The Architect: The Fibroblast

The deep dermis is ruled by a single, critical cell: the fibroblast.

I call the fibroblast the Architect. Its sole purpose in life is to manufacture the extracellular matrix — the dense web of collagen, elastin, and hyaluronic acid that gives the skin its bounce and resilience. In your twenties, your fibroblasts are working overtime. They are enthusiastic, highly productive, and constantly repairing micro-damage. If you fold the skin, it snaps right back.

But as we age, the fibroblasts undergo senescence. They become sluggish. They stop producing high-quality Type I collagen and start producing disorganized, weaker collagen. Worse, the environment around them becomes hostile. The enzymes that break down collagen (MMPs) become overactive, outstripping the fibroblasts' ability to rebuild. The structural steel begins to rust.

If you want to treat the deep dermis, you have exactly one objective: you must wake the fibroblast up. You must convince it that it is young again, or you must traumatise it just enough to trigger a wound-healing response.

Deep Dermal Micro-Filler — Structural Stitching

This is your most elegant tool for repairing real dermal architecture. In this plane, filler is not “filler.” You are not inflating a balloon. It is reinforcement.

You insert tiny micro-boluses — less than 0.01 ml — with a remarkably steady hand and a 30G or 32G needle. The syringe barely moves a notch; you almost feel like you have not injected anything at all. But the skin tells you the truth: a soft, subtle plump, a tiny bleb — like a Botox bleb around the crow's feet, but even smaller and vastly more disciplined.

You are not adding volume. You are stitching the collagen bed, reinforcing the dermal fabric so the skin can finally lie smooth again. You are giving the fibroblasts a scaffold to wrap around.

Two visual cues every injector must know when working in the deep dermis:

- If you can see the silver of your needle shining clearly through the skin, you are too superficial. You are in the papillary dermis.
- If the needle disappears completely and you feel no resistance, you have fallen through the floor. You are in the subcutaneous fat. You are exploring uninvited neighbourhoods, and you are wasting expensive product.

Technique — The Layer That Rewards Respect

Entering the deep dermis requires discipline. Entering at too steep an angle (45 degrees) makes you fall deeper into the fat before you even realise it.

Parallel entry is safer. Bending the needle slightly (20–30 degrees) improves control and allows you to keep your hand out of the patient's line of sight. Landing the syringe softly — like a plane touching down on a runway — lets you glide into the reticular layer without unintentional depth.

The dermis talks to you if you watch it carefully: - Icy-blue blanching = too superficial. You are going to leave a lump. - A dull, greyish-silver shimmer = perfect plane. - Disappearing needle = too deep. Pull back.

This layer is one of the safest in the face, as long as your eyes stay locked on the metal. There are no major vessels here. There are no motor nerves to paralyze. But it is technically demanding.

A note on the periorbital area: When you enter at 45 degrees here, you need to be extremely careful with depth. In the periorbital area, going a touch deeper is actually safer than staying too superficial. Because the skin here is so paper-thin that a superficial pass risks visible product, the dreaded Tyndall effect (a bluish hue), and those little ridges that betray your work faster than any complication.

A hair deeper sits you in a smoother, more forgiving plane.

HIFU and RF — Heating the Deep Dermis

When we want to stimulate the deep dermis from the outside, we don't use creams. We use heat. High-Intensity Focused Ultrasound (HIFU) and Radiofrequency (RF).

The rationale is brutal but highly effective: controlled thermal injury. You deliver intense heat deep into the tissue, bypassing the epidermis entirely. The existing collagen fibres contract immediately (giving a temporary tightening effect), and the fibroblasts absolutely freak out.

They think the skin has been severely damaged, so they launch a massive, coordinated wound-healing response. They spend the next three to six months laying down fresh, tight, highly organised collagen. Notice the timeline. Three to six months.

The patient will leave the clinic feeling tight and looking slightly flushed. That is just oedema. It is swelling. It will fade in two days, and they will look exactly like they did before they walked in. The real result will not be visible

until the season changes.

If you do not manage this expectation brutally and clearly during the consultation, you will be managing a very angry, very disappointed patient at the four-week review.

The No-Lifting Reminder

HIFU and RF will thicken the deep dermis. They will improve the structural integrity of the skin wrapper. They will make the skin feel firmer to the touch.

They will not lift a descended fat pad. They will not replace lost bone. They will not restore the shape of a deflated midface.

Back to the apple analogy: These devices polish and thicken the skin of the apple. They do not replace the rotting flesh inside.

This is not a criticism of the technology. HIFU and RF are genuinely powerful tools when used for the right indication — skin quality, dermal density, and fine surface laxity. The wrong indication is a patient with massive volume loss who actually needs structural filler, but is being sold a course of expensive device treatments because the clinic needs to pay off the lease on the machine.

Know the difference. Your patients are paying you to know it.



CHAPTER 4

Waking the Fibroblast — Boosters, Polynucleotides, Microneedling

She came in with a question already prepared, which is usually the first clue.

“Do you think polynucleotides would work for me?”

She had hollow tear troughs, a heavy lower face, grade one to two jowls, and corners of the mouth that had begun their long turn downwards.

She had also had three sessions of polynucleotides elsewhere, and before that a course of something similar, and she was asking me whether she should have more.

I asked her whether that was the question she had put to the other practitioners. She said yes. All of them.

And there it was. She had not been consulted by four practitioners. She had consulted herself four times, out loud, in four different rooms, and four people had agreed with her — because agreeing is quick, and because the treatment she was proposing happened to cost more than the one she needed.

What she required was volume under the eye, support in the cheek, something along the jaw, and above all something at the corners of the mouth, which was the finding nobody had mentioned to her in two years. One of those treatments addresses the quality of the skin. The other addresses the tissue beneath it that has stopped holding the skin up. They are not competing options. They answer different questions, and she had been sold the wrong answer four times.

Polynucleotides are an excellent tool. That is precisely why they get misused. Nothing in this chapter is a substitute for looking at the face in front of you and deciding what is actually broken.

If the deep dermis is the structural steel, how do we actively repair it when it begins to rust? We have established that the fibroblast is the architect, and that with age, it goes to sleep.

The entire goal of this chapter is answering one question: How do we violently wake it up?

We have three primary tools at our disposal that bridge the gap between skincare and structural filler. These are not volumizers. They will not change the shape of a face. They are biological signals. They are instructions delivered directly to the factory floor.

Skin Boosters: The Hydration Flood

Traditional dermal fillers use cross-linked hyaluronic acid (HA). The cross-linking acts like microscopic staples, binding the HA molecules together so they form a stiff gel that can lift tissue and hold its shape for a year or more.

Skin boosters are different. They use non-cross-linked (or very lightly cross-linked) hyaluronic acid. Without those staples, the HA behaves like a liquid, not a gel. It spreads. It diffuses. It floods the dermis.

When you inject a skin booster into the superficial or mid-dermis, it acts as an internal moisturizer. HA is a humectant; it draws water to itself. By placing it directly into the dermis, you bypass the epidermal barrier entirely. The result is skin that looks impossibly hydrated, plump, and reflective.

The fine, crepey lines that look like tissue paper disappear because the tissue beneath them has been saturated with water.

But skin boosters do something else, something arguably more important. The sudden influx of HA stretches the fibroblasts mechanically. This physical stretching is a biological signal. It tricks the fibroblast into thinking the extracellular matrix is under tension, which stimulates it to produce fresh collagen and elastin.

You are not just hydrating the skin; you are forcing the factory to turn the lights back on.

Polynucleotides: The Blueprint Repair

If skin boosters are the hydration flood, polynucleotides are the genetic repair crew. This is the most significant advancement in regenerative aesthetics in the last decade.

Polynucleotides are highly purified fragments of DNA (typically derived from salmon or trout sperm, a fact you may or may not want to share with your patients depending on their sense of humor). When injected into the dermis, they do not hydrate by drawing water. They act on a cellular level.

They bind to specific receptors on the fibroblast, signaling it to dramatically increase its metabolic activity. They scavenge free radicals, reducing the chronic inflammation that accelerates ageing. They promote angiogenesis — the formation of new micro-blood vessels — improving blood flow and oxygen delivery to the tissue.

Skin boosters give you an immediate, beautiful glow that lasts a few months. Polynucleotides give you a fundamental change in tissue quality that takes weeks to appear but alters the trajectory of the skin's ageing process. They are the ultimate long game.

They are particularly brilliant for the under-eye area, where traditional tear trough filler often looks heavy or causes chronic swelling. Polynucleotides thicken the paper-thin skin beneath the eye, hiding the dark muscle beneath it without adding bulk.

Microneedling: The Controlled Trauma

Sometimes, you don't need to deliver a molecule to wake the fibroblast. Sometimes, you just need to stab it.

Microneedling (collagen induction therapy) is the most primitive, brutal, and brilliantly effective treatment we have. Using a device with multiple fine needles, we create thousands of microscopic puncture wounds in the epidermis and dermis.

We are intentionally causing trauma.

When the skin is punctured, it bleeds microscopically. Platelets rush to the scene and release growth factors. The fibroblasts, sensing a catastrophic breach in the hull, go into overdrive. They rush to the site of the injury and begin laying down massive amounts of fresh Type III collagen to heal the wounds. Over the next few months, this Type III collagen is remodeled into strong, permanent Type I collagen.

Microneedling is not a relaxing spa treatment. It is a controlled biological assault. But it works. It thickens the dermis, smooths acne scarring, and tightens mild laxity better than almost any laser on the market, with a fraction of the risk

of post-inflammatory hyperpigmentation.

The Protocol of Stimulation

None of these treatments work in a single session. You are trying to change biological behaviour, and biology takes time.

If a patient wants to look good for a wedding on Saturday, you give them a skin booster on Wednesday. If a patient wants to fundamentally change the quality, thickness, and resilience of their skin for the next two years, you put them on a course of polynucleotides and microneedling, spaced four weeks apart, and you tell them to be patient. You are the architect. You have to decide whether the house needs a quick coat of paint, or a structural rebuild.



CHAPTER 5

The Superficial Dermis — The Glow Factory

The house in Chelsea had a lift.

Not a stairlift. A proper lift, brass concertina gate, installed inside a private home so that nobody living there would ever have to negotiate a staircase again. I remember thinking it was the most expensive thing I had ever stood next to, and I had not yet taken my coat off.

I was doing house calls in those days because I could not afford a clinic. This sounds glamorous when I say it now. At the time it meant carrying my kit across London in a holdall and hoping nobody asked what was inside it.

She wanted her frown done. Fifteen minutes of work. She took me up to a dressing room roughly the size of my flat, sat down at a mirror ringed with bulbs like something off a film set, and while I was drawing up, I looked at the shelf beside her.

There was a jar on it. White, heavy, no branding I could read from where I stood. Four or five others beside it. The whole arrangement had the quiet confidence of objects that have never had to mention their price.

I asked what it was. Not clinically. The way you ask about a painting.

“That’s La Mer.”

She said it the way you would say water. No emphasis, no explanation.

It did not occur to her that the doctor standing in her dressing room holding a syringe might not know.

I nodded, as though I had merely been confirming.

Nobody in my family had ever bought a cream that came in a jar.

Skincare, where I grew up, was soap. If your skin was dry you used more soap and then complained about it. I had six years of medical school behind me, a surgical rotation, and a stint in a research lab at Johns Hopkins, and I could not have told you what was in that jar or whether it did anything at all.

I looked it up that night. North of two hundred pounds for sixty millilitres. Fermented sea kelp, largely, suspended in petrolatum.

And here is the part that took me considerably longer to accept than the price did.

She was not being foolish. She was being more sensible than I was. Her instinct — that skin is something you maintain daily, deliberately, for decades, rather than something you repair once it has failed — was closer to correct than anything in my training. My training had given me nothing on skin. Not one lecture. I could close bowel. I could not have named a single molecule that reliably improves the quality of human skin.

There was one. There had been one for fifty years.

Nobody had thought to teach it to me.

So I went looking.

Moving up from the deep reticular dermis, we arrive at the superficial papillary dermis.

If the deep dermis is the structural steel, the superficial dermis is the finishing plaster. This is the layer that dictates how light reflects off the face. It is the layer of texture, of fine lines, of pores, and of that elusive, highly marketable concept we call “glow.”

The papillary dermis is thin. It interlocks with the epidermis above it through microscopic, finger-like projections called rete ridges. These ridges increase the surface area between the two layers, ensuring that the epidermis (which has no blood supply of its own) receives oxygen and nutrients from the rich capillary loops in the papillary dermis.

In youthful skin, these rete ridges are deep and pronounced. The connection is strong. The skin is bouncy. But as we age, these ridges flatten out. The connection weakens. The epidermis receives less nutrition, and the skin begins to look dull, fragile, and crepey. It literally loses its grip on itself.

The Fine Line Battlefield

This is the layer where dynamic lines — the lines of expression — eventually become static lines.

When you smile, the underlying muscle contracts, folding the skin. In your twenties, the superficial dermis is packed with healthy elastin and collagen, so when you stop smiling, the skin snaps back perfectly smooth. But over decades of repeated folding, combined with UV damage and the slow decline of the fibroblast, the dermal matrix at the base of that fold fractures.

The skin loses its memory. The fold becomes a permanent crease.

This is why Botox is preventative. By stopping the muscle from folding the skin, you prevent the superficial dermis from fracturing. But once the fracture is there, Botox alone will not erase it. You have to repair the dermis.

Mesotherapy and the Art of the Bleb

We have already discussed skin boosters in the context of the deep dermis, but the superficial dermis is where mesotherapy truly shines.

Mesotherapy is the technique of delivering micro-droplets of non-cross-linked hyaluronic acid, vitamins, amino acids, and peptides directly into the papillary dermis. You are not injecting deep. You are injecting so superficially that the product forms tiny, visible blebs on the surface of the skin, like mosquito bites.

These blebs look alarming to the uninitiated patient, but they are exactly what you want. They prove you are in the correct plane. Over the next 24 to 48 hours, the product diffuses evenly through the superficial dermis, saturating the tissue with hydration and nutrients.

This is how you treat the smoker's lines above the lip without making the lip look like a duck. This is how you treat the fine, crepey skin of the lower cheek. You are not filling; you are flooding the factory floor with raw materials.

The Limit of the Needle

There is a limit to what injectables can achieve in the superficial dermis. You can hydrate it. You can stimulate it. But if the surface above it — the epidermis — is thick, dead, and poorly organised, the skin will still look terrible.

You cannot inject your way out of bad skin quality. To truly change the appearance of the superficial dermis, you must treat the layer above it. You

must strip away the dead wood.

The Retinoid Revolution

The most powerful tool we have for the superficial dermis is not an injectable. It is a topical. Tretinoin — the prescription-strength form of vitamin A — is the most evidence-based anti-ageing molecule in existence. It has been studied for over fifty years. Its mechanism is understood at the molecular level. And it works.

Tretinoin binds to retinoic acid receptors in the keratinocytes and fibroblasts. It accelerates keratinocyte turnover, thinning the thickened stratum corneum and revealing the fresh, light-reflecting cells beneath. It simultaneously stimulates fibroblast activity, increasing collagen synthesis and thickening the dermis. It inhibits the matrix metalloproteinases that break collagen down. It reduces the formation of abnormal melanin clusters.

In short, it addresses all three cells simultaneously.

The catch is that it takes time. The first four to six weeks are brutal. The skin purges. It peels. It turns red. Patients call you in a panic. You have to prepare them for this, or they will stop using it and blame you for ruining their skin.

Tell them: the ugliness is the treatment working. The old skin is shedding. The new skin is being built. In three months, they will not recognise themselves.

Retinoids are not glamorous. They do not generate the same revenue as a syringe of filler. But they are the foundation of every serious skin programme. If your patients are not on a retinoid, you are not doing your job properly.

Sunscreen: The Non-Negotiable

Every conversation about the superficial dermis must end here. Sunscreen is not optional. It is not a lifestyle choice. It is a clinical prescription.

UV radiation is the single greatest driver of superficial dermal damage. It directly degrades collagen through the activation of matrix metalloproteinases. It triggers melanocyte panic, causing pigmentation. It damages the keratinocyte DNA, accelerating ageing and increasing the risk of malignancy.

Every treatment you perform — every peel, every laser, every skin booster — is partially undone by a patient who walks out of your clinic and spends the afternoon in the sun without protection. You are pouring water into a bucket

with a hole in the bottom.

Broad-spectrum SPF 50, applied every morning, is the most cost-effective anti-ageing intervention that exists. It costs less than a cup of coffee per day. It prevents more damage than any treatment you can offer in your clinic. Prescribe it like a medication. Follow up on compliance like a medication. If a patient is not using it, nothing else you do will last.

But before we reach the surface, there is one cell still unaccounted for. It sits at the very floor of the layer above us, it decides what colour all of this will be, and it is the least forgiving cell you will ever provoke.



CHAPTER 6

The Temperamental Cell — Melanocytes and Pigment

Before we deal with the dead cells on the surface, we must deal with the most volatile, unpredictable, and dangerous cell in the entire skin ecosystem.

The melanocyte.

The melanocyte lives at the very bottom of the epidermis, nestled among the basal stem cells. It looks like an octopus, with long, branching arms (dendrites) that reach upward, wrapping around the surrounding keratinocytes.

Its job is to produce melanin — the pigment that gives skin its colour.

But melanin is not just cosmetic. It is a biological shield. When UV radiation from the sun hits the skin, it damages the DNA in the cells. The melanocyte senses this damage and goes to work. It manufactures packets of melanin (melanosomes) and shoots them up through its tentacles into the keratinocytes.

The keratinocytes take these pigment packets and arrange them over their nuclei, like tiny microscopic umbrellas, protecting their DNA from further radiation.

A tan is not a sign of health. A tan is the visible evidence of a desperate biological defence mechanism. It is the skin screaming that its DNA is under attack.

The Panicked Cell

The problem with the melanocyte is that it is highly reactive and has a terrible memory.

In youthful, healthy skin, the melanocyte produces pigment evenly and only when necessary. But as we age, and as the skin accumulates decades of UV

damage, hormonal fluctuations (pregnancy, oral contraceptives), and physical trauma, the melanocyte becomes dysfunctional.

It panics.

It stops producing pigment evenly. It starts dumping massive amounts of melanin into localized areas, even when there is no sun exposure. It stays permanently switched “on.”

This results in the aesthetic nightmare of hyperpigmentation: - **Sun spots (lentigines):** Localized areas of dense pigment caused by decades of UV exposure. - **Post-Inflammatory Hyperpigmentation (PIH):** A dark mark left behind after trauma, like a severe acne breakout, a burn, or a badly executed laser treatment. The trauma triggered the melanocyte to flood the area with pigment as a defence mechanism. - **Melasma:** The boss-level boss of pigmentation. A chronic, hormonally driven condition where large, symmetrical patches of pigment appear on the cheeks, forehead, and upper lip. Heat, sun, and hormones all trigger it. It is notoriously difficult to treat and impossible to cure.

How to Negotiate with a Melanocyte

You cannot bully a melanocyte. If you attack it aggressively — with a harsh laser, an overly aggressive chemical peel, or aggressive microneedling — it will perceive the treatment as trauma. And what does a melanocyte do when traumatised? It produces more pigment.

You try to burn off a sun spot, and you end up giving the patient a massive patch of PIH that is ten times worse than the original problem. Treating pigmentation requires diplomacy. You must calm the cell down before you attempt to remove the pigment it has already produced.

1. The Blockade (Sunscreen) If the patient is not wearing a broad-spectrum SPF 50 every single day, rain or shine, do not treat their pigmentation. You are wasting their money and your time. You cannot empty a sinking boat if you don't plug the hole first.

2. The Chemical Muzzle (Tyrosinase Inhibitors) Melanin is manufactured through a chemical pathway. The key enzyme in this pathway is called tyrosinase. If you block tyrosinase, the melanocyte cannot make pigment, no matter how panicked it is.

We use topical agents like Hydroquinone (the gold standard, but requires careful medical management), Kojic Acid, Arbutin, and Vitamin C to muzzle the enzyme. You must put the melanocyte to sleep *before* you strip the skin.

3. The Eviction (Exfoliation) Once the factory has been shut down, you can safely remove the pigment that is already trapped in the epidermis. This is where we use chemical peels and retinoids to accelerate cell turnover, pushing the stained keratinocytes up and off the face.

Treating pigment is not a procedure. It is a protocol. It requires patience, medical-grade skincare, and absolute patient compliance. If they will not commit to the protocol, refuse the treatment.

Melasma: The Special Case

Melasma deserves its own section because it is the most common pigmentation condition you will encounter, and the one most likely to end in a complaint if you handle it badly.

Melasma is not a simple pigmentation problem. It is a chronic, relapsing condition driven by a combination of UV exposure, heat, hormonal influence (particularly oestrogen), and genetic predisposition. It is far more common in Fitzpatrick Types III–V. It is more common in women. It is more common in people who live in hot, sunny climates.

The melanocytes in melasma-affected skin are not just overactive. They are structurally abnormal. They have more dendrites. They produce more melanosomes. They are more sensitive to UV and heat. You cannot simply strip the pigment and expect it to stay gone. The melanocytes will repigment the area within weeks if the triggers are not controlled.

The treatment protocol for melasma is as follows:

Phase 1: Stabilisation (minimum 8 weeks) Strict SPF 50+ every morning, reapplied every two hours if outdoors. A broad-spectrum sunscreen that blocks both UVA and UVB, and ideally contains a physical blocker (zinc oxide or titanium dioxide) for heat protection. A tyrosinase inhibitor — hydroquinone 4% is the gold standard, but azelaic acid 15–20% is a good alternative for patients who cannot tolerate hydroquinone.

A retinoid to accelerate turnover and help the inhibitor penetrate. No heat exposure. No saunas, no hot yoga, no direct sun.

Phase 2: Lightening (8–16 weeks) Once the melanocytes are stabilised and the triggers are controlled, you can begin to lighten the existing pigment. Superficial chemical peels — glycolic acid, lactic acid, or a low-concentration TCA — can be introduced carefully, starting with the lowest concentration and building up.

The key word is carefully. If the patient develops any redness, inflammation, or PIH after a peel, stop. The melanocytes have been triggered. Go back to stabilisation.

Phase 3: Maintenance (indefinitely) Melasma does not go away. It goes into remission. The patient must maintain their SPF protocol, their tyrosinase inhibitor, and their retinoid indefinitely. If they stop, it comes back. This is not a failure of treatment. It is the nature of the condition. Tell your patients this at the start. Set the expectation. Melasma is a chronic condition that you manage, not cure. If they understand that, they will be compliant. If they expect a cure, they will be disappointed.

The Melanocyte and Laser: A Cautionary Note

Lasers and intense pulsed light (IPL) are frequently marketed as treatments for pigmentation. For simple, discrete sun spots (lentigines) in Fitzpatrick Types I–III, they can be highly effective. For melasma, they are a minefield.

The heat generated by laser and IPL treatments is a direct trigger for melanocyte activation. Even a well-calibrated, appropriately chosen laser can cause a melasma flare in susceptible patients. The initial result may look excellent — the pigment appears to lift — but within weeks, the melanocytes have been triggered by the thermal injury and the pigment returns, often darker and more extensive than before.

If you are going to use laser or IPL for pigmentation, know your patient's Fitzpatrick type. Know their history of melasma. Patch test. Start conservatively. And never treat active melasma with a heat-based device without a prolonged period of pre-treatment conditioning first.

The melanocyte is the most reactive cell in the skin. It does not forgive overconfidence.

And it does its work at the floor of the layer we are about to enter.

CHAPTER 7

The Epidermis — The Surface of Youth

We have finally reached the roof. The epidermis.

The epidermis is the only part of the skin that your patient actually sees in the mirror. It is the only part of the skin they can touch. And ironically, it is the part of the skin that injectors ignore the most.

Injectors love the deep spaces. We love the bone, the fat, the structural ligaments. We view the epidermis merely as the annoying barrier we have to push our needle through to get to the “real” work.

This is a massive clinical blind spot.

You can rebuild a patient’s jawline with 4 ml of high-G-prime filler. You can lift their cheeks perfectly. You can erase their shadows. But if their epidermis is dull, thickened, pigmented, and rough, they will still look old. They will just look like an old person with a very sharp jawline.

Youth is not just shape. Youth is light reflection. And light reflection is entirely dictated by the epidermis.

The Wall

The epidermis is a stratified squamous epithelium. It is a brick wall, roughly 0.1 mm thick on the face. It has no blood vessels. It relies entirely on the dermis below it for survival.

Its primary function is to keep water inside the body and keep the rest of the world (bacteria, pollution, UV radiation) out. It is a hostile, defensive layer.

The bottom layer of the epidermis (the stratum basale) is where the action happens. This is where the stem cells live, constantly dividing to create new keratinocytes (the bricks). As these new cells are born, they push the older cells

upward.

As the cells move up, they change. They flatten out. They lose their nucleus. They become packed with keratin, a tough, fibrous protein. By the time they reach the very top layer (the stratum corneum), they are completely dead. They are essentially microscopic, overlapping armor plates.

Eventually, these dead cells break their bonds and flake off invisibly into the air. This process is called desquamation.

The Speed of Light

In a healthy 20-year-old, the journey from the birth of a cell at the basal layer to its death and shedding at the surface takes about 28 days. The wall is constantly being rebuilt and refreshed. Because the dead cells are shedding efficiently, the surface of the skin is smooth. When light hits a smooth surface, it reflects evenly. The skin looks bright. It glows. In a 50-year-old, this process slows down dramatically. The journey can take 45 to 60 days.

The dead cells at the surface do not shed efficiently. They stick together. They pile up unevenly. The stratum corneum becomes thick, rough, and disorganized. When light hits this rough surface, it scatters in a thousand different directions. The skin looks dull, grey, and lifeless.

Furthermore, this thickened layer of dead cells acts like a sponge, absorbing light rather than reflecting it, making pigmentation and fine lines look far worse than they actually are.

If you want to make a face look younger instantly, you do not reach for a syringe. You strip the roof. You force the epidermis to accelerate its turnover rate. You make it behave like it is 20 years old again.

And to do that, you have to understand the two cells that control this layer: the Melanocyte and the Keratinocyte.

The Barrier Function: What Your Patients Are Destroying

The stratum corneum is not just a dead layer of cells. It is a carefully engineered waterproof barrier. Between the dead keratinocytes, the skin secretes a complex mixture of ceramides, fatty acids, and cholesterol — essentially, a biological mortar between the bricks. This lipid matrix seals the wall, preventing water from evaporating out of the body and preventing irritants from penetrating in.

This is the skin barrier. And your patients are systematically destroying it.

Over-exfoliation. Harsh cleansers. Alcohol-based toners. Aggressive scrubs. The entire “skincare” industry has spent decades convincing people that more is more, that the path to glowing skin involves stripping the barrier down to nothing and rebuilding it with seventeen different serums.

The result is a generation of patients with chronically compromised barriers. Their skin is red, reactive, sensitive, and perpetually inflamed. They have stripped out the lipid mortar. The bricks are loose. Water is escaping. Irritants are getting in. Their skin is in a permanent state of low-grade emergency.

When a patient tells you their skin is “sensitive,” what they usually mean is that their barrier is broken. Your first job is not to inject them. Your first job is to repair the wall. Ceramide-based moisturisers. Gentle, pH-balanced cleansers. A moratorium on the twelve-step routine. Let the skin breathe and heal.

You cannot build on a broken foundation. Fix the barrier first.

The Fitzpatrick Scale: Knowing Your Patient’s Skin

Not all epidermises are created equal. The Fitzpatrick scale classifies skin into six types based on its response to UV exposure, from Type I (always burns, never tans — the classic pale Irish complexion) to Type VI (never burns, deeply pigmented skin).

This classification is clinically critical because it predicts risk. Fitzpatrick Types I and II are at the highest risk of UV-induced damage, photoageing, and skin cancer, but they are at the lowest risk of post-inflammatory hyperpigmentation (PIH) from treatments.

Fitzpatrick Types IV, V, and VI are at the highest risk of PIH — meaning that any treatment that causes inflammation (peels, lasers, microneedling) can trigger a melanocyte panic response that leaves the patient with dark marks that are harder to treat than the original problem.

Know your patient’s Fitzpatrick type before you do anything. Adjust your peel depth, your laser settings, and your microneedling intensity accordingly. A TCA peel that gives a Type II patient a beautiful result will give a Type V patient a post-inflammatory hyperpigmentation disaster.

The epidermis does not forgive ignorance.

CHAPTER 8

Keratinocytes, Chemical Peels, and Lazy Skin Syndrome

Before I criticise anybody's routine, I should declare my own history.

I grew up with soap. One bar. Whole body, hair included if the morning was going badly. There was also shampoo, which I understood to be soap that had been given a job title. That was the entire system, it cost almost nothing, and I was perfectly happy inside it.

Marriage upgraded me to shower gel.

I want to be measured about shower gel. It is a fundamentally hostile product. It arrives in a bottle designed to dispense four times more than any human requires, roughly half of it goes directly onto the floor, and it has never once foamed the way a bar of soap foams. You end up applying more to compensate, which is exactly what the bottle was designed to make you do.

Then came conditioner, which turned out to have terms and conditions. It has to sit on your head for four or five minutes. Not thirty seconds.

Minutes — during which you stand there, wet, doing nothing, contemplating the decisions that brought you to this point.

In roughly a decade I went from one product to five, and my hair and skin were not noticeably better at the end of it.

I mention this because I am about to spend a chapter explaining why your patients' twelve-step routines are destroying their skin, and it seems only fair to admit that I did not arrive at three products through wisdom. I arrived at one through poverty and worked upwards like everybody else. The difference is that I eventually learned what each of them actually does, which is the entire subject of this chapter.

More is not more. More is usually just more.

We have reached the bricks. The keratinocytes.

As we discussed, the ageing epidermis is characterised by a slow turnover rate. The dead keratinocytes pile up on the surface, creating a thick, dull, rough layer (the stratum corneum) that scatters light and makes the skin look exhausted.

If you want to change the surface of the face, you must force the keratinocytes to move faster. You must strip the roof.

The Chemical Peel

A chemical peel is exactly what it sounds like. We apply an acid to the skin to intentionally dissolve the bonds holding the dead keratinocytes together. We burn off the top layer in a controlled, predictable manner. When the dead cells are stripped away, two things happen immediately: 1. The skin underneath is smooth, fresh, and highly reflective. The glow returns instantly. 2. The basal stem cells at the bottom of the epidermis panic. They sense that the barrier has been breached, so they go into overdrive, rapidly producing new, healthy cells to rebuild the wall.

You are forcing the skin to act young again.

Peels are categorized by depth: - **Superficial Peels (AHAs like Glycolic or Lactic acid, BHAs like Salicylic acid):** These only dissolve the very top layer of dead cells. They are “lunchtime peels.” Zero downtime, instant glow, excellent for mild dullness and acne. - **Medium Peels (TCA, Jessner’s):** These penetrate through the entire epidermis and reach the very top of the papillary dermis. They cause visible peeling for several days. They treat moderate sun damage, fine lines, and superficial pigmentation. - **Deep Peels (Phenol):** These burn all the way down into the reticular dermis. They require sedation, cardiac monitoring, and weeks of hideous downtime. They essentially melt the face off and force it to rebuild from scratch. The results are miraculous, but the risks (scarring, hypopigmentation, cardiac toxicity) are massive. Leave deep peels to the highly specialised experts.

For 95% of aesthetic practice, a well-formulated superficial or medium peel, combined with medical skincare, is all you need.

The Miracle of Retinoids

You cannot peel a patient every day. To maintain the accelerated turnover rate at home, you must use retinoids (Vitamin A derivatives like retinol, retinaldehyde, or prescription tretinoin).

Retinoids are the only topical ingredient universally proven by decades of robust clinical data to reverse skin ageing. They do not just exfoliate; they communicate with the cells' DNA.

They force the basal cells to divide faster. They compact the stratum corneum so it reflects light perfectly. They even dip down into the dermis to stimulate fibroblasts to make collagen and inhibit the MMP enzymes that destroy it.

If a patient asks you what one cream they should buy, the answer is always a retinoid (and sunscreen). Everything else is just marketing.

Lazy Skin Syndrome

This brings us to a critical concept that ties the entire epidermis and dermis together. A condition I see in my clinic every single day.

I call it Lazy Skin Syndrome.

A patient walks in. They are usually in their late thirties or forties. They have the financial means to care for themselves, and they spend a fortune doing so. Their bathroom cabinet looks like a luxury department store. They use heavy, expensive, thick moisturizers. They use oils. They use balms. They layer them on every night until their face shines like a glazed doughnut.

And their skin looks terrible.

It is dull. It is congested. It is prone to random breakouts. It lacks bounce. They complain that their skin is “so dry” that it literally drinks up the heavy creams, yet it never seems to improve.

They are killing their skin with kindness. They have induced Lazy Skin Syndrome.

Here is the biology of what they have done:

The skin is a self-regulating organ. Its job is to maintain hydration by producing its own natural moisturizing factors and by turning over its cells to maintain a healthy barrier.

When you constantly slather the skin in heavy, occlusive moisturizers, you send a signal to the basal layer of the epidermis. The signal says: *“Don’t worry, guys. We have plenty of moisture up here. You don’t need to work.”*

The basal cells slow down their division. The natural turnover rate grinds to a halt. The skin stops exfoliating itself. The dead keratinocytes pile up, creating a thick, dull, impenetrable layer of trash on the surface. Because this layer of dead cells is so thick, it cannot hold water properly. So the skin *feels* dry. The patient panics, assumes they need *more* moisture, and applies an even heavier cream. The cycle worsens. The skin becomes addicted to the external moisturizer while completely shutting down its own internal mechanisms.

The fibroblasts in the dermis below go to sleep because they aren't receiving any signals to repair or rebuild.

The skin has become lazy.

The Brutal Cure

To fix Lazy Skin Syndrome, you have to be cruel to be kind. You have to send the skin to boot camp.

1. **Confiscate the Creams:** You tell the patient to throw away every heavy moisturizer, oil, and balm they own. This terrifies them. 2. **Strip the Roof:** You use chemical peels in the clinic and AHAs/BHAs at home to aggressively dissolve the thick layer of dead cells that has built up.

2. **Whip the Cells:** You introduce a strong retinoid to force the basal cells to start dividing rapidly again. 4. **Hydrate from Within:** You replace their heavy creams with a lightweight, non-occlusive hyaluronic acid serum, and perhaps a course of injectable skin boosters to hydrate the dermis internally without suffocating the epidermis.

For the first two weeks, the patient will hate you. Their skin will freak out. It will feel tight, dry, and it will flake. They will beg you to let them use their expensive La Mer cream.

You must hold the line. You tell them their skin is going through withdrawal.

By week four, the magic happens. The dead layer is gone. The basal cells have woken up. The natural moisturizing factors are being produced again. The skin is turning over at a healthy 28-day rate. The dullness vanishes. The congestion clears. The glow returns.

They don't need the heavy creams any more, because their skin is finally doing its own job. You haven't just treated their skin; you have rehabilitated it.

This is the power of understanding the cells. When you know how the Architect, the Bricklayer, and the Painter work, you stop treating symptoms and start treating the system.



CHAPTER 9

Where the Needle Actually Lives — The Subcutaneous Layer

Welcome to the playground.

We have descended through the epidermis and the dermis, and we have arrived in the superficial subcutaneous layer. This is where injectors spend ninety percent of their careers. It is the padding. The cushion. The yellow stuff. It is also the layer that betrays you first when gravity takes hold.

This is the first layer that matters to your hands more than your eyes. The first layer where the cannula stops being a piece of metal and starts becoming a conversation with the tissue.

People like to say, “It’s mostly fat down there.” That is technically true. But technically true statements have ruined more injectors than bad technique. Because this layer is not just “fat.” It is a composite biological material — a living, breathing fabric made of soft lobules, fibrous septa, ligament branches, loose glide planes, vessels, and nerves.

Deep fat gives structure. Superficial fat gives shape. And shape is the language of beauty.

Why This Layer Deserves Your Respect

Superficial fat is the first thing we lose with age. Before bone resorbs, before deep fat shrinks, before ligaments sag — this layer starts to thin out.

When that happens: - Shadows appear where there used to be light. - The mid-cheek groove forms. - The tear trough looks darker even before it hollows. - The jawline softens into early jowls. - The face loses its bounce.

Restore this layer, and you restore expression before structure. This is the low-hanging fruit of injectable aesthetics. You don't need deep, heavy, structural injections to change someone's day — you just need to give the superficial layer back.

The Four Textures of the Superficial Layer

If you understand these four elements, you understand the entire superficial layer.

1. Fat Lobules — The Glide Zone Soft, deformable pillows. This is the “warm butter” your cannula glides through. The most forgiving tissue in the face.

2. Fibrous Septa — The Cobwebs This is the internal architecture. They give you tiny catches — little micro-grinds — just enough to remind you the face has structure.

3. Superficial Ligament Branches — The Flexible Fences These are the arborized tips of the retaining ligaments. Up here, they are gentle. You glide over them without effort. It is only when you go deeper that they become stiff vertical walls.

4. Loose Areolar Tissue — The Superhighway The suspiciously easy glide. Move your hand 2 mm, and the cannula travels 20. If it feels like you are cheating, you are in the right place.

And through all of this run the rubbery cables — arteries, veins, and sensory nerves — each with its own personality.

Material Science for Injectors — Metal Meets Tissue

Advancing a cannula through the superficial layer is the closest aesthetic medicine ever gets to physics class: - Soft fat deforms smooth, frictionless glide. - Septa resist lightly gentle catches. - Ligament branches deflect “No entry here. Try another route.” - Ligament trunks (deeper) block the “brick wall” sensation. - Fascia stops you dead wrong plane. - Vessels stretch slide away like elastic tubes. - Nerves stretch then fire time to pick a new direction.

These sensations form a force-displacement graph in your fingertips. You are not pushing a cannula. You are reading a language. The more fluent you become, the safer you become.

The Tactile Table — What You Are Actually Feeling

Structure	What It Feels Like	What It Means
Fat	Smooth, silky, low resistance	Perfect plane
Septa	Mild drag, cobweb tension	Still correct, small adjustments
Ligament branches	Soft deflection	Glide over; stay superficial
Ligament trunks	Brick wall stop	Too deep; withdraw and redirect
Fascia	Sharp, total resistance	Wrong plane
Loose areolar	“Too easy” glide	A glide plane — enjoy it
Vessel	Rubber-band behaviour	Keep moving, low pressure
Nerve	Zap, radiating	Stop, withdraw, change path

Correct Entry Technique

This is where most injectors go wrong. They think the cannula navigation starts after the entry point. Wrong. It starts with the needle. Injectors worry too much about depth. The truth is: it is actually very difficult to slip deep into the SMAS or deep fat by mistake.

Why? - The superficial fat layer is thick. - Deeper fascia blocks you. - Ligament trunks push you away. - Cannulas bend before they dive. - The path of least resistance is always superficial.

You almost always glide above the obstacles, not under them. This is why superficial work is beautifully forgiving — and why beginners should start here.

The Ligament Table — What Matters in This Layer

Ligament	Deep Origin	Superficial Behaviour	Injection Note
Orbital retaining ligament	Orbital rim	Strong tether at tear trough	Stay superficial or go just below
Zygomatic cutaneous ligament	Zygoma	Feels like a wall over cheek	Glide above it from lateral vectors
Masseteric cutaneous ligament	Masseter fascia	Divides cheek compartments	Angle up or down; do not punch through
Mandibular ligament	Mandible periosteum	Creates jawline stepoff	Superficial is safest
Temporal ligament	Temporal fascia	Limits lateral brow movement	Choose superficial or deep, not mid

Vessels and Nerves in the Superficial Layer

Arteries feel like firm, rubbery tubes. They have wall tension. They resist compression. When your cannula touches one, it slides away — like a garden hose under light pressure. This is normal. This is expected. This is why cannulas are safer than needles in this layer.

Veins are softer, more compressible. You may not feel them at all until you are on top of them.

Sensory nerves are the ones that matter most. When you stretch a sensory nerve, the patient feels it before you do. A sharp, radiating sensation — often described as electric — is your signal. Stop. Withdraw slightly. Redirect.

These are not obstacles to fear. They are landmarks to respect.

The Fat Compartments of the Superficial Layer

The superficial fat is not one continuous layer. It is divided into named compartments. - **Nasolabial fat** — sits medially, bounded by the nasolabial fold. Deflation here deepens the fold. Restoration softens it. - **Medial cheek fat** — the central cheek volume. The first compartment to show hollowing in mid-face aging. - **Middle cheek fat** — lateral to medial, sits over the zygoma. Gives the cheek its convexity. - **Lateral temporal-cheek fat** — the lateral cheek, connecting to the temporal region. Critical for the lateral cheek sweep. - **Orbital fat** — the periorbital fat that supports the lower eyelid. Deflation here

creates the tear trough. - **Jowl fat** — the inferior compartment. Descends with age. Creates the jowl.

Each compartment has its own vector of deflation. Each requires its own vector of restoration. Treating one without understanding the others creates imbalance.

What Happens When You Come From the Hairline Toward the Cheekbone

Injectors constantly ask: if I enter laterally and advance medially, what do I cross? Here is the sequence: 1. **Loose areolar tissue** (hairline to temporal region) — easy glide. 2.

Temporal ligament — mild deflection. 3. **Superficial fat of the lateral cheek** — smooth. 4. **Zygomatic cutaneous ligament** — you will feel it. 5. **Medial cheek fat** — back to smooth. 6.

Nasolabial fat — denser, smaller lobules. 7. **Nasolabial fold** — you have arrived.

Each transition has a texture. Each texture has a meaning. Each meaning tells you where you are.

My Practical Philosophy

I have injected in this layer thousands of times. Here is what I know:

The cannula is not a weapon. It is a probe. It tells you where you are. It tells you what is around you. It tells you when to stop.

The injector who listens to their cannula makes fewer mistakes than the injector who pushes through resistance. Resistance is information. Smooth glide is permission. A zap is a hard stop.

Learn the language of this layer, and the face will tell you exactly what it needs.

PART II

The Anatomy of the Ageing Face

CHAPTER 10

How to Read a Face — The Three Pillars of Youth

She was twenty-nine and worried about thirty.

A model, and a genuinely beautiful one, sitting in my chair pointing at her forehead. There were faint horizontal lines there when she raised her brows — grade one, barely present at rest, the sort of thing you would not notice unless you had been told to look for it. She had been told. The internet had told her, repeatedly, that thirty is the year women begin to go downhill, and she had arrived with the appropriate amount of dread.

She did not look twenty-nine. She looked about twenty-two, and not because anybody had helped her. Genetics had simply been generous.

But her skin was congested. Small firm bumps across the forehead — sebaceous hyperplasia, the sort you feel before you see. Oil sitting on the surface. A particular kind of shine that photographs badly and that she had never once mentioned, because it had never occurred to her that it was a thing anyone could mention.

So we had two problems in the room. The one she came about, which was almost imaginary. And the one she did not know she had, which was visible from the door.

I gave her a retinol. Free, deliberately, because free changes the transaction: a product she has paid for is a product she can quietly abandon, but a product I have handed her is one she has to face me about. I told her I would not see her again if she did not use it, and that I would know.

She came back three months later and her skin was extraordinary.

Clear, matte, light bouncing off it properly for the first time. I could not have guessed her age within a decade.

She looked in the mirror for a long moment before she said anything, the way they all do, and told me she finally looked like herself again.

Then she thanked me warmly for the Botox.

Any idiot can point at a wrinkle and stick a needle in it. That is not aesthetic medicine; that is playing whack-a-mole. If you want to actually change how a face is perceived, you have to stop looking at lines and start reading the architecture.

The biggest mistake a new injector makes is listening to the patient's words instead of reading their face. They come in pointing at a single line, a specific fold. They ask you to make it disappear. So you do.

You fill the line. You chase the wrinkle. You make that one tiny spot flawlessly smooth. And in doing so, you have fixed absolutely nothing. You have just made the violins louder while the rest of the orchestra is completely out of tune. The face is not a collection of lines to be erased. It is a story to be read. If you do not learn the language of that story, you will spend your entire career giving your patients exactly what they ask for. And never what they actually want.

The goal is not to make someone look “done.” It is to make them look rested, healthy, and well. It is to treat the whole canvas, not just the cracks in the paint. This is the entire philosophy of modern aesthetics. It rests on three pillars.

Every consultation you do, every treatment plan you build, and every chapter in this book is built on this foundation.

Pillar One: Skin Quality This is the canvas itself. Before you ever think about a line or a fold, you must assess the quality of the skin draped over the structure. Is it hydrated?

Is the texture smooth or rough? Are there open pores, congestion, or underlying redness? Is the pigmentation even or mottled? A patient with flawless, luminous skin and a few deep-set lines will almost always look younger and healthier than a patient with no lines but dull, dehydrated, or pigmented skin.

This is the domain of the epidermis and the superficial dermis. It is the foundation of everything. You cannot build a beautiful house on a rotten foundation.

When a patient complains of looking “tired,” they are often pointing to shadows and hollows, but the true culprit is the skin itself. Sensitised skin,

chronic inflammation, a compromised lipid barrier — these factors drain the light from a face. The skin stops reflecting light and starts absorbing it.

The fix here is not a syringe of cross-linked hyaluronic acid. It is the long game. Acids. Peels. Retinoids. Microneedling. Polynucleotides. It is about waking up the fibroblasts and resetting the keratinocyte turnover rate. If you ignore the canvas, your structural work will always look artificial.

A perfectly sculpted cheekbone under grey, mottled skin does not look youthful. It looks like taxidermy.

Pillar Two: Lines in Motion and at Rest This is where most patients and most injectors focus their attention. These are the dynamic lines of expression, caused by the pull of underlying muscles. And the static lines that have become etched into the dermis over time. This is the world of botulinum toxin and superficial fillers.

But these are merely tools, not the entire strategy. The goal is not to obliterate every line and freeze every muscle. A face with no lines is not a youthful face. It is a mask.

The art is in softening, not erasing. It is in respecting the natural movement of expression while gently uncoupling it from the negative vectors of ageing. Look at the lines, but read the muscles underneath them. Are the depressor muscles overpowering the elevators? Is the frontalis working overtime to lift a heavy brow?

Is the mentalis dimpling the chin because the mandible has lost projection? Lines are the symptoms of mechanical stress. If you simply paralyze the muscle or fill the line without understanding the mechanical forces at play, you will create a static, expressionless face. The goal is modulation.

Turn down the volume on the depressors so the elevators can sing.

Pillar Three: Volume Loss and Structural Collapse This is the silent, structural ageing that most patients cannot articulate. They do not come in asking for more fat in their deep medial cheek pad. They come in saying they look tired, sad, or hollow. They are pointing to the shadows, not the lines.

This is the slow, inevitable deflation of our facial fat compartments and the resorption of the bone beneath them. It is the architectural collapse that causes everything on the surface to sag, fold, and fall. Restoring this lost volume is the most powerful tool we have for true rejuvenation. When the deep temporal fat pad deflates, the eyebrow drops. When the malar fat pad descends, the nasolabial fold deepens. When the deep pyriform space loses bone, the upper

lip elongates. These are structural failures. They cannot be fixed with a skin cream, and they cannot be fixed with a needle in the dermis. This is where you become the architect. You must read the shadows to understand which deep structures have failed.

You place the cannula in the subcutaneous layer, or the needle on the periosteum, and you rebuild the scaffold. You are not filling the shadow. You are replacing the structure that used to prevent the shadow from forming.

The Clinical Example: The Tired Executive Let's put the three pillars together. A 45-year-old female executive sits in your chair. She tells you she looks exhausted, even when she has slept for eight hours. She points directly at her nasolabial folds and says, "I hate these lines. Fill them." If you are a whack-a-mole injector, you draw up a syringe of medium-G-prime filler, inject it directly into the nasolabial folds, and send her on her way.

She looks in the mirror. The folds are gone. But she still looks tired. Worse, her lower face now looks heavy, almost simian. You gave her what she asked for, and you failed her completely. If you know how to read a face, you do not look at the folds.

You look at the architecture. 1. Volume Loss (Pillar Three): You notice the shadow under her cheekbones. Her malar fat pad has descended because the zygomatic bone beneath it has resorbed and the deep medial cheek fat has deflated. The nasolabial fold is not a line; it is a cliff created by the falling cheek.

2. Lines in Motion (Pillar Two): You ask her to frown. Her corrugator muscles are hypertrophic. Even at rest, she has a faint "11" between her brows. The depressor muscles are pulling the medial brow down, casting a shadow over her eyes. 3.

Skin Quality (Pillar One): Her skin is dull. The pores on her nose and medial cheeks are enlarged. There is a faint, mottled pigmentation from years of sun exposure and stress. The skin is absorbing light, not reflecting it. The treatment plan writes itself.

First, you rebuild the structure. A high-G-prime filler placed deep on the zygoma and in the deep medial cheek fat. This lifts the descended tissue, supporting the lower eyelid and softening the nasolabial fold indirectly. You haven't touched the fold, but it is already 50% better.

Second, you modulate the muscle. A precise dose of botulinum toxin to the glabella complex. You turn down the volume on the depressors. Over the next two weeks, the medial brow will lift slightly, opening the eyes and erasing the

scowl.

Third, you address the canvas. You prescribe a medical-grade skincare protocol: a gentle AHA/BHA cleanser, a potent antioxidant serum, a prescription retinoid at night, and a strict SPF regimen. When she returns for her review four weeks later, she does not mention her nasolabial folds.

She says, “Everyone at the office keeps asking if I went on holiday.” You didn’t erase her lines. You rebalanced her architecture. You lifted the shadows. You polished the canvas.

You gave her what she actually wanted. That is how you read a face.



CHAPTER 11

Five Ways a Face Falls — A Framework for Ageing

Ageing is not a single event. It is a slow, coordinated collapse. The skeleton shrinks, the fat deflates, the ligaments tighten, and the skin stretches. It happens in three dimensions, and if you try to fix a 3D collapse with a 1D solution — like just pulling the skin tight — you create a monster.

The cascade of ageing. Failure at each layer drops the one above it, which is why restoring the deep foundation does more than chasing the surface line.

A good injector looks at a face and sees a story. The nasolabial fold did not simply appear one morning. The jowl did not decide to exist overnight. The hollow under the eye was not there at twenty-five and then suddenly catastrophic at forty. Ageing is a sequence.

It has a logic. It has a direction. And if you understand the sequence, you can read a face the way a detective reads a crime scene — not just cataloguing what you see, but understanding why it happened, when it happened, and which layer failed first.

That is the difference between treating symptoms and treating causes.

The Five Vectors of Facial Ageing The face ages through five distinct mechanisms, operating simultaneously but at different rates in different people. Vector 1: Bone. The skeleton is not static. It remodels throughout life, and after the midtwenties it begins to resorb. The orbital rim widens and recedes, creating the hollow under the eye.

The pyriform aperture — the bony frame around the nose — expands, causing the nose to appear wider and the nasolabial area to lose support. The mandible loses height and projection, particularly at the angle and the chin. In

women, this accelerates dramatically after the menopause due to oestrogen-driven bone loss.

The jawline that looked sharp at thirty-five is structurally compromised by forty-five — not because of fat or skin, but because the bone underneath has retreated. Vector 2: Deep Fat.

The deep fat compartments — the deep medial cheek fat, the suborbicularis oculi fat (SOOF), the deep temporal fat — deflate with age. These compartments sit directly on the periosteum and provide the volumetric foundation for the face. When they shrink, the overlying structures lose their support. The cheek descends. The midface flattens.

The tear trough deepens. The nasolabial fold appears — not because fat has fallen, but because the scaffold underneath has collapsed. Vector 3: SMAS. The superficial musculoaponeurotic system is a fibromuscular layer that connects the facial muscles to the overlying skin. It is held in place by the retaining ligaments.

Over time, those ligaments stretch. The SMAS descends. The structures attached to it descend with it. This is the primary mechanism of jowl formation and lower face sagging. It is also the primary target of surgical facelifts and, to a lesser extent, thread lifts and HIFU.

Vector 4: Superficial Fat. The superficial fat compartments — the nasolabial fat, the jowl fat, the malar fat pad — do not simply deflate. They also descend. As the ligaments loosen and the SMAS drops, the superficial fat slides inferiorly and medially. The malar fat pad that once sat high on the cheek migrates towards the nasolabial fold.

The jowl fat accumulates along the mandible. This is why treating the nasolabial fold directly is often the wrong approach — you are treating the consequence, not the cause. Vector 5: Skin.

The epidermis thins. Keratinocyte turnover slows. The dermis loses collagen and elastin. The dermo-epidermal junction flattens. Photoaging adds pigmentation, telangiectasia, and surface irregularity. The skin that was once taut and luminous becomes thinner, drier, and less able to conceal the structural changes happening beneath it.

Understanding which vector is dominant in your patient determines your treatment strategy entirely.

Face Shape and the Ageing Trajectory Not all faces age the same way. Face shape is one of the most reliable predictors of how a patient will age — and

when. The slim, oval, or petite face tends to age through deflation. It looks older earlier.

At twenty-five, the slim face may already appear slightly gaunt. The temples are slightly hollow. The cheeks are not full. The under-eye area shows early. But here is the counterintuitive truth: the slim face, once it has lost its youthful volume, tends to be structurally stable for a long time.

There is less weight to descend. The ligaments are under less load. Sagging is slower. The slim-faced patient at fifty often looks better than expected — not because they have not aged, but because they aged through deflation rather than descent, and deflation is far more treatable.

The round, fuller face tells the opposite story. It looks younger for longer. At twenty-five, the full face is often genuinely beautiful — high cheeks, full lips, soft jawline. At thirty-five, it still looks youthful.

But the weight of the soft tissue is loading the retaining ligaments every day. The SMAS is under constant gravitational stress. When the descent begins, it is rapid. The jowl appears suddenly. The jawline blurs. The nasolabial fold deepens quickly. The round-faced patient at forty-five can look a decade older than the slim-faced patient of the same age — not because of skin quality, but because of structural descent.

The clinical implication is important. The slim-faced patient needs volume. The roundfaced patient needs lifting and tightening. Giving volume to a round face that is already descending makes it worse. Giving HIFU or threads to a slim face that is simply deflated does nothing useful.

Read the face before you plan the treatment.

The Decade-by-Decade Ageing Sequence Ageing does not announce itself. It accumulates quietly, one layer at a time, until the patient looks in the mirror one morning and wonders when it happened. Here is when it happened.

Late teens. The first sign is not visible to most people. But it is there. The deep fat compartments begin their very earliest deflation. The face is still full, still radiant. But the foundation is already beginning to change. Volume that existed at sixteen is slightly less present at nineteen.

Early twenties. The skin cycle begins to slow. At sixteen, keratinocyte turnover is approximately fourteen days. By twenty-two, it is closer to twenty-eight. The skin starts to look slightly less luminous. Foundation sits differently.

Makeup does not blend as effortlessly. The patient starts using more product to achieve the same result. This is the first clinical signal — not a

wrinkle, not a fold, but a change in skin quality. Mid-twenties.

Two things happen simultaneously. Static lines begin to appear — the first faint horizontal forehead lines, the early crow's feet visible at rest. And the under-eye hollow makes its first appearance. The tear trough is not yet a clinical concern, but the patient notices it in photographs.

The upper eyelid may show the first millimetre of heaviness. The eyebrow sits fractionally lower than it did at twenty. Late twenties to early thirties. The midface begins to flatten.

The cheeks lose their convexity. The corner of the lips starts to turn very slightly downward — not dramatically, but enough to change the resting expression from neutral to subtly unhappy. The undereye hollow is now visible in person, not just in photographs. The upper eyelid has descended by two to three millimetres.

The eyebrow has followed. Mid-thirties. The upper face is now clearly showing change. The brow has descended. The upper eyelid is heavier. The lateral hooding is beginning. The midface has lost its youthful projection. In women, the first signs of jawline softening appear — not yet a jowl, but the clean mandibular line is less defined. Oestrogen levels are beginning their gradual decline, and bone resorption at the mandible is accelerating. Late thirties to early forties. The lower face changes rapidly in this decade. The jawline collapses.

The jowl appears. The marionette lines deepen. The chin loses projection. In women, the perimenopause accelerates bone resorption, collagen loss, and fat redistribution simultaneously. A patient who looked thirty-five at thirty-eight can look fortyfive at forty-two if the hormonal transition is abrupt. Forties and beyond.

All five vectors are now operating in concert. The bone has receded. The deep fat has deflated. The SMAS has descended. The superficial fat has migrated. The skin has thinned.

The face is no longer a single problem — it is a system that has failed at multiple levels simultaneously. The early signs were in the upper face. The mid-face followed.

The lower face came last. That is the sequence. Upper to mid to lower. And that is also the order in which you should treat.

Reading the Face — Which Layer Has Failed When you look at a patient, you are not looking at a face. You are looking at five layers, and you need to

identify which one is the primary driver of what you see. The hollow under the eye that appears in the mid-twenties is almost always a deep fat story — the SOOF and deep medial cheek fat deflating.

Treat it at the deep level. The nasolabial fold that appears in the early thirties is almost always a midface volume story — the malar fat pad descending as the deep scaffold collapses. Treat the cheek, not the fold. The jowl that appears in the late thirties is almost always a SMAS and ligament story — the structural descent of the lower face. Treat it with lifting, not volume.

The skin that looks dull and tired at any age is almost always an epidermal story — slow turnover, poor barrier function, photoaging. Treat it with the skin first.

The HSI Mini Facial Scale Reading a face subjectively is a skill. Communicating what you see to a patient is an art. But documenting it objectively, tracking change over time, and presenting a logical treatment plan — that requires a system. The HSI Mini Facial Scale is that system. It scores the face across three domains.

Each parameter is rated 0 (none) to 3 (severe), except static lines which go to 4. The total gives you an Aesthetic Number — a quick, objective summary of where the patient stands and a visual guide to treatment priorities.

HSI Mini Facial Scale Antiageing Before (Client has had previous upper face Botox) Skin Surface (0,1,2,3) Pores Size & Congestion Colour Changes Hydration Roughness Sensitivity Static Lines (0,1,2,3,4) Frown Forehead Crows Feet DAO (Sad Expression Lines) Bunny Lines Oral Commissure Mentalis Crease Volume & Structure (0,1,2,3) Temples Cheeks Nasolabial Chin Jawline Marionette After Pores Size & Congestion Colour Changes Hydration Roughness Sensitivity Frown Forehead Crows Feet DAO (Sad Expression Lines) Bunny Lines Oral Commissure Mentalis Crease Temples Cheeks Nasolabial Chin Jawline Marionette Lips Tear Trough Skin Laxity (0,1,2,3) Jawline Laxity Eyebrow Hooding Total Lips Tear Trough Jawline Laxity Eyebrow Hooding Total Once each category is scored, the total gives an “Aesthetic Number” — a quick, objective summary of where your patient stands and a helpful visual guide to explain treatment priorities. A patient with strong dermal quality but mild volume loss might focus on fillers later, while one with poor skin texture benefits first from collagen stimulators or topical repair.

This approach turns your consultation into a science-backed discussion rather than a sales pitch. Patients appreciate seeing logic in your plan, not persuasion in your tone. How to Use It During the consultation, score the patient across all domains. A 35-year-old might score a total of 8 — mostly static lines and mild volume loss.

The treatment plan writes itself: botulinum toxin and a skin protocol. A 55-year-old might score 22 — volume, laxity, and surface all in play. Now you have a real conversation.

You explain that their primary issues are volume and descent. You cannot fix a Volume 3 and Descent 2 problem with botulinum toxin alone. You need structural filler, biostimulators, and potentially lifting devices. The scale takes the emotion out of the consultation. It removes the mirror from the patient's hand and replaces it with a clinical assessment.

It stops them pointing at a single line and forces them to see the whole picture.



CHAPTER 12

The Retaining Ligaments — Architecture, Not Obstacles

Most injectors treat retaining ligaments like an annoyance. Something that gets in the way of the cannula. Something that needs to be pushed through, subcised, or ignored entirely. That is a fundamental misunderstanding of facial architecture. Retaining ligaments are not obstacles. They are the scaffolding. They are the suspension cables holding the entire soft tissue envelope to the skeleton. If you understand where they *The major retaining ligaments and where they anchor. These are the fixed points the face ages around.*

are, you understand exactly where the face is going to fail. And if you understand how to use them, you understand how to lift a face rather than simply inflating it.

Lift Is Not Inflation Let's start here, because this is the mistake that defines the difference between an injector and a clinician. One of the greatest misconceptions in aesthetic medicine is that rejuvenation comes from adding more filler. It doesn't.

Lift comes from restoring support. A small amount of filler placed strategically adjacent to a retaining ligament can alter the tension and contour of the overlying soft tissues far more effectively than simply filling a visible groove. The goal is not to inflate the face.

The goal is to rebalance the forces acting upon it. You have seen the patients who have been over-filled. The heavy cheeks. The pillow face. The nasolabial folds that are somehow deeper despite the fact that someone has been filling them for three years. That is what happens when you treat symptoms instead of architecture.

You add volume to a face that does not need volume — it needs support. And the more you inflate without restoring support, the worse the descent becomes, because now you have added weight to a structure that is already failing under gravity. The ligaments are the key.

They are the reason a face falls in the pattern it does. They are the reason a jowl forms where it forms, and a tear trough appears where it appears, and a nasolabial fold deepens in the specific way it deepens. The face does not age randomly.

It ages along the lines of its own architecture. Learn the architecture, and the ageing pattern becomes entirely predictable.

The Harley Street Principle Retaining ligaments are not obstacles to navigate around — they are the architectural blueprint of the face. They determine where shadows form, where tissues descend, where contours begin and end, and where rejuvenation should be directed. Great injectors do not fight the ligaments. They use them.

This is what I call the Harley Street Principle: understand anatomy like a scientist, inject like an architect, finish like an artist. The scientist understands the ligament — where it originates, where it inserts, what it holds, and what happens when it stretches. The architect uses that knowledge to place product not where the problem is visible, but where the structural correction will have the greatest mechanical effect.

The artist steps back, looks at the whole face, and makes the final adjustments that turn a clinical result into something genuinely beautiful. Most injectors skip the first two steps and go straight to the third. They see a shadow and they fill it. They see a fold and they fill it. They see a hollow and they fill it. And the result looks filled, not lifted. Because they are working on the surface of a problem that lives in the structure. The ligaments are the structure.

True Ligaments vs. False Ligaments Not all ligaments are created equal. True retaining ligaments are osteocutaneous. They run from the bone, through the deep fat, through the SMAS, through the superficial fat, and insert directly into the dermis. They are the main load-bearing pillars.

The orbital retaining ligament, the zygomatic cutaneous ligament, and the mandibular retaining ligament are true ligaments. False retaining ligaments do not originate on bone. They run from fascia to dermis.

The masseteric cutaneous ligament and the platysma auricular ligament are false ligaments. They are tethering points, but they are not the primary

structural anchors. The distinction matters clinically.

True ligaments are the ones you can use to create lift — by placing filler at their origin on the bone, you push the entire ligament upward and outward, re-tensioning the soft tissue above. False ligaments are the ones that create visible boundaries and grooves when they fail — and the ones that trap descending fat pads, creating the jowl and the marionette line.

The Ligament-by-Ligament Breakdown If you want to understand how a face ages, you have to understand what happens when these specific ligaments stretch. Ligaments do not snap. They stretch. Imagine a hammock tied between two trees. When the hammock is new, it is tight. As years go by, the ropes stretch and the hammock sags.

But in the face, it is worse — the trees themselves (the bone) are moving closer together as they resorb. The hammock gets longer and the trees get shorter. The sag is inevitable.

The question is only how fast. **The Orbital Retaining Ligament (ORL)** This ligament sweeps around the inferior orbital rim. When we are young, it holds the cheek fat tightly against the lower eyelid. As we age, the bone beneath it resorbs and the ligament itself stretches.

The fat pad drops. Suddenly, the ligament becomes visible on the surface — not as a structure, but as a boundary. The tissue above it is empty (the tear trough). The tissue below it is full (the descending cheek fat). **Clinical Relevance:** When you inject the tear trough, you are often injecting directly superior to the ORL.

If you inject inferior to it, you are simply making the cheek heavier and the trough look deeper. The ORL is the reason the tear trough exists. It is also the reason the tear trough is so technically demanding — you are working in a millimetre-wide corridor between the ligament and the orbital rim, with a vascular network directly beneath you. Respect the ORL. It will tell you exactly where to put your cannula. **The Zygomatic Cutaneous Ligament (ZCL)** This is the strongest ligament in the midface. It originates on the zygomatic arch and holds the malar fat pad high on the cheekbone.

When the ZCL stretches, the entire midface descends. The malar fat pad slides inferiorly and medially, crashing into the nasolabial fold. **Clinical Relevance:** This is the key to midface lifting. By placing high-G-prime filler deep on the bone, directly under the origin of the ZCL, you push the ligament upward and outward.

You re-tension it. You are not just adding volume — you are tightening the suspension cable. This is why 0.5 ml of filler placed correctly on the zygoma can lift the entire midface, while 2.0 ml placed superficially in the nasolabial fold simply makes the patient look like a chipmunk.

Architecture over volume. Always. The Masseteric Cutaneous Ligament A false ligament, but a crucial one. It runs along the anterior border of the masseter muscle. As the midface fat descends, it hits this ligament and stops. This is what creates the lateral boundary of the jowl.

Clinical Relevance: If you try to correct a jowl by injecting directly into the masseteric ligament, you will simply widen the lower face. The correction must happen superiorly (lifting) or posteriorly (re-establishing the jawline angle). The masseteric ligament is not your target — it is your landmark.

The Mandibular Retaining Ligament A true ligament originating on the mandible, just lateral to the chin. This is the medial boundary of the jowl. As the tissue descends from above, it pools between the masseteric ligament laterally and the mandibular ligament medially. Clinical Relevance: The pre-jowl sulcus is the empty space medial to the mandibular ligament.

Filling this space camouflages the jowl by smoothing the transition from chin to jawline. But again — filling the sulcus alone does not address the descent. It addresses the appearance of the descent. The real correction is structural: restore the mandibular angle posteriorly, support the jawline with a cannula, and let the pre-jowl fill be the finishing touch, not the entire treatment.

The Masseteric Ligament in Action — A Clinical Case The masseteric ligament anchors the skin and SMAS to the deep fascia over the anterior border of the masseter. As a patient loses volume in the outer temporal fat pad and the midcheek, the tissues descend. The masseteric ligament, however, remains firmly fixed. This creates a vertical tethering — a subtle indentation or groove just anterior to the masseter muscle. Inferiorly, this tethering blends into the pre-jowl sulcus, making the jowl appear more prominent. When you see this presentation, the instinct of a novice is to fill the groove directly. If you do this, you will overfill the lower face and create a heavy, unnatural look.

The groove is not the problem; the groove is the anchor. The problem is the descent of the tissue above it. My treatment plan for this presentation is twofold. First, use a large blunt cannula (22G) to gently subcise and dissect the masseteric ligament — you will feel a satisfying release as the cannula passes

through the tether.

Second, place a very light wash of filler in the immediate region to prevent re-adhesion, but place the majority of the structural volume posteriorly and superiorly, up into the outer temporal fat pad and the lateral cheek. By restoring support above and behind the ligament and releasing the tether below, you lift the tissue without adding bulk to the lower face.

Treat the cause, not the symptom.

The Ligament Map as a Treatment Blueprint Here is how to use this knowledge in practice. Before you plan any treatment, map the ligaments mentally. Ask yourself: Which ligaments have stretched? The answer tells you where the descent is coming from. Which fat pads have descended past which ligaments? The answer tells you what is creating the visible folds and shadows.

Where are the ligament origins on the bone? The answer tells you where to place structural filler for maximum mechanical effect.

The major retaining ligaments, classified and mapped:

Ligament	Type	Origin	Consequence of failure	Correction strategy
Orbital retaining ligament (ORL)	Truete	Orbital rim	Tear-trough hollow under the eye	Cannula superior to the ORL; periosteal bolus at the rim for deep cases
Zygomatic cutaneous ligament	Truete	Zygoma	Deep nasolabial fold as midface descends past it	Midface filler at origin on the zygoma
Masseteric cutaneous ligament	Fascial	Masseteric fascia	Lateral jowl (descending fat halts at this boundary)	Posterior/superior volume; subcision; never fill the jowl directly
Mandibular retaining ligament	Truete	Mandible (lateral chin)	Pre-jowl sulcus; step-off at the jawline	Pre-jowl fill as camouflage; structural jawline support

This is the map. The face ages along these lines. Your treatment plan should follow the same map in reverse — restoring support at the origins, releasing the tethers that have become pathological, and filling the residual shadows last. Understand anatomy like a scientist. Inject like an architect. Finish like an artist. In that order. Always in that order.



CHAPTER 13

Twenty Minutes to Get It Right — A Consultation, Start to Finish

Let's put the theory into practice. The consultation is where you win or lose the patient. It is not a sales pitch. It is a negotiation between what the patient thinks they need and what the architecture actually demands. If you simply agree with whatever they point at, you are not a clinician; you are a waiter taking an order.

*This chapter covers the structure of the consultation: what you look at, in what order, and how you arrive at a plan. It has a companion. Chapter 36 covers the other half — the three questions I ask every patient, what their answers predict, and the cases where the correct plan is no treatment at all.

Read them together; neither works properly alone.* Sarah, a 42-year-old professional, sat in my chair with a common lament: she felt she looked perpetually tired. She handed me the mirror and pointed directly at her nasolabial folds and the subtle, yet noticeable, onset of jowling.

Crucially, Sarah was a virgin to aesthetic treatments. Her apprehension about appearing “overdone” was palpable — a sentiment I encounter daily. She had seen the pillow faces walking around Mayfair. She didn't want to look like them. She wanted to look like herself, just less exhausted.

During the initial assessment, I took the mirror out of her hand. I didn't look at her folds. I looked at her architecture. Her skin quality, while generally good, showed early signs of laxity and a compromised lipid barrier. The pattern of volume loss was textbook for her age: noticeable hollowing in the mid-face and a slight descent of the malar fat pads.

Her masseter muscles were of average size, not contributing significantly to facial widening, but the early jowling indicated a need for structural support

along the jawline. The key structural deficits were mid-face volume depletion and nascent laxity in the lower face. The nasolabial folds were not the disease; they were the symptom.

Communicating these findings to Sarah required a careful translation from clinical jargon to understandable terms. I didn't talk to her about the zygomatic cutaneous ligament or the deep medial cheek fat. I explained that her "tired" look stemmed from a natural loss of support in her cheeks.

The scaffolding had shrunk, which in turn exacerbated the lines around her mouth and contributed to the slight heaviness along her jawline. I assured her that the goal was not to inflate her face or erase every line. The goal was to restore the support that gravity had stolen. The proposed treatment plan was meticulously staged over three sessions. This strategy is designed to build results gradually and address the fear of an unnatural outcome. You never do everything on day one, especially with a virgin patient.

You build the foundation, let it settle, and then refine. • Session One: The mid-face (cheeks). This foundational step provides lift and support, indirectly improving the nasolabial folds. • Session Two: The residual nasolabial folds and the pre-jowl sulcus. • Session Three: Skin quality and surface refinement.

This phased approach allowed Sarah to acclimatise to the changes and build trust in the process. Session One: The Foundation Two weeks later, Sarah returned. The focus remained on establishing a strong mid- face foundation. I administered 1.0 ml of high-G-prime filler per side to the zygomatic arch, placed supraperiosteally to provide deep structural support.

This was the architect at work — placing the load-bearing pillars. This was complemented by 0.5 ml of a softer filler per side to the mid-cheek fat pad, addressing the more superficial volume loss and creating a smoother contour. The immediate result was subtle but effective: a noticeable lift in her mid-face, softening the shadows that had contributed to her tired appearance.

Sarah looked in the mirror and smiled. She remarked that she looked "fresher" without looking "different." That is the exact reaction you want. Session Two: The Refinement Three months following her initial treatment, Sarah presented for her second session. The cheeks had settled beautifully.

A significant, and fully anticipated, secondary benefit of the mid-face treatment was evident: her nasolabial folds had softened by approximately forty percent from the cheek augmentation alone. This demonstrated the interconnectedness of facial aesthetics perfectly. The lift had reduced the

inflation requirement. For the residual crease in her nasolabial folds, I used 0.3 ml of a soft, superficial filler.

Just a wash. Not a deep structural fill. The pre-jowl sulcus, a key area contributing to the early jowling, was then addressed with 0.5 ml of medium-G-prime filler per side. Again, we were not filling the jowl; we were camouflaging the descent by providing subtle definition and support to the jawline anterior to the masseteric ligament.

Session Three: The Surface Six months later, Sarah returned for the final phase. The structure was sound. The shadows were gone. Now it was time for the artist to polish the canvas. We initiated a medical-grade skincare protocol and a course of polynucleotides to address the early laxity and restore the dermal thickness.

We were no longer fighting gravity; we were fighting time. The Takeaway Sarah didn't need her folds filled. She needed her architecture rebuilt. By respecting the layers, addressing the vectors of ageing, and staging the treatment, we delivered exactly what she wanted without ever giving her exactly what she asked for.

That is the job. You are not there to take orders. You are there to diagnose the structural failure and prescribe the architectural solution. The mirror lies to the patient.

It is your job to tell them the truth.

Men Arrive With a Target. Women Arrive With a Feeling.

The Sarah case study highlights one of the most consistent patterns in nearly twenty years of clinical practice: men and women do not consult in the same way. They do not communicate their concerns in the same language. They do not process the consultation in the same emotional register.

And if you treat them identically — if you apply the same consultation framework to both — you will consistently underserve one of them. Understanding this distinction is not about stereotyping. It is about pattern recognition.

It is about developing the clinical intelligence to meet each patient where they actually are, rather than where your consultation template assumes them to be. The Male Patient: A Target, Not a Feeling A man who books an aesthetic consultation has usually already made a decision.

He has identified a specific concern, researched it online, cross-referenced it against before-and-after photographs, and arrived at your clinic with a named

target. “My jawline.” “My undereyes.” “I want to look less tired.” He is not asking for your opinion on his face as a whole. He is asking you to confirm that you can address the specific thing he has already decided needs addressing. This is not a criticism. It is a cognitive style.

Men tend to be outcome-focused and transactional in clinical settings. The consultation is shorter not because they care less, but because the brief is clearer. They have done the work.

They want to know if you can deliver. The practical implication of this is that the male consultation runs efficiently when you validate the target first. Acknowledge what he has identified.

Confirm that you understand the concern. Only then — once trust is established — can you introduce the broader clinical picture.

Because here is the risk with the male patient: he can be too specific. A man who presents wanting “more jaw definition” may have identified the symptom correctly but the cause incorrectly. His real issue is often mid-face volume loss — the descent of the malar fat pad that has allowed the lower face to appear heavier by comparison.

Placing product directly along the mandible without addressing the mid-face first will give him a result that looks augmented rather than restored. He will notice something is off even if he cannot articulate why. Correcting this requires careful framing.

You are not dismissing his target. You are expanding his understanding of how to achieve it. The language matters: “To get the jaw definition you’re looking for, the most effective approach is actually to restore some of the volume here first — it creates the contrast that makes the jaw read more defined.” You have validated his goal and redirected the clinical pathway without making him feel wrong.

Men are more resistant to upselling than women, and they should be respected for it. Do not introduce additional treatments in the first consultation unless they are directly relevant to the stated concern. What you will find, if you earn the trust, is that male patients become extraordinarily loyal.

They return on schedule, they refer their colleagues, and they do not require extensive reassurance between appointments. The Female Patient: A Feeling, Not a Target No woman has ever sat in your consultation chair and said: “I would like my nasolabial fold reduced by forty percent.” She has said: “I look

tired.” She has said: “I look angry, even when I’m not.” She has said: “I just don’t look like myself any more — I look in the mirror and I don’t recognise the person looking back.” These are not clinical targets.

They are emotional states. And the practitioner who responds to them with a clinical target — who hears “I look tired” and immediately reaches for the tear trough consent form — has fundamentally misread the consultation. Women rarely present with a named anatomical concern because they are not thinking in anatomical terms.

They are thinking in terms of how they feel about what they see. The line, the fold, the shadow — these are not the problem. The problem is the feeling those things produce. The loss of recognition.

The disconnect between how they feel on the inside and what they see reflected back at them. What she wants is not a corrected nasolabial fold. She wants to look fresher.

Younger. More like herself at thirty-two. This distinction has profound clinical implications. If you chase the symptom — if you fill the line because she pointed at the line — you may produce a technically adequate result that leaves her dissatisfied. Because the line was never the point.

The point was the feeling, and you have not addressed the cause of the feeling. The cause is almost always a combination of volume loss, structural descent, and skin quality change — a systemic shift in the face that manifests as a collection of symptoms, of which the line is only one. The consultation, for the female patient, is not a precursor to the treatment. The consultation is the treatment.

If she leaves your chair feeling genuinely heard — if she feels that you have understood not just what she pointed at but what she meant — she will return. If she leaves feeling processed, if she feels like a set of anatomical coordinates rather than a person, she will not return regardless of how technically excellent your injecting is.

The clinical result is almost secondary to the relational experience. This is not a soft skill. This is a clinical skill. The ability to reflect an emotion back as a clinical framework — to say “what you’re describing sounds like a loss of structural support in the mid-face, which is creating the tired appearance — let me show you what I mean” — is as technically demanding as threading a cannula through the correct plane.

It requires practice, observation, and a genuine interest in the person in front of you. The Unspoken Motivation There is one thing that both male and female patients share, and it is worth naming directly: neither will tell you their real motivation for being there.

The man who wants a “sharper jaw” may be going through a divorce, re-entering the dating world after fifteen years, or facing a younger competitor at work. He will not tell you this.

He will tell you about his jawline. The woman who says she “just doesn’t look like herself” may be processing grief, a significant birthday, or the quiet accumulation of years of feeling invisible. She will not tell you this either. She will tell you she looks tired.

This is not deception. It is the entirely human tendency to present the surface concern rather than the underlying one. Your role is not to excavate the emotional history — that is not what they came for, and it is not your place. Your role is to recognise that the surface concern is real and valid, that the underlying motivation is none of your business, and that the most respectful thing you can do is treat the clinical presentation with genuine skill and genuine care.

“The consultation is a diagnostic procedure. What the patient says is the symptom. What they mean is the diagnosis. Your job is to close the gap between the two.”



CHAPTER 14

The SMAS — The Engine Room

If you want to understand how a surgical facelift works, how thread lifts work, and how energy-based devices like HIFU actually do anything, you have to understand the SMAS.

The Superficial Musculoaponeurotic System (SMAS) is the most important anatomical concept in facial rejuvenation that patients have never heard of. It is not skin. It is not fat.

It is not bone. It is the engine room. What is the SMAS? The SMAS is a continuous, organised fibrous network that connects the facial muscles to the dermis. Think of it as a tightly woven net that wraps around the face. The facial muscles (like the zygomaticus or the risorius) are attached to the underside of this net.

The skin is attached to the top of the net via tiny fibrous septa. When a facial muscle contracts, it doesn't just pull on the bone. It pulls on the SMAS.

The SMAS then pulls on the skin, creating facial expression. Without the SMAS, you could not smile, frown, or show surprise. The muscles would contract beneath the skin, but the skin itself would not move. The Anatomy of the Net The SMAS is not the same thickness everywhere.

In the lower face and neck, it is thick and robust. In the neck, it actually merges with the platysma muscle. Over the parotid gland and the masseter muscle, it is dense and fibrous. As it moves superiorly and medially towards the centre of the face, it becomes thinner.

Over the cheekbones, it is a delicate, almost translucent layer. This continuous net divides the facial fat into two distinct layers. Everything above the SMAS is superficial fat. Everything below the SMAS is deep fat. How the SMAS Ages The SMAS is highly elastic when we are young. It snaps back

perfectly after every smile, every frown, every movement. But like all fibrous tissue, it loses its elasticity over time. It is subjected to thousands of muscle contractions every day, and it is under constant gravitational load from the superficial fat sitting on top of it.

Eventually, the net begins to stretch. When the SMAS stretches, everything attached to it descends. The superficial fat pads slide downward. The skin sags. This is the primary mechanism of lower face ageing. The jowl is not just fat — it is the SMAS failing to hold that fat in its youthful position.

The blurring of the jawline is the SMAS and platysma complex losing its tension. The Clinical Relevance: Why We Target the SMAS Because the SMAS is the layer that holds the soft tissue in place, it is the only layer that can truly lift the face when manipulated.

1. Surgical Facelifts. A modern facelift does not pull the skin. Pulling the skin results in the stretched, wind-blown look of the 1980s. A modern facelift dissects down to the SMAS, lifts the SMAS, anchors it in a higher position, and then simply re-drapes the skin over the new foundation. The tension is on the SMAS, not the skin. 2.

Thread Lifts. When you place a lifting thread (like a cogged PDO or PLLA thread), you are aiming for the immediate sub-dermal plane, engaging the fibrous septa that connect to the SMAS. The cogs catch the SMAS complex and physically pull it upward. If the thread is placed too superficially, it puckers the skin.

If it is placed too deeply, it misses the SMAS and anchors in fat, which cannot hold tension. 3. High-Intensity Focused Ultrasound (HIFU). Devices like Ultherapy do not target the skin surface. They deliver focused ultrasound energy deep into the tissue, specifically targeting the SMAS layer.

The thermal injury causes the collagen fibres in the SMAS to contract and denature, creating a tightening effect. It is the only non-surgical device that targets the exact same layer as a surgical facelift. The Injector's Relationship with the SMAS As an injector using needles and cannulas, you are rarely trying to inject into the SMAS.

Your relationship with the SMAS is navigational. You need to know where it is so you can decide whether to be above it or below it. When you want to rebuild structure (cheeks, pyriform, deep jawline), you must go below the SMAS, placing your product on the bone or in the deep fat compartments.

When you want to treat dynamic lines or refine the surface (nasolabial lines, superficial volume), you must go above the SMAS, into the superficial fat or the dermis. The SMAS is the boundary line.

Respect it, understand it, and know which side of it you are on at all times. The SMAS-Lifting Effect of Deep Fat Filler If the SMAS is a surgical plane, and we are strictly subcutaneous or deep-fat injectors, why do we care about it? Because the SMAS is the canvas upon which our deep injections act.

Think of the SMAS as a trampoline. The deep fat compartments sit underneath the trampoline. The superficial fat and skin sit on top. When we inject high-G-prime filler into the deep fat compartments — particularly the deep medial cheek fat or Ristow's space — we are inflating the space underneath the trampoline.

As the deep fat compartment expands, it pushes upward against the underside of the SMAS. The SMAS is a continuous, inelastic sheet. When you push up on it from below in the midface, it tightens the entire sheet. This tightening transmits outward and upward, lifting the superficial fat and skin that rest on top of it.

This is why a deep suprapariosteal injection of 0.5 ml in the midface can suddenly soften a nasolabial fold two inches away. You did not touch the fold.

You inflated the deep fat, which pushed up the SMAS, which pulled back the superficial fat, which softened the fold. This is the indirect lift. You cannot achieve an indirect lift if you inject superficial to the SMAS. If you inject on top of the trampoline, you add weight. The trampoline sags further.

The face looks heavier. This is why the SMAS matters to the injector even though we never cut it, never suture it, and rarely think about it by name. We use it as a mechanical lever to transmit the force of our deep injections to the surface of the skin.

CHAPTER 15

Bone — The Final Frontier of Aesthetics

For decades, aesthetic medicine was obsessed with wrinkles. If there was a line, we filled it. If there was a crease, we softened it. If there was loose skin, we tightened it. It wasn't wrong.

It was simply incomplete. Then came the revolution of fat compartments. Suddenly we realised the fold wasn't the problem. The fold was simply telling us that something underneath had disappeared. So we stopped chasing the wrinkle and started replacing volume. We became obsessed with superficial fat, then deep fat.

The entire industry shifted. Patients looked softer, more natural, less “filled.” But here's the interesting thing. Every decade we peel back another anatomical layer and discover an even deeper explanation. Skin wasn't the answer.

Fat wasn't the final answer either. Because beneath every fat compartment lies bone. And bone changes everything. The Face Doesn't Just Sag. It Shrinks. Ask almost any patient why they look older. “My face has dropped.” “My cheeks have fallen.” “My skin is loose.” They aren't wrong.

But they're describing the consequence, not the cause. Imagine hanging a tent over a frame. If the canvas becomes loose, the tent sags. Most injectors spend their careers pulling the canvas tighter. Adding filler.

Adding threads. Adding skin tightening. But what if the frame itself has become smaller? No amount of tightening the fabric will restore the original shape. You have to rebuild the frame.

That's exactly what happens in facial ageing. The skeleton slowly retreats. The face literally shrinks from the inside out. Bone Is Alive One of the biggest misconceptions in medicine is that bone is permanent. It isn't.

Bone is one of the most metabolically active tissues in the human body. Every second of every day, specialised cells are working. Osteoblasts build bone. Osteoclasts remove bone.

Throughout childhood and early adulthood, these two teams work beautifully together. Formation equals resorption. The skeleton constantly renews itself. Then age changes the rules. Osteoclasts slowly begin winning. Bone is removed faster than it is rebuilt. The result isn't dramatic. Nobody wakes up looking twenty years older. It happens millimetre by millimetre.

Year after year. Until one morning someone says, "I don't recognise myself any more." The Face Ages In Three Dimensions This is where many injectors make a fundamental mistake. They think ageing is vertical.

Everything falls. Gravity. End of story. But facial ageing occurs in three dimensions. The skeleton moves backwards. The fat compartments become smaller. The retaining ligaments become relatively tighter.

The skin loses elasticity. Everything is happening simultaneously. Imagine demolishing the foundations of a house while shrinking the bricks and stretching the roof. That's facial ageing. No single treatment can solve every layer. Bone Directs Every Layer Above It This is perhaps the most important concept in modern facial anatomy.

Bone dictates where the deep fat sits. Deep fat supports the SMAS. The SMAS suspends the superficial fat. Superficial fat supports the skin. The skin reflects everything underneath.

Every layer depends upon the one below it. Which means when bone disappears, everything above it changes. You don't simply lose bone. You lose projection. Support. Ligament tension.

Fat position. Skin drape. Even the direction that muscles pull begins to change. One millimetre of skeletal resorption can produce five millimetres of visible ageing. The Orbit — Why Eyes Suddenly Look Older Patients rarely complain, "My orbital aperture has enlarged." Instead they say, "I look tired," or "My bags appeared overnight." The problem isn't the skin.

The eye socket slowly becomes larger, particularly along its lower and outer margins. The bony platform supporting the lower eyelid retreats. Imagine removing the edge from a shelf. Everything resting on it begins to overhang.

The orbital fat hasn't suddenly become larger; it has lost its support.

At the same time, the tear trough becomes deeper because the skeleton beneath it has disappeared. The eye hasn't changed. The frame holding it has. The Pyriform Aperture — The Centre of Facial Ageing If I could inject only one area in facial rejuvenation, it would often be the pyriform aperture.

Not because patients ask for it — most have never heard of it — but because it quietly influences almost everything around it. As the maxilla resorbs, the base of the nose loses support. The nasal tip rotates downwards.

The upper lip becomes longer. The cheeks appear flatter. The nasolabial fold deepens. Most injectors chase the fold. Experienced injectors rebuild the foundation underneath it. The fold is only the shadow.

The skeleton created it. The Mandible — The Vanishing Jawline A young jaw is sharp. Defined. Confident. Age slowly rounds it. The gonial angle opens. The mandibular body shortens. The chin retreats. Suddenly the platysma has less support.

The SMAS loses its posterior anchor. Jowls appear. The neck softens. Patients often ask for neck tightening. Sometimes what they're really asking for is their skeleton back. Men and Women Do Not Age the Same This is one of the reasons aesthetic medicine is so fascinating.

Male and female skeletons follow different biological clocks. Men usually begin with larger facial bones and greater cortical thickness. Their testosterone declines gradually. Bone loss occurs slowly. Women experience something entirely different.

Menopause changes everything. Oestrogen protects bone. When it disappears, bone resorption accelerates dramatically. It's one of the reasons so many women say, "I suddenly aged." They're not imagining it. The face often changes more in five years after menopause than during the previous fifteen. The scaffolding retreats.

Everything above it follows. Why Bone-Level Filler Looks So Natural People often think placing filler on bone is about adding volume. It isn't. It's about restoring structure. When you inject a high-G-prime filler supraperiosteally (directly on the bone), you are not trying to make the face bigger.

You are trying to make the bone behave as if it hasn't resorbed. You are lifting the tent by raising the pole, rather than stuffing the canvas. This is why bone-level injections, when done correctly, are almost undetectable. The

patient doesn't look "filled." They simply look like they did ten years ago.
They have their scaffolding back.



CHAPTER 16

The Deep Fat — The Foundation of Youth

If bone is the concrete foundation of the house, deep fat is the load-bearing walls. We used to think of facial fat as a single layer. We thought it just hung there, slowly sagging under the weight of gravity. We were wrong.

Superficial fat compartments (left, dashed) give surface contour and descend with age; the deep compartments (right, solid) provide structural support and are the target for lift.

Facial fat is highly compartmentalised, and it is divided into two distinct systems: superficial and deep. The SMAS is the dividing line. Everything above the SMAS is superficial fat. Everything below the SMAS, resting directly on the periosteum or deep fascia, is deep fat. And here is the crucial difference: superficial fat sags.

Deep fat deflates.

The Deflation Story Deep fat compartments are tightly bound. They do not slide around the face. They stay exactly where they are. But as we age, they shrink. They lose volume like a balloon slowly letting out air. When a deep fat compartment deflates, two things happen. First, the area directly above it hollows out.

Second, the superficial fat that was resting on top of it loses its support and begins to slide downward. This is the dual mechanism of ageing: deflation deep down, descent on the surface. If you try to fix descent by adding more weight to the surface, you make the problem worse.

You have to address the deflation before you chase the descent. But — and this is where I part company with most of what is taught — addressing it does

not have to mean refilling the pad that deflated. Hold that thought; I come back to it at the end of this chapter, because it is the single most consequential disagreement in this book.

The Deep Fat Compartments You do not need to memorise every single fat compartment in the face, but you must understand the architecture of the deep ones. They are the true structural support system of the face. 1.

The Deep Medial Cheek Fat (DMCF) This is the holy grail of midface rejuvenation. The DMCF sits right on the maxilla, beneath the SMAS and the superficial malar fat pad. When we are young, it is plump and robust, pushing the midface outward and upward, creating the classic “apple cheek.” When it deflates, the midface flattens, and the superficial fat crashes into the nasolabial fold.

2. **The Sub-Orbicularis Oculi Fat (SOOF)** The SOOF sits just below the orbital rim, beneath the orbicularis oculi muscle. It provides the platform for the lower eyelid. When it deflates, the tear trough deepens and the eye looks tired. 3.

The Deep Temporal Fat The deep temporal fat pad sits beneath the deep temporal fascia, resting on the temporal bone. When it deflates, the temple hollows out, the lateral brow drops, and the face takes on a skeletal appearance. 4.

The Deep Pyriform Space (Ristow’s Space) A small but critical pocket of deep fat sitting right next to the nose. Deflation here causes the base of the nose to drop and the upper nasolabial fold to deepen dramatically. 5. **The Retro-Orbicularis Oculi Fat (ROOF)** Located under the brow, beneath the orbicularis oculi muscle. It provides the soft, youthful cushion to the upper eyelid and brow. When it deflates, the brow bone becomes sharp and the eyes look hollow.

How much fat is actually down there Before the argument, the numbers, because people quote them at me and it is worth knowing what they do and do not say.

Across the face and cheek, the most defensible working estimate is roughly **56 to 58 per cent superficial fat and 42 to 44 per cent deep** — about a 1.3:1 ratio. Raskin and LaTrenta dissected ten fresh hemiacadavers and weighed fat above and below the SMAS: 57 per cent above, 43 per cent below, with about three quarters of the deep mass sitting in the anterior and middle cheeks. Boehm and colleagues, imaging nineteen patients on CT over a mean of 11.3

years, found 26.10 ml superficial midface fat against 11.01 ml labelled deep plus 9.36 ml buccal — which, if you group buccal with the rest of the deep pool, reproduces almost exactly that same 56:44 split. Tower and colleagues, in 109 patients, found 4.26 ml superficial cheek fat against 3.04 ml deep: 58 to 42.

Two things about those numbers deserve emphasis.

First, the ratio is **region-dependent, and in places it reverses**. Cotofana's forehead work found roughly 9.0 ml across the superficial compartments against 12.1 ml deep — the opposite of the cheek relationship. There is no single facial ratio, and anyone quoting one at you as though it were a constant has not read carefully.

Second — and this is the point — **native tissue volume is not a treatment-volume prescription**. That roughly 43 per cent of facial fat lies deep does not mean 43 per cent of your syringe should go deep. That a compartment has lost volume does not oblige you to replace that volume in that compartment. Those are separate claims, and conflating them is how the industry talked itself into refilling everything it could measure.

Boehm's cohort also showed the deep compartment losing a greater *proportion* of its volume than the superficial — 18.4 per cent against 11.3 per cent — which is exactly the statistic used to justify deep refilling. Note what it does not establish: that refilling the deep pad is the best way to correct what the patient sees. Tower's longitudinal subgroup, meanwhile, *gained* an average 0.23 ml of deep cheek fat over 10.3 years, possibly from buccal fat descending into the measured region. Not every age-related contour change is simple atrophy of the thing beneath it.

Here is the line I would have you take from all of it: **deep anatomy explains the face. It does not dictate where every syringe must**

go.

The Great Deep Fat Fallacy For years, the industry taught a very simple equation: if a deep fat pad deflates, you must inject filler into that pad to replace it, millilitre for millilitre. I disagree, and I have built nearly twenty years of practise on disagreeing.

Here is the distinction that matters, and it is the spine of this whole book: *anatomical ageing is not a treatment target*. That a deep fat pad has deflated does not mean the deflated pad is the thing you should treat. Those are two

different claims, and the industry has spent years confusing them.

I correct from the two ends of the stack instead — a little projection on the bone below, contour in the subcutaneous above — and I leave the deep pad itself alone. A small, firm deposit on the periosteum changes the geometry of everything sitting on top of it; the surface effect is far larger than the volume you placed, which is exactly why you do not need to refill the pad. The deep fat, as a routine target, is out of scope — not because it does not age, but because chasing it is rarely the most elegant way to fix what the patient actually sees. I make the full version of this argument in the companion journal paper. Here is the short version: treat the consequence, not the compartment.

Because ageing creates a surface contour deficit, not simply a “deep fat volume deficit.” The plane in which you replace volume should be the one that most predictably restores the external contour while remaining safe. When a deep fat pad atrophies, it is not an isolated event.

The retaining ligaments become apparent, the superficial fat descends, the skin loses elasticity, and the bone remodels. The visible mid-cheek groove is therefore not a direct window into an empty deep fat pad. It is the final result of all these changes. Imagine a mattress.

If one of the internal springs weakens, you can either replace the broken spring (deep filler), or add a carefully shaped topper (superficial filler). If the goal is a smooth sleeping surface, the topper may achieve that better, even though the underlying spring remains unchanged.

When filler is placed in the superficial subcutaneous plane over the mid-cheek, it directly fills the visible depression, smooths transitions, creates a continuous contour, and camouflages ligament-related grooves. It is often easier to shape precisely. Clinically, many injectors find that this produces a more natural result than aggressively filling deep compartments alone.

There are limits, and honesty demands I state them plainly. There are faces where a deep deficit genuinely dominates — marked medial cheek deflation, significant temporal hollowing, certain infraorbital shapes — and in those, selective deep treatment is the right call. Those are the exception, and they sit at the advanced, sometimes surgical, end of the work. They are not the patient in front of you on a Tuesday afternoon. For the vast majority, the two ends of the stack are the safest, most effective place to be. We are not trying to recreate a 25-year-old’s anatomy.

So the question at the consultation is not “which compartment has aged?” It is a diagnostic one, and it runs like this: is the visible change driven by superficial volume and contour, by deep support, by skeletal projection, by tissue displacement — or by some combination? Answer that, and the treatment chooses itself.

What you find	What it usually calls for
Superficial contour deficiency	Superficial subcutaneous correction, cannula
A transition or shadow without real deep deficit	Superficial blending, not deep reconstruction
Local skeletal underprojection	Selective structural correction on bone
Genuine deep support deficit driving the appearance	Selective deep treatment may be considered
Descent with adequate volume	Do not assume filler is the answer at all
Excess superficial volume, heaviness	More filler — at any depth — will worsen it
Distortion from previous filler	Diagnose and correct what is there before adding

Notice that only one row of that table sends you deep, and it is conditional. That is not timidity; it is the diagnosis doing its job.

It is worth adding that even the literature advocating multilayer correction does not inject in the proportions the anatomy might suggest. In published midface examples from the multilayer school, the authors used 0.7 ml deep against 3.0 ml superficial in one case, and 1.2 ml deep against 2.4 ml superficial in another. Those are illustrations, not dosing guidance — but they make the point that even an explicitly deep-and- superficial philosophy does not treat native fat ratios as a prescription. We are trying to optimise the face they have today.

PART III

Materials, Tools and Technique

CHAPTER 17

The Architect vs. The Archaeologist

If you attend enough aesthetic conferences, you will eventually notice a pattern. The speakers stand on stage, pointing at 3D anatomical models, and declare that the only way to treat an ageing face is to identify exactly which deep fat pad has deflated and refill it. They call it “anatomical restoration.” It sounds incredibly scientific. It sounds precise. It is also fundamentally flawed.

The Illusion of Restoration The aim of facial rejuvenation is not to recreate a patient’s 25-year-old anatomy. It is to create the most harmonious, natural-looking face for the anatomy they have today. Ageing affects every single layer of the face. The bone resorbs and shrinks.

The retaining ligaments stretch. The deep fat deflates. The superficial fat descends. The muscles change their resting tone. The skin loses collagen, elastin, and hydration. Because the entire underlying framework has changed, there is absolutely no evidence that recreating youthful anatomy, compartment by compartment, is the optimal aesthetic solution.

If the bone beneath the fat pad has shrunk, simply refilling the fat pad will not produce the same surface contour it did twenty years ago. The foundation has moved.

If you put a 25-year-old’s fat pad on a 50-year-old’s skeleton, you do not get a 25-year-old face. You get a strange, uncanny hybrid. You get a face that looks “done.” Our role is not to rebuild every deep fat compartment exactly as it once was.

Our role is to restore balance, contour, and proportion.

Architecture vs. Archaeology The difference between a good injector and a great one is the difference between an archaeologist and an architect. Archaeology tries to reconstruct exactly what was there before. The

archaeologist digs through the layers, finds a missing piece of the deep medial cheek fat, and tries to put it back exactly where it was.

They are obsessed with the past. They look at an MRI scan of a youthful face and try to force the ageing face to match it. Architecture designs the most functional and beautiful structure using the foundations that exist today. The architect looks at the current skeleton, the current ligaments, the current skin quality, and asks: What is the most elegant way to support this structure right now?

An ageing face is like a historic building. You don't restore it by replacing every brick exactly where it once was. You strengthen the structure, improve its proportions, and preserve its character. The result should respect the building's history while making it beautiful and functional for today.

Sculpting Light and Shadow Injecting fillers is not simply replacing missing tissue; it is sculpting light, shadow, and facial harmony. Two patients with identical deep fat atrophy may require completely different treatments because their skeletal structure, skin quality, ligament support, and aesthetic goals are different. One may need deep structural support on the zygoma.

The other may simply need a smooth, superficial subcutaneous wash of filler to camouflage a ligamentous groove. If you are obsessed with being an archaeologist, you will treat both patients exactly the same way. You will push a needle down to the bone and inject.

And you will wonder why one patient looks rejuvenated and the other looks like a chipmunk. When you look at a face, you are not looking at anatomy. You are looking at light.

A shadow under the eye makes a patient look tired. A shadow under the cheekbone makes them look gaunt. A shadow at the oral commissure makes them look sad. The architect asks: How do I manipulate the tissue to change how the light hits this face?

Sometimes that means a deep periosteal bolus to project a cheekbone into the light. Sometimes it means a superficial cannula wash to blend a shadow. The anatomy dictates the tool and the safety profile, but the light dictates the goal.

The Subcutaneous Advantage This is why I advocate so strongly for the subcutaneous plane. Current anatomical research clearly demonstrates that visible facial ageing results from changes in bone, ligaments, muscles, superficial fat, deep fat, and skin — not deep fat alone. Most recommendations for deep

fat pad replacement are based on anatomical rationale and expert practice rather than randomized comparative trials showing it is superior to superficial correction in every patient.

Deep fat pads may atrophy with age, but our objective is not to recreate every anatomical compartment. Our objective is to restore the external three-dimensional contour of the face. In many patients, this can be achieved more predictably by replacing volume in the superficial subcutaneous plane overlying the visible deficit rather than by attempting to refill the deep fat pad itself.

The optimal injection plane should be chosen according to the desired surface contour, safety, and tissue mechanics — not solely according to which compartment has lost volume. If a bridge is sagging because a pillar has weakened, the archaeologist demands you rebuild the pillar. The architect realises that you can simply reinforce the suspension cables from above, achieving the exact same functional result with half the risk and half the materials.

Understand anatomy like a scientist. Inject like an architect. Judge your results by harmony, not by whether you've replaced every structure that has aged.

CHAPTER 18

Needle vs. Cannula — The Only Rule That Matters

The aesthetic industry loves a religious war. Botox vs. Dysport. Monophasic vs. Biphasic. But the loudest, most tedious argument of all is Needle vs. Cannula. Half the conference hall insists the needle is the only way to achieve true precision. The other half claims the cannula is the only way to sleep at night without dreaming of necrosis.

They are both missing the point. The choice between a needle and a cannula is not a stylistic preference. It is not about what you feel “comfortable” with. It is a decision dictated entirely by the anatomical plane you are trying to reach.

The Only Rule That Matters Here is the rule. Write it down. Memorise it. Never violate it. If you are injecting the dermis or the periosteum, use a needle. If you are injecting the subcutaneous fat, use a cannula. That is it.

That is the whole debate resolved in two sentences. But to understand why this rule is absolute, you have to understand the micro-anatomy of the face — specifically, the size of the blood vessels, the nerves, and the tools you are holding in your hand.

The Mathematics of Safety A 27G cannula has an outer diameter of 0.40 mm. A 25G cannula has an outer diameter of 0.50 mm. Now, let’s look at the blood vessels. In the dermis, the blood vessels are microscopic capillaries. They are measured in micrometres (μm).

An average dermal capillary is roughly 0.01 mm in diameter. When you put a 30G needle (0.30 mm diameter) into the dermis, it is 30 times larger than the vessels around it. It pushes them aside.

It might nick one, causing a tiny pinpoint bleed, but it cannot enter the vessel and cause a systemic occlusion. The physics simply do not allow it. The vessel is too small; the needle is too big. This is why dermal injections (like skin boosters or fine-line filling) are incredibly safe from a vascular perspective, provided you are truly in the dermis.

But when you move down into the subcutaneous layer, the architecture changes.

The subcutaneous fat is the superhighway of the face. This is where the major facial arteries and veins travel before branching off. The facial artery, the angular artery, the infraorbital artery — these vessels have diameters ranging from 1.0 mm to 2.5 mm. If you put a sharp needle into the subcutaneous layer, you are putting a sharp object into a space filled with tubes that are larger than the object itself.

It will slide right into the lumen of the artery. If you push the plunger, you will fill the arterial tree with hyaluronic acid. That is how necrosis happens.

That is how blindness happens. This is why we use a cannula in the subcutaneous space. A blunt-tipped 25G or 22G cannula will encounter a 1.5 mm artery and, because it has no cutting edge, it will bounce off it. It will slide past it.

It will navigate the superhighway without puncturing the pipes.

The Tactile Feedback of the Cannula The cannula is not just a safety device; it is a sensory instrument. When you are in the subcutaneous plane, you cannot see what you are doing. You have to feel it.

As you glide a cannula through the subcutaneous fat, you will feel different types of resistance: • **Loose Areolar Tissue:** A smooth, buttery glide. This is the safe zone.

• **Small Fibrous Septa:** Gentle, intermittent resistance. A slight push will pop you through. • **Retaining Ligaments:** A firm, springy resistance. You will feel a distinct “click” or “pop” as the blunt tip pushes past the ligamentous band. • **Nerves:** If a cannula bumps a major sensory nerve, the patient will feel a sudden, sharp “zing” or electric shock.

Because the cannula is blunt, it rarely damages the nerve permanently — it simply alerts you to its presence so you can redirect. A sharp needle would slice right through it.

The Cannula Transfer-of-Skill

This tactile feedback leads to one of the most important concepts in injectable training: the transfer-of-skill.

Injectors often look at the face as a series of terrifyingly separate procedures. They think, *I know how to do a nasolabial fold, but I don't know how to do a cheek*. They view the cheek as a completely different animal, requiring a completely different set of hands.

This is a mental block. The subcutaneous layer is continuous. The fat in the nasolabial fold feels exactly the same as the fat in the cheek. The glide is the same. The resistance is the same. The vessels behave the same way.

Once you know the cannula, it doesn't matter where you are putting it in. It is easy to do the rest. You can watch videos of cheek injections, or temple injections, or jawline injections, but the physical skill is already in your hands.

If you haven't done cheeks, that does not mean you will not do cheeks confidently. You just need to have done enough nasolabial folds with a cannula, fanning it out towards the cheek, to understand the plane. Once you feel that smooth, buttery glide in the nasolabial fold, you possess the exact physical skill required to treat the midface. The anatomy changes, but the tissue mechanics do not.

This is why mastering the cannula early is non-negotiable. It unlocks the entire face.

The Exception: The Periosteum If the subcutaneous layer belongs to the cannula, and the dermis belongs to the needle, what about the deepest layer? What about the bone? When we want to build structural support — the cheeks, the pyriform aperture, the chin — we need to place high-G-prime filler directly onto the periosteum.

For this, we return to the needle. Why? Because a cannula cannot easily pierce the SMAS and the deep fascia to reach the bone cleanly without causing unnecessary tissue trauma along the way. A sharp needle, inserted perpendicular to the skin, passes cleanly through the dermis, through the subcutaneous fat, through the SMAS, and taps firmly onto the bone. Is it dangerous? It can be, if you are sloppy. But if you know your anatomy, you know where the major foramina (the holes in the bone where the big vessels and nerves emerge) are located.

You avoid the infraorbital foramen. You avoid the mental foramen. You touch the bone, you aspirate to prove you are not in a vessel, and you inject a slow, steady bolus.

The Summary Do not argue about which tool is better. Argue about which plane you are in. • Dermis: Needle. The vessels are too small to occlude. • Subcutaneous: Cannula. The vessels are large, and the blunt tip will slide past them. • Periosteum: Needle. Clean, direct access to the structural foundation. Master the planes, and the tools choose themselves.

One warning before you leave this chapter, because it is the mistake I see most often in people who have just fallen in love with the cannula.

A cannula does not keep you superficial. It keeps you blunt.

Those are not the same thing.

The plane a cannula occupies is decided by the layer you introduced it into and the layer its tip subsequently tracks through — not by the instrument itself, and not by the depth of your introducer nick. Two millimetres or three at the entry tells you very little about where the tip ends up eight centimetres later. Cadaver work has demonstrated blunt cannulas achieving deep and even supraperiosteal placement without difficulty. A cannula can cross fascial interfaces and enter deep spaces, and while the vascular-occlusion signal is generally lower than for sharp needles, cannulas are not incapable of penetrating a vessel or producing a serious vascular complication.

So the sentence “I used a cannula, therefore I was safe and superficial” is not a safety argument. It is an assumption wearing the costume of one. If you genuinely want to know which plane your habitual technique occupies in a given region, put an ultrasound probe on it and watch. Tactile feedback is worth a great deal, and it is not proof.

CHAPTER 19

Rheology — The Physics of Filler

If you ask a room full of injectors how they choose a dermal filler, you will hear a lot of marketing disguised as science. “I use this one for cheeks because it gives a nice pop.” “I use this one for lips because it feels soft.” “I use this one because the rep brought me coffee and bagels.” This is not medicine.

This is guesswork. If you are going to inject a foreign substance into a patient’s face — a face that moves, smiles, chews, sleeps on its side, and ages — you need to understand exactly how that substance behaves under physical stress. You need to understand rheology.

Rheology is the study of how materials deform and flow under mechanical stress. In aesthetics, it is the language we use to predict how a hyaluronic acid (HA) gel will behave once it leaves the syringe and enters the tissue. Will it stay where you put it, or will it spread?

Will it lift the tissue, or will it be flattened by the muscle above it? Will it move naturally when the patient smiles, or will it look like a rigid lump under the skin? To answer these questions, you must stop looking at the brand name on the box and start looking at the physics inside the syringe.

The Four Pillars of Rheology There are four primary measurements that define how a filler behaves. If you understand these, you can predict the clinical outcome of almost any product on the market.

1. **Elastic Modulus (G')** — “Firmness and Lift” G' (pronounced “G prime”) measures the elastic properties of the gel. Specifically, it measures the gel’s ability to recover its original shape after a shearing force is applied. Think of G' as the “firmness” or “stiffness” of the filler.

It represents the energy stored in the material. When a muscle presses down on the filler, does the filler push back, or does it flatten out? A filler with a high

G' is firm. It resists deformation.

It holds its shape. This is what you want when you are injecting deep onto the bone to mimic the skeleton — the jawline, the chin, the pyriform aperture, the zygoma. You need a high G' filler to lift the heavy tissues above it. A filler with a low G' is soft.

It yields easily to pressure. This is what you want in areas of high dynamic movement or thin tissue — the lips, the tear trough, the superficial perioral lines. If you put a high G' filler in the lips, it will feel like a rubber tire and look unnatural when the patient speaks.

2. Viscous Modulus (G'') — “Flow and Spread” G'' (pronounced “G double prime”) measures the viscous properties of the gel. It represents the energy lost during deformation. If G' is how much the gel bounces back, G'' is how much the gel permanently deforms and flows.

It describes the inability of the filler to recover its shape. HA fillers generally have a low G'' . They need to be viscous enough to flow through a needle and be molded immediately after injection, but elastic enough to stay in place once molded. If a filler had a very high G'' , it would act like honey — it would flow everywhere and provide zero structural support.

3. Tan Delta ($\tan \delta$) — “The Ratio” Tan delta is simply the ratio between the viscous component and the elastic component (G'' divided by G').

It tells you whether the filler behaves more like a liquid or more like a solid. • If $\tan \delta > 1$, the filler is mostly viscous (liquid-like).

- If $\tan \delta < 1$, the filler is mostly elastic (solid-like). Almost all crosslinked HA fillers have a $\tan \delta$ less than 1. A very low $\tan \delta$ (e.g., Juvéderm Volux at 0.074) means the product is highly elastic and solid-like — perfect for a sharp jawline.

A higher $\tan \delta$ (e.g., Belotero Balance at 0.641) means the product is softer and more fluid — perfect for superficial blending. 4. Cohesivity — “Sticking Together” Cohesivity is arguably just as important as G' , but it is often misunderstood. Cohesivity measures the internal adhesion forces of the gel.

How well does the gel stick to itself?

Imagine a dollop of honey and a dollop of gelatin. Both might feel somewhat firm to the touch, but if you press them between your fingers and pull your fingers apart, they behave differently. The honey strings out and stays connected to itself (high cohesivity).

The gelatin breaks apart into chunks (low cohesivity). When a filler is subjected to the compressive forces of the face (like the cheek muscles contracting), a filler with high cohesivity will resist separating. It will maintain its integrity as a single deposit. A filler with low cohesivity might spread, separate, or migrate.

- High G' , High Cohesivity: Firm, lifts well, stays together. Ideal for deep structural support (chin, jawline).
- High G' , Low Cohesivity: Firm, but can break apart under pressure.
- Low G' , High Cohesivity: Soft, flexible, but stretches and moves as a single unit without migrating.

Ideal for dynamic areas (lips, superficial cheeks).

- Low G' , Low Cohesivity: Soft, spreads easily, blends seamlessly. Ideal for the tear trough or superficial fine lines.

Water Uptake (Swelling Factor)

While not strictly a rheological property, water uptake is a critical physicochemical characteristic. HA is a highly hydrophilic molecule.

Once injected, it pulls water from the surrounding tissue. The amount of water it pulls depends on the HA concentration and the degree of crosslinking. A tightly crosslinked gel cannot expand much, so its water uptake is low. A loosely crosslinked gel can expand significantly.

You must know a filler's water uptake before you inject it near the eye. The periorbital area is highly sensitive to minimal volume changes and prone to lymphatic compromise. If you inject a filler with high water uptake (like Juvéderm Ultra) into the tear trough, the patient will return in three months looking like they haven't slept in a decade, plagued by chronic malar oedema.

For the tear trough, you need a filler with low water uptake (like Teosyal Redensity II or Belotero Balance). A Personal Clinical Note: The Reality of RHA Fillers in the Lips Science is beautiful in a laboratory. But the face is not a laboratory. The rheological data will tell you one thing; clinical experience will occasionally teach you another.

Let me give you a specific example from my own practice regarding the lips. The lips are an incredibly demanding environment. They are highly vascular, highly dynamic, and subjected to constant shearing and compressive forces from speaking, eating, and kissing. You need a filler that is soft enough to feel natural, cohesive enough to stay in place, and elastic enough to move seamlessly with the orbicularis oris muscle.

When the Teosyal RHA (Resilient Hyaluronic Acid) line was introduced, it was marketed specifically for dynamic areas. The manufacturing process

preserves longer chains of HA, creating a mobile, stretchy network. On paper, RHA3 looks like an excellent choice for a structured lip. It has a moderate G' (264 Pa) and high cohesivity. And indeed, when you inject RHA3 into the lips, it feels fantastic on day one. It gives beautiful projection, it holds the border well, and the initial integration seems flawless.

But over time — usually around the three-to-four-month mark — I noticed a pattern. The RHA3 in the lips begins to stiffen. The dynamic flexibility that was present at the beginning starts to fade. The lips feel firmer, slightly more rigid, and less natural to the patient.

The rheology at the time of injection does not perfectly predict the rheology at month four in this specific high-movement environment. Because of this stiffening effect over time, I have completely shifted my preference. I now prefer RHA2 (or equivalent softer fillers) for the lips. RHA2 has a slightly higher G' initially (319 Pa), but its overall crosslinking profile and behaviour in the tissue mean it maintains a much softer, more natural feel over the long term. It integrates beautifully and does not develop that late-stage rigidity. This is why you must track your patients over years, not just weeks.

The data table gets you into the right ballpark; your clinical observation refines the pitch. The Master Rheology Table To practise aesthetic medicine at a high level, you should have a general understanding of where your preferred products sit on the rheological spectrum. The following table provides the standardized in vitro measurements for the most common HA fillers on the market.

Keep in mind that different studies use different rheometers and testing parameters, so you can only truly compare numbers if the products were tested under the exact same conditions (as they were in this dataset).

Product by layer — a working quick-reference

Layer	What the gel must do	Rheological profile	Typical use
Dermis (needle, perpendicular)	Sit shallow without ridging or showing	Low G', low cohesivity, low water uptake	Fine lines, skin quality, tear trough
Subcutaneous fat (cannula, along the plane)	Spread and blend, integrate as a unit	Low-moderate G', higher cohesivity	Volume, contour, blending over an area
Periosteum / bone (needle, on bone)	Hold shape and project against tissue weight	High G', high cohesivity	Structural projection: cheekbone, chin, jaw angle

Populate the product names into this table from your own standardised dataset — the profile column, not the brand, is what should drive the choice.

Master Rheology Table — standardised in-vitro values

Publisher's note: the rheology dataset below is being re-typeset from the source publication for this edition. Values are drawn from Hee et al. and de la Guardia et al., Facial Plast Surg 2022, in which all products were tested under identical standardised methodologies at 5 Hz. Because these are precise clinical figures, the full per-product table is being verified directly against the source rather than reproduced from a secondary summary. Refer to the cited papers for the complete dataset.

The key relationships to carry from that dataset are the ones that change your product choice:

G' (elasticity / firmness) ranks the products by how well they resist deformation and lift tissue. The high-G' products (for example Juvéderm Voluma, Restylane Lyft, Juvéderm Volux) are your structural, suprapariosteal choices. The low-G' products (for example Belotero Balance, the softer Teosyal

RHA grades) are your superficial, blending choices.

Tan (G''/G' — fluidity) is the single most useful number for matching a filler to a plane. A low tan means a firm, elastic, solid-like product that holds a sharp edge — a jawline or chin. A higher tan means a softer, more fluid product that spreads and blends — superficial cheek, fine lines. As a worked reference point, Belotero Volume+ sits at a low **0.074**, while Belotero Balance sits at a high **0.641** — the two ends of that spectrum within a single manufacturer's range.

Cohesivity describes how well the product holds together as a bolus versus spreading. High cohesivity supports and projects; low cohesivity integrates and blends.

(Data adapted from Hee et al., and de la Guardia et al., *Facial Plast Surg* 2022. All products tested under the same standardized methodologies at 5Hz.)
Putting It Into Practice How do you use this information? You match the physics of the filler to the anatomy of the face.

The Upper Face (Temples, Forehead)

The temporal fossa requires deep volume restoration. You need a filler that resists deformation and provides structural lift. Look for high G' and medium-to-high cohesivity (e.g., Juvéderm Voluma, Restylane Lyft).

The Midface (Cheeks, Zygoma)

The zygomatic arch is subject to the dynamic contraction forces of the lip and cheek elevators.

The filler must resist shearing and withstand the compression of the overlying tissue to maintain projection. You need a high G' and high cohesivity (e.g., Juvéderm Voluma, Teosyal RHA4).

The Tear Trough

This is thin tissue over bone. The filler must spread easily, not form lumps, and above all, not pull in water.

You need low G' , low cohesivity, and very low water uptake (e.g., Teosyal Redensity II).

The Lower Face (Jawline, Chin)

This is an area of high compression, with taut skin and muscle over bony structures. To mimic the mandible, the filler must have maximum resistance to deformation to prevent lateral spreading and maintain a sharp vertical projection.

You need the highest G' available combined with high cohesivity (e.g., Juvéderm Volux, Restylane Lyft). The LipsAs discussed, the lips require a delicate balance. You want low-to-medium G' for softness, but enough cohesivity to prevent the product from migrating beyond the vermilion border. And you want a low swelling factor to avoid the dreaded “sausage lip” look (e.g., Restylane Kysse, Teosyal RHA2).

The Final Rule of Rheology Rheology is not a marketing gimmick. It is the biomechanical blueprint of your treatment. If you place a soft, low G' filler on the periosteum of the chin, it will flatten out under the pressure of the mentalis muscle, providing zero projection and making the chin look wider and doughy.

You have failed the patient. If you place a stiff, high G' filler into the superficial dermis of the nasolabial fold, it will look like a piece of wire trapped under the skin every time the patient smiles. You have failed the patient.

Learn the physics. Respect the anatomy. Match the two together. That is how you become a master injector.



CHAPTER 20

Nobody Teaches This — Small-Volume Field Blocks

There is a strange culture of martyrdom in aesthetic medicine. We slather numbing cream on a patient's face, wait twenty minutes, wipe it off, and then proceed to push needles and cannulas through their tissue while they grip the chair in silent agony. We tell them, "Just a little scratch," as if pretending it doesn't hurt somehow makes it true.

It is barbaric. And it is entirely unnecessary. Topical anaesthetic cream penetrates about two millimetres into the skin. It numbs the epidermis and the very superficial dermis. If you are doing microneedling or superficial skin boosters, it works perfectly. But if you are using a cannula in the subcutaneous space, or placing a needle on the periosteum, the skin cream is doing absolutely nothing.

The cream is sitting on the roof. You are working in the basement. The solution is the small volume field block. It is the most underrated, underutilised skill in aesthetic medicine.

The Dentist Fallacy When most injectors hear "nerve block," they immediately think of dentistry. They picture intra-oral injections — pushing a needle through the mucosa of the upper lip to hit the infraorbital nerve, or through the lower gum to hit the mental nerve. It sounds invasive.

It sounds complicated. It sounds like something that belongs in a surgical theatre, not an aesthetic clinic. But you don't have to be a dentist. You don't have to go inside the mouth. The nerves we care about eventually come up to the surface. They have to, in order to innervate the skin.

You just have to catch them before they branch out.

The Ring Block Principle Look at what hair transplant surgeons do. When they need to numb the entire scalp for an eight-hour surgery, they don't block the spinal cord. They perform a ring block.

They inject a continuous line of local anaesthetic around the circumference of the scalp, just above the ears and eyebrows. Every sensory nerve travelling up to the top of the head has to pass through that ring of anaesthetic. The entire scalp goes numb.

We use the exact same principle in the face, just on a smaller scale. We place a small volume of lidocaine (0.1 to 0.2 ml) directly in the path of the sensory nerve as it emerges from the deep tissue. We block the signal before it reaches the area we want to treat.

The Common Aesthetic Blocks You do not need to memorise the entire trigeminal nerve tree. You only need to know how to block the specific territories where we do the most painful work. 1.

The Jawline Block (Mandibular/Mental Nerve) Jawline contouring with a cannula can be incredibly uncomfortable, especially as you navigate the retaining ligaments and the pre-jowl sulcus. Instead of torturing the patient, find the mental foramen. It sits on the mandible, usually in line with the second premolar.

Do not inject directly into the hole — that is how you damage the nerve. Inject 0.1 ml of lidocaine just above or adjacent to the foramen. Within two minutes, the entire chin and anterior jawline will be numb. You can now sculpt the prejowl sulcus and the chin without the patient flinching. 2. The Lower Lip Block The lips are the most sensitive area of the face. Numbing cream takes the edge off the initial needle prick, but the tissue expansion hurts. To block the lower lip, use the same mental nerve block described above. The mental nerve supplies sensation to the lower lip and chin.

Block it on both sides, and the lower lip goes completely numb. The patient will feel pressure, but no pain. 3. The Midface/Upper Lip Block (Infraorbital Nerve) The infraorbital nerve exits the maxilla just below the orbital rim, in line with the pupil. It supplies the lower eyelid, the cheek, the side of the nose, and the upper lip.

Again, do not go inside the mouth. Palpate the orbital rim, drop down about one centimetre, and you will feel the slight depression of the foramen. Inject 0.1 ml of lidocaine into the subcutaneous tissue overlying the foramen. Do not fish for the nerve.

Do not try to enter the hole. The anaesthetic will diffuse. The upper lip and midface will go numb, making nasolabial fold and cheek treatments entirely painless. 4.

The Forehead Block (Supraorbital/Supratrochlear Nerves) If you are doing deep lines in the forehead or placing threads, a block is essential. The nerves exit the skull just above the eyebrow. The supraorbital nerve is roughly in line with the pupil; the supratrochlear is slightly more medial.

A simple line of local anaesthetic injected subcutaneously just above the eyebrow (a mini ring block) will catch both nerves. The entire forehead, all the way up to the hairline, will be numb.

The Rules of the Block 1. Volume: Keep it small. 0.1 to 0.2 ml of 1% or 2% lidocaine (with or without adrenaline) is all you need per site. If you use too much, you will distort the anatomy and make your aesthetic assessment impossible. 2.

Location: Subcutaneous, never intra-foraminal. You want the fluid to bathe the nerve, not crush it inside its bony tunnel. 3. Aspiration: Always aspirate. Nerves and blood vessels travel together.

Where there is a foramen, there is an artery. 4. Time: Give it two minutes. Don't inject the block and immediately pick up your filler syringe. Wait for the magic to happen.

When you master the small volume field block, your practice changes. Your patients stop squirming. They stop sweating. They stop dreading their appointments. They let you take your time, which means you do better work. Pain is not a necessary part of the process. It is just a sign that you haven't learned how to control the field.



Making the Face Build Its Own — Biostimulation

Hyaluronic acid is a beautiful thing, but it is lazy. It sits exactly where you put it, holds water, and waits to be dissolved. It is a passive implant. Eventually, you will meet a face that doesn't need more volume. It needs a wake-up call. HA is exceptional for restoring volume, mimicking bone, and hydrating tissue. But it is fundamentally a passive implant. It sits in the tissue, holds water, and eventually degrades. It does not change the biological behaviour of the skin.

It does not make the tissue act younger. When the primary problem is not volume loss, but tissue quality — thinning skin, profound laxity, crepey texture, or dermal exhaustion — HA filler is the wrong tool. This is where biostimulation begins.

Biostimulators do not just fill space. They provoke a biological response. They trigger the body's own fibroblasts to produce new Type I and Type III collagen, new elastin, and new extracellular matrix. They force the tissue to rebuild itself. The Four Pillars of Biostimulation 1.

Poly-L-Lactic Acid (Sculptra) PLLA is the grandfather of biostimulators. It is a synthetic, biodegradable polymer that is injected deep into the dermis or superficial fat. The Mechanism: The body recognises the PLLA particles as foreign bodies and mounts a controlled, subclinical inflammatory response. Macrophages surround the particles, followed by fibroblasts, which begin laying down dense networks of new collagen. Over several months, the PLLA degrades into lactic acid and is exhaled as CO₂, leaving behind only the patient's own new collagen. The Patient: The patient with global volume loss, panfacial laxity, and thin skin.

Sculptra is not for spot-treating lines; it is for thickening the entire facial envelope. It requires patience (results take 3–6 months) and commitment (usually 3 sessions), but the results can last for over two years. 2.

Calcium Hydroxylapatite (Radiesse)CaHA consists of smooth calcium microspheres suspended in a gel carrier. The Mechanism: It provides immediate volume (from the gel), but as the gel degrades, the calcium microspheres act as a scaffold for fibroblasts to build new collagen. Radiesse is unique because it is highly structural — it has a very high G- prime, making it excellent for jawlines and cheekbones, but it can also be hyper-diluted and washed over the neck or décolletage purely for skin tightening without adding volume.

The Patient: The patient who needs both immediate structural lifting and long-term skin thickening. Excellent for the jawline, the hands, and the crepey neck. 3. Platelet-Rich Fibrin (PRF) and PRP This is autologous biostimulation — using the patient's own blood. The Mechanism: Blood is drawn and centrifuged to isolate the platelets, growth factors, and (in PRF) the fibrin matrix.

When injected back into the skin, these growth factors (PDGF, TGF- beta, VEGF) signal the local stem cells and fibroblasts to begin massive tissue repair and angiogenesis (new blood vessel formation). PRF forms a gel matrix in the tissue, releasing growth factors slowly over weeks, whereas PRP releases them rapidly.

The Patient: The patient who wants completely natural, chemical-free rejuvenation. Exceptional for the under-eye area where synthetic fillers carry a high risk of swelling, and for overall skin luminosity. 4.

Exosomes The newest frontier in regenerative aesthetics. Exosomes are not cells; they are the messenger vesicles secreted by stem cells. The Mechanism: Think of exosomes as envelopes full of instructions (mRNA, growth factors, cytokines). When injected or microneedled into the skin, they deliver these instructions directly to the patient's ageing cells, effectively telling them to behave like young cells again.

They dramatically reduce inflammation, accelerate healing, and trigger massive collagen and elastin production. The Patient: The patient with highly inflamed skin, rosacea, severe photoaging, or the patient who wants the regenerative power of stem cells without the blood draw required for PRF. The Paradigm Shift: Combination Therapy The modern aesthetic practitioner does not choose between HA fillers and biostimulators.

They use both, often in the same patient, sometimes in the same session. This is the concept of layered rejuvenation. You use HA filler deep on the bone to restore the skeletal structure (the foundation). You use a biostimulator like Sculptra or Radiesse in the superficial fat and deep dermis to thicken the tissue envelope and tighten the ligaments (the walls). You use Skin Boosters, Polynucleotides, or Exosomes in the superficial dermis to refine the texture and glow (the paint). A face that is simply inflated with HA filler looks like a balloon. A face that is structurally supported and biologically stimulated looks like it did ten years ago.

That is the difference between filling a face and regenerating it. The Big Three: Sculptra vs. Radiesse vs. Polynucleotides Not all biostimulators are created equal. They use different mechanisms, operate on different timelines, and are suited to entirely different patients. Poly-L-Lactic Acid (Sculptra) is a synthetic polymer that causes a controlled, subclinical inflammatory response.

The body encapsulates the PLLA particles with fibroblasts, which lay down massive amounts of Type I collagen. Over six to nine months, the PLLA degrades into lactic acid and is exhaled as CO₂, leaving behind only the patient's own collagen. The timeline is slow — the patient will look exactly the same at week four as they did on day one.

The real result hits at month three and peaks at month six. The right patient is the volume-depleted, gaunt patient who needs global pan- facial volumisation: the marathon runner, the post-menopausal patient with diffuse laxity. It is not for spot-treating; it is for rebuilding the entire foundation.

Calcium Hydroxylapatite (Radiesse) consists of CaHA microspheres suspended in a carboxymethylcellulose gel. It provides immediate one- to-one volume from the gel carrier, while the CaHA spheres stimulate fibroblasts to produce both Type I collagen and elastin over the next three months. When hyper-diluted, it acts purely as a biostimulator without adding volume.

The right patient is one who needs structural contouring (jawline, cheekbones) but also has poor skin quality. It is stiffer than HA, providing excellent projection while simultaneously thickening the dermis over time. Polynucleotides (PN/PDRN) are derived from salmon or trout DNA. Unlike PLLA or CaHA, which rely on an inflammatory response to trigger collagen, polynucleotides work at the cellular level. They bind to adenosine receptors on the fibroblast, upregulating its metabolism, increasing hydration, and scavenging free radicals. It is cellular medicine, not an inflammatory trigger.

The timeline is faster than PLLA: initial glow at week two, structural improvement at weeks four to eight. The right patient is one with thin, crepey, sun-damaged skin who does not want or need volume — the under-eye area, the neck, the patient with rosacea where you do not want to trigger an inflammatory biostimulator.

The Golden Rule of Biostimulation Biostimulators rely on the patient's immune system and cellular health to generate the result. If you inject Sculptra into a 75-year-old heavy smoker with a poor diet, you will get a poor result. Their fibroblasts are exhausted; they cannot mount the necessary response.

Hyaluronic acid works regardless of the patient's health. Biostimulation requires the patient to meet you halfway. Patient selection is everything.



CHAPTER 22

The Long Game — Planning and Sequencing

If there is one thing that separates a master injector from a beginner, it is not the steadiness of their hand. It is not their choice of needle or cannula. It is not even their anatomical knowledge. It is their ability to plan.

The staged plan: structure first, then volume, then skin. Deep before superficial, cause before symptom.

A beginner looks at a patient who points to their nasolabial folds, picks up a syringe of filler, and injects the nasolabial folds. The patient leaves happy for a month, then returns complaining that their face looks heavy. A master looks at the same patient, explains why the nasolabial fold is actually a symptom of midface deflation, treats the cheekbones, strengthens the jawline, and ignores the fold entirely.

The patient leaves confused, wakes up the next morning looking five years younger, and stays with that practitioner for life. Treatment planning is the art of sequencing. It is understanding what must happen first, what must happen second, and what must never happen at all.

The Golden Rules of Sequencing There is a logical order to facial rejuvenation. If you violate this order, you will create strange, unnatural, or heavy results.

Rule 1: Treat Deep Before Superficial

You must rebuild the foundation before you paint the walls. If a patient has deep skeletal volume loss (deflated deep medial cheek fat, receding pyriform aperture) and you treat their superficial fine lines with skin boosters, you have

achieved nothing.

The face will still look tired. You must place your high-G-prime structural fillers deep on the bone first. Once the scaffold is restored, the superficial tissues will often drape so much better that the fine lines resolve themselves.

Rule 2: Treat Structure Before Volume

Volume without structure is just weight.

If a patient has a sagging jawline and poor cheekbone definition, and you inject their lips and tear troughs, you have simply added heavy focal points to a collapsing canvas. You must establish the structural frame — the zygomatic arch, the angle of the mandible, the chin — before you add soft volume to the centre of the face.

Rule 3: Treat the Cause, Not the Symptom

The symptom is almost always medial and inferior (the nasolabial fold, the marionette line, the jowl). The cause is almost always lateral and superior (the cheek, the temple, the pre-auricular area). Always pull before you fill. Always support before you inflate.

Stage, not age

Stage	What you see	The first move	What to resist
Prevention	No structural loss; early dynamic lines	Skin quality and sun protection; minimal, targeted toxin	Volume the face does not need yet
Early	Subtle midface flattening; lines at rest beginning	Support the foundation; begin collagen-directed skin work	Chasing individual lines with filler
Established	Clear midface descent; folds, jawline softening	Structure first — restore support, then refine the surface	Filling the symptom (the fold) and ignoring the cause
Advanced	Significant descent, skin laxity, volume loss throughout	Stage across visits; know the ceiling of what injectables can do	Trying to achieve with filler what needs surgery

Stage is not age. A well-preserved fifty-year-old may be at “early”; a sun-damaged thirty-five-year-old may be at “established.” Treat the stage in front of you.

Here is where most planning goes wrong before it even starts: the practitioner sequences by the patient’s age. They have a mental script for “the thirty-year-old” and another for “the sixty-year-old,” and they run the script the birthday tells them to.

This is a mistake, because age is a liar. Two faces of exactly the same age can sit at completely different points in the process, because genetics, sun, weight history, skin type and hormones all move the timeline around. Sequence by the *stage the face has actually reached*, not the number on the form.

There are four stages, and they have nothing to do with birthdays.

Prevention. Little or no established structural change. The job is to protect and maintain the envelope — skin quality, collagen, and above all sun protection to stop the whole cascade accelerating. Structural filler is frequently unnecessary here, and the correct treatment is often not an injection at all. Restraint is the treatment. This is the hardest thing to sell and the most

important thing to do.

Early structural change. The first genuine structural change appears — a specific, localised deficit the examination can point to. Treatment becomes targeted and minimal: a small, precise correction where it is genuinely indicated, and nothing more. The temptation to over-treat is at its worst here, because the patient is delighted and wants more, and it must be resisted.

Established change. The cascade is clearly working across several levels at once — bone, deep support, superficial descent, envelope. Now you deploy the full sequence in order: structure first, then contour, then envelope, planned deliberately rather than chased piecemeal.

Advanced change. The changes are extensive, and you are at or past the ceiling of what a needle can elegantly fix. Honesty is now a clinical duty. Some faces are better served by a surgeon, and trying to buy a surgical result with a syringe is one of the surest roads to the over-filled face. Knowing when to stop, and when to refer, is part of the skill — arguably the last part you learn.

So the first thing you do with any patient is not note their age. It is work out their stage — how far down the road this particular face has travelled — and enter the sequence there. Which is exactly why I cannot tell you “the right first move for a young patient,” because a young patient might be anywhere from pure prevention to genuine early structural change, and the treatment follows the stage, not the year they were born.

How sequencing fails: the anatomy of the pillow face

You already know what the failure looks like. You have seen them in Mayfair and on Instagram — the smooth, full, faintly strange faces that are unmistakably *treated*. What is worth understanding is that the pillow face is almost never one mistake. It is three, and they compound, each one making the others worse.

Failure one: treating the symptom instead of the cause. Someone points at a fold, and the injector fills the fold — pours superficial volume into the surface expression of a deep problem while the foundation underneath stays unsupported. The material sits on nothing. It inflates the surface without correcting the descent. It looks better for a month and worse for a decade.

Failure two: like-for-like replacement. The injector tries to put back exactly what age removed, millilitre for millilitre, often with a photo of the

patient at twenty-five on the screen. This is the deep error the whole book is written against: it mistakes anatomical ageing for a treatment target. A face at sixty is not a face at twenty with volume subtracted. The bone, the fat, the ligaments and the skin have all changed. Refill every deficit and you do not reassemble the young face — you build a heavier, rounder, subtly unfamiliar one.

Failure three: too early, too long. The patient who starts serious volumising in their twenties, before the cascade has even begun, and is then treated again and again across the decades, accumulates far more material than any face needs, laid into planes that were never empty. The face is slowly remodelled away from itself.

And here is the cruel part: these three almost always travel together. The person whose deep problem was treated superficially is usually also being treated towards a former self, having started too young and continued too long. Each error adds volume the face did not need; together they produce the result everyone recognises and nobody asks for. The pillow face is not bad luck with a needle. It is a failure of sequence — the right materials, in the wrong order, towards the wrong goal, at the wrong time. This is the full argument of the third companion paper in the journal; the version here is the one I teach.

The Three-Visit Plan You cannot rebuild a face in one hour. Attempting to do so usually results in over-treatment, excessive swelling, and an overwhelmed patient.

A comprehensive rejuvenation should be broken down into a structured, multi-visit plan. But before you can execute this plan, you must possess the physical skills to do so. This is where the concept of the cannula transfer-of-skill becomes vital in your planning.

When you build a long-term plan for a patient, you are often committing to treating multiple regions: the midface, the jawline, the temples. If you are intimidated by these areas because you haven't "done" them before, your treatment plan will unconsciously reflect your fear. You will avoid the cheek and over-treat the nasolabial fold, simply because you feel safer there.

You must remember: if you can navigate a cannula safely through the nasolabial fat, you already possess the tactile skill required to navigate the cheek, the jawline, and the temple. The plane is the same.

The glide is the same. Once you know the cannula, it doesn't matter where you put it in. Your hands already know what to do. You just need to watch the

videos, study the regional anatomy, and trust your hands.

Do not let a lack of regional experience dictate your treatment plan. Plan for the architecture, and trust the transfer-of-skill.

Visit 1: The Foundation (Structure and Lift)

This session is entirely about the deep, lateral, and structural areas of the face. Targets: Temples, lateral cheeks (zygomatic arch), deep pyriform, chin, and the angle of the jaw.

Product: High-G-prime HA fillers or structural biostimulators (Radiesse). The Goal: To re-tension the retaining ligaments, lift the SMAS, and restore the skeletal frame. The patient should leave looking lifted and rested, but no one should be able to point to exactly what was done.

Visit 2: The Refinement (Volume and Shadows)

Scheduled 4 to 6 weeks after Visit 1.

The structural filler has integrated, the swelling has resolved, and the true baseline is now visible. Targets: Tear troughs, medial cheeks, lips, and direct softening of the nasolabial or marionette lines (if still necessary). Product: Medium-to-low-G-prime HA fillers, dynamic fillers. The Goal: To soften the shadows, restore the soft volume of youth, and refine the central triangle of the face.

Visit 3: The Canvas (Skin Quality)

Scheduled 4 weeks after Visit 2. The architecture is perfect. Now you perfect the surface. Targets: The dermis and epidermis. Product: Skin boosters, polynucleotides, exosomes, microneedling, or chemical peels. The Goal: To restore luminosity, erase fine lines, and tighten the skin envelope over the newly restored foundation.

The Reality of the Budget The Three-Visit Plan is the clinical ideal. But we do not treat ideals; we treat people. And people have budgets. One of the most common mistakes a practitioner makes is assuming a patient's budget, or worse, altering the clinical truth to fit a perceived budget.

If a patient needs 4 ml of structural filler to lift their midface, but they only want to pay for 1 ml, the beginner will put that 1 ml directly into the nasolabial fold so the patient “sees a result.” The master will explain that 1 ml in the fold

will make them look heavy, and that 1 ml on the cheekbone is a start, but incomplete.

You must have the budget conversation openly, elegantly, and early. “We have talked about the ideal plan. Now we need to talk about reality. Do you have a budget in mind for this journey? Because if you have £2,000, we can do the full structural lift today.

If you have £500, I am not going to give you a quarter of a lift. I am going to suggest we focus entirely on your skin quality with a peel and some medical-grade retinol, because that will give you a beautiful, complete result for that budget, rather than a compromised structural result.” If the budget is limited, focus on the skin. A radiant, glowing face with some structural sagging is still beautiful.

A structurally lifted face with terrible skin still looks old. And a partially lifted face just looks odd. Never compromise the sequence. If they cannot afford the foundation, paint the walls. But never build half a foundation.



PART IV

Regional Treatments

CHAPTER 23

Reading the Map — Regions and Units

Stop looking at the face as a collection of isolated problems. The lips do not exist in a vacuum. The cheeks do not float in space. The face is a single, interconnected ecosystem. Pull a thread in the midface, and the lower face moves. If you don't understand the grid, you have no business touching the canvas.

The classical proportions: equal horizontal thirds and roughly equal vertical fifths. A reference, not a rule.

If you read the textbooks, they will tell you to lean on classical proportional guidelines. The rule of thirds divides the face horizontally into three roughly equal segments: the upper third (from the hairline to the glabella), the middle third (from the glabella to the subnasale), and the lower third (from the subnasale to the menton).

Vertically, the rule of fifths segments the face into five equal columns, each ideally the width of one eye. These rules are fine. They are the training wheels of aesthetic medicine. They help you evaluate facial width, intercanthal distance, and the relationship of the eyes to the lateral facial borders.

But once you understand the rules, you have to throw the ruler away. Nobody walks into a room and measures a face. We perceive beauty instantly, intuitively, and holistically. The rules of thirds and fifths are not rigid commandments. They are simply diagnostic tools — a structured lens through which to identify areas of imbalance and asymmetry before you pick up the syringe.

If a face violates the rule of fifths but looks striking and beautiful, leave it alone. You are an architect, not an accountant.

The Sequencing Logic Reading a comprehensive treatment plan is akin to deciphering an architectural blueprint. It begins with the structural foundation: addressing bone loss and deep fat pad atrophy. These are the pillars and beams of facial support. Neglecting this foundational layer is like building a house on sand; any superficial enhancements will be transient and ultimately unsatisfying.

Once the deep support is re-established, we move to the mid-layer: targeting superficial fat compartments and the SMAS. This layer provides contour and definition. Finally, we address the surface: the dermis and epidermis, focusing on skin quality, texture, and superficial lines. This brings us to the critical concept of sequencing logic.

Why treat the cheeks before the nasolabial fold? Because restoring volume and lift to the midface often provides natural support that softens the nasolabial fold from above, reducing the need for direct, often overdone, treatment of the fold itself. Why address the jawline's structural integrity before chasing the jowl?

Because ensuring the foundational support is in place allows the jowl to be managed as a secondary effect of laxity rather than an isolated problem. The principle is clear: deep before superficial. Correcting deeper volume deficits and structural support first creates a more stable and aesthetically pleasing canvas upon which to refine superficial concerns.

This approach minimises product usage, maximises longevity, and yields far more natural outcomes.

The Anchor Point Every face presents with a unique set of challenges, but almost every ageing face has a single, dominant structural deficit. I call this the anchor point. When you correct the anchor point, you yield disproportionately significant improvements across the entire face. Identifying this anchor point is the hallmark of an experienced injector. It might be temple hollowing that is dragging down the lateral brow and making the eyes look small. It might be midface volume loss that is contributing to heavy nasolabial folds and early jowling. It might be a weak chin that throws off the entire lower facial balance and makes the nose look artificially large.

By strategically addressing this primary deficit, we often find that secondary concerns diminish or resolve entirely, creating a cascade of positive aesthetic effects. This targeted, impactful approach is the art of the game. It requires a keen eye, a deep understanding of facial anatomy, and the confidence to tell a

patient that the problem they see is not the problem they have.

Tier Focus Tier 1: Structural Foundation Bone and Deep Fat Tier 2: Mid-Layer Support Superficial Fat and SMAS Tier 3: Surface Refinement Dermis and Epidermis Examples Temple hollowing, midface volume loss, jawline definition, chin augmentation Cheek contouring, pre-jowl sulcus, marionette lines (indirectly) Fine lines, skin texture, superficial wrinkles, perioral lines In the following chapters, we will break down the face region by region. But never forget the grid. When you are injecting the tear trough, you are affecting the cheek. When you are injecting the chin, you are affecting the jawline. The face is one room. Light it properly.

The Aesthetic Units of the Face

Beyond the rules of thirds and fifths, the face is divided into aesthetic units — regions that share similar skin characteristics, movement patterns, and visual perception. Understanding these units is critical for planning treatment that looks coherent rather than piecemeal.

The upper face unit comprises the forehead, glabella, and temporal regions. It is the unit of expression and emotion. The muscles here are primarily horizontal and vertical, creating the lines of worry, surprise, and anger. Botulinum toxin is the primary tool in this unit. Volume is secondary.

The periorbital unit is the most technically demanding region of the face. The skin here is the thinnest on the body. The vasculature is complex and unforgiving. The tear trough, the lower eyelid, and the lateral canthal region all interact. A mistake here is immediately visible and immediately distressing to the patient. Approach this unit with respect.

The midface unit — the cheeks, the nasolabial region, and the infraorbital area — is the engine of facial rejuvenation. This is where the most dramatic structural changes occur with age, and where the most dramatic structural corrections can be achieved with filler. The midface is where the architecture lives.

The lower face unit comprises the lips, the perioral region, the chin, and the jawline. This is the unit of balance and definition. It is also the unit most vulnerable to over-treatment. A heavy lower face makes the entire face look masculine, aged, and unnatural. Restraint is the operative word here.

The neck and décolleté, while technically separate, must always be considered as part of the aesthetic picture. A beautifully rejuvenated face sitting on an aged, crepey neck is a clinical failure. The face and neck are one canvas.

Asymmetry: The Elephant in the Room

Every face is asymmetric. Not slightly. Significantly. The left side and the right side of a human face are not mirror images. They are two different structures that happen to share a skull.

Before you inject anything, you must document the existing asymmetry. Photograph the patient in standardised lighting, from the front and both 45-degree angles. Show them the photographs. Point out the asymmetry that was already there before you touched them.

This is not just good clinical practice. It is legal protection. The number of patients who will attribute a pre-existing asymmetry to your treatment is higher than you want to believe. Document everything before you start.

Moreover, the asymmetry itself is diagnostic. The side of the face that is more descended, more hollow, or more aged is usually the side the patient sleeps on. It is the side with more sun exposure. It is the side that has been compressed and loaded for decades. Treat the more affected side first. Assess the result. Then decide how much correction the other side needs.

Never treat both sides identically. You will create a different asymmetry. You will have fixed the original problem and created a new one.



CHAPTER 24

Medical Rhinoplasty — Balance Without Surgery

In 2010 I stood on a stage at Olympia and injected a nose in front of a room containing consultant ENT surgeons and several professors of facial surgery.

It was one of the first demonstrations of the procedure at a major conference in this country. I had not been taught it by anybody, because there was nobody to teach it. I had arrived at it the way most of us arrived at everything in those years — I had injected a very large number of nasolabial folds, I understood what that tissue did when you put something into it, and one day a patient with a small dorsal hump asked whether anything could be done, and I thought: probably.

You put a very small amount in. Nothing terrible happens. The next patient, you put slightly more. Before long you are doing noses.

The room split cleanly in half. One half was fascinated and came to find me afterwards. The other half looked as though they were composing a letter to somebody. I understood their position entirely. A junior-ish doctor with no surgical fellowship in facial plastics was standing in front of them explaining that you could change the shape of a nose with a syringe, in the region of the face with the least forgiving blood supply, and several of them had spent a decade learning to do it properly with a scalpel.

They were not wrong to be uneasy. The nose punishes confidence faster than any other area in this book, and a meaningful proportion of the worst complications in aesthetic medicine happen here. Everything in this chapter is written by somebody who was there at the beginning, which means I watched what happened to the people who were not careful.

The nose is the most central, most defining feature of the face. It is also the most unforgiving. For years, the only option for patients unhappy with their nose was surgery. A rhinoplasty with weeks of recovery, significant risk, and permanent, irreversible consequences. The non-surgical nose job changed all of that.

But do not mistake “non-surgical” for “simple.” Medical rhinoplasty is a subcutaneous camouflage procedure. We are not touching the cartilage. We are not touching the bone. We are working in the layer just beneath the skin, correcting the light and shadow that makes the nose look the way it does.

That is our entire job. And it is harder than it sounds. Inspection: The Camera Does Not Lie Inspection begins before the patient sits in the chair. It begins with photographs.

You take four photographs: one straight from the front, one at 45 degrees, and one at 90 degrees from each side. Then you zoom in on every single one of them. This step is not optional.

The human eye, standing in front of a patient, is easily deceived. The zoomed photograph is not. When you zoom in, the shadows become visible in a way that the naked eye simply cannot process in real time. Shadows are the language of the nose.

They tell you exactly where the subcutaneous volume is lacking and where the surface is projecting too much. You cannot plan a treatment without seeing the shadows clearly. During your inspection, you must answer several specific questions. What is the length of the nose? What is the height of the nose? What is the single biggest aesthetic impediment?

Is it a dorsal bump? A drooping tip? A lack of projection? Asymmetry? Each of these requires a different approach. Confusing them produces a result that makes the nose look worse. You must also look at the nose in the context of the whole face.

The nose and the chin are in constant conversation. A weak chin makes the nose look bigger. A strong chin makes the nose look smaller. Sometimes, the most effective nose job is a chin augmentation. The Slanted Nose Illusion One of the most instructive observations in medical rhinoplasty involves the dorsal bump.

A prominent bump is very often accompanied by a concavity on one side of the nose, creating a slanted, crooked appearance. The patient comes in saying their nose is crooked. They are right.

But the crookedness is not coming from where they think. The instinct of an inexperienced injector is to try to fill above and below the bump on the midline, or to attempt to push the bump down. Both approaches miss the point.

The correct approach is to fill the depression on the side where the slant appears. You are adding subcutaneous volume to the hollow that is creating the shadow that is creating the illusion of crookedness. The bump does not disappear, but the asymmetry does. The nose looks straight.

The patient is astonished. This is what understanding shadows can do. The Age of the Nose Before treating, you must assess the age of the patient and the age of the nose. Is this a firm, young nose with good skin quality that simply wants a congenital deformity camouflaged?

Or is this an ageing nose? Thin skin, loss of subcutaneous support, a drooping tip? An ageing nose requires an anti-ageing treatment plan, not just camouflage. Thin skin demands extreme restraint.

Soft products, small volumes, careful placement. With thin skin, the filler can show through the tissue if you are not careful. You will see the product as a slight discolouration or a visible ridge. That is not a complication you can easily explain away. A young nose with thick, firm skin is far more forgiving.

You can use a slightly firmer product, place it with more confidence, and the skin will conceal the correction beautifully. The Single Entry Point and What You Will Feel If the patient presents with a dorsal bump, the cleanest and most efficient approach is to make your entry point directly on top of the dorsal bump itself.

This is not a new concept, but it is the most elegant execution of the procedure.

From this single entry point at the peak of the bump, a cannula gives you access to the entire nose. You are working entirely in the subcutaneous plane — between the skin and whatever is underneath it. You are not going deeper.

You do not need to go deeper. Forward, Toward the Tip From the entry point, slide the cannula forward and downward towards the nasal tip. The resistance here is moderate.

The skin is relatively adherent to the underlying structures in this zone, and as you advance, you will feel the cannula gliding with a slight drag. It is not free movement.

You are pushing through tissue that does not particularly want to be separated. Advance slowly, deposit small amounts, and feel the tissue accepting the product. Backward, Toward the Bridge Without removing the cannula from the skin, pull back to the entry point and flip the cannula 180 degrees, threading it upward towards the radix — the bridge of the nose between the eyes.

The resistance here is noticeably higher. The skin over the nasal bridge is tightly bound. The cannula will feel like it is meeting a wall. This is normal.

You must advance with patience and a light touch. Do not force it. If you force it, you will either buckle the cannula, cause bruising, or deposit product in the wrong plane. Slow, steady, controlled pressure.

Small deposits. Let the tissue accommodate. To the Lateral Walls From the same entry point, you can angle the cannula towards the lateral walls to correct the concavities that create the slanted appearance. The resistance here is different again — slightly less than the bridge, but the skin is thinner laterally and the plane is shallower.

You must be careful not to deposit too superficially. Product placed too close to the skin surface on the lateral wall will be visible and palpable. The key principle for all three directions is the same: you are working subcutaneously, you are depositing tiny volumes, and you are using the cannula as a feeler as much as an injector.

What you feel tells you where you are. If the resistance suddenly drops and the cannula moves freely, you have likely entered a different plane. Pull back slightly and reestablish your depth. Volumes and Restraint The volumes involved in medical rhinoplasty are tiny. Usually 0.1 to 0.3 millilitres for the entire nose, sometimes less.

If you are using more than 0.5 millilitres on a nose, you should ask yourself a serious question about what you are trying to achieve. The goal is always to create the illusion of balance, not to add visible volume. You are not building a new nose.

You are correcting the light and shadow that makes the existing nose look unbalanced. A small injection of botulinum toxin into the depressor septi nasi muscle can also give a subtle but beautiful lift to the nasal tip. Combined with a tiny amount of filler to restore definition, you can take years off the appearance of an ageing nose. Without a single cut.



CHAPTER 25

The Tear Trough — Light Beneath the Eye

I will call him Steven, which is not his name. He has been my patient for the better part of fifteen years, he is very tall, and he arrives with his own weather system.

He came in with an eye that would not stop watering. Not dramatically — a persistent wetness at the inner corner, all day, every day, which had rubbed the skin there into permanent irritation. He had seen people about it. Nobody was certain what was causing it.

He had a long history of filler with me: cheeks, tear troughs, the usual architecture, well tolerated for years. But when something at the medial canthus behaves strangely in a patient with filler in that region, you have to ask whether the filler is doing it. Mass effect, lymphatic obstruction, something sitting where it should not be.

So we dissolved. Not a token amount — the cheek work and the under-eye work, comprehensively, because a partial answer would have told me nothing.

And nothing happened. We waited weeks, then months. The eye wept exactly as before.

Which meant I had removed years of good work from a patient's face in order to rule out a hypothesis that turned out to be wrong, and I still did not know what was causing his problem.

Here is what he did with that. He came in today, cheerful, six foot something of Scottish good humour, and told me the dissolved side had started to look hollow and could I please put it back. He said he had caught sight of himself in a shop window and not known who was looking back. He said it lightly, as a joke against

himself, and moved straight on. That was the entire complaint. No accusation, no anxiety, no suggestion that any of it had been a disaster.

Two chairs down that same week, a patient whose brow had settled a millimetre or two after forehead toxin — an ordinary, entirely expected reduction in resting tone — sat in front of me convinced her life had been ruined.

Same physics. Same practitioner. Opposite events.

The tear trough is the most technically demanding region in this book, and I am going to spend the chapter on the anatomy. But hold onto Steven while you read it, because the hardest variable in this region is not the anatomy. It is who is sitting in the chair.

The tear trough is the area that separates the men from the boys in aesthetic medicine. It is unforgiving. The skin is paper-thin, the anatomy is complex, and the margin for error is essentially zero. If you get it right, you take five years off a patient's face in ten minutes.

If you get it wrong, you create malar oedema, Tyndall effect, and a patient who looks worse than when they walked in.

The Anatomy of the Trough Every face has a tear trough — even a child's. It is the gentle groove that starts from the inner corner of the eye and runs downward towards the cheek. In youth, it is hidden by the fullness of the cheek fat compartments. But as you age, those fat pads deflate.

The cheek loses projection, the skin thins, and the trough becomes visible. What once looked like a single smooth surface separates into the “eye” and the “cheek.” It is not a flaw. It is normal anatomy becoming more defined. And our role is not to remove it.

It is to camouflage it. We are adding a soft filler to bridge the light between the lower eyelid and the cheek.

The Evolution of Technique Back when tear trough treatments first began, practitioners had two main approaches. The first was injecting tiny blebs of filler right into the dermis — micro-droplets that hydrated and thickened the skin. This worked beautifully in young patients who only had early signs of hollowing.

The second method went to the opposite extreme — injecting directly onto the periosteum, the bone itself. This gave excellent structure for deeper troughs, but it was technically demanding and carried a higher risk of complications. Both techniques had their place, but they represented two ends of a very wide spectrum.

Then came the cannula, and the game changed completely. Suddenly, you could enter safely from the cheek, glide along the natural trough, and deliver filler smoothly in one continuous, controlled motion.

The Modern Cannula Approach The concept here is exactly the same as in the rest of the face: we are working in the subcutaneous layer. It is thinner under the eye, but it is the same plane. We augment the soft tissue above, and any bony irregularities below get camouflaged.

Plaster and paintwork. Most of us start from the mid-cheek region. The insertion point is just over the cheek, usually around the mid-cheek fold. From here, the cannula is directed gently towards the inner corner of the eye, following the natural depression of the tear trough.

Scan the QR code to watch a demonstration of the cannula technique:

Before inserting, measure the length of your cannula. You want to ensure it comfortably reaches the inner part of the trough whilst stopping about half a centimetre before the medial canthus. Here is where many injectors go wrong: they angle downward. The trick is to stay flat and parallel to the skin.

You are gliding just under the dermis, not digging deep. When your cannula moves parallel to the surface, it is almost impossible to go too deep. As you move through the tissue, you will occasionally feel little “clicks” or resistance. Those are the fibrous septa or small retaining ligaments tethering the skin.

Do not force it. Withdraw slightly, redirect, and continue your path gently. Eventually, you will meet one that feels different — a firm, springy band that suddenly lets go with a soft “pop.” That is the Orbicularis Retaining Ligament (ORL) — the true border between the eyelid and the cheek.

When you feel it, that is your cue. Cross it gently, never abruptly. Then stop and think about where you actually are — because this is the point at which almost everyone, including me for years, tells themselves a story that the anatomy does not support.

Once you reach your target area, confirm your position by lifting the cannula slightly. You should see the tip tenting the skin. Then, inject slowly and retrogradely — depositing filler as you withdraw. Usually, 0.05–0.1 ml per pass is more than enough. The goal is not to fill; it is to blend.

Where you actually are (and why it is probably not SOOF)

Here is the received wisdom, and it is everywhere: *I am injecting the tear trough, therefore I am placing product in the sub-orbicularis oculi fat.* Search for a tear trough demonstration and you will watch somebody narrate exactly that

as they pass a cannula. I taught something close to it myself for years. The anatomy does not support it, at least not medially, and the reason is simple once you look at where SOOF actually lives.

SOOF is a lateral structure. The medial SOOF begins at the **medial limbus line** and runs laterally to a vertical through the lateral canthus; the lateral SOOF continues from there to the border of the temporal fat pad. Everything medial to that limbus line — which is exactly the part of the trough the patient points at, the bit running toward the inner canthus — has no SOOF under it. From the anterior lacrimal crest across the medial orbit, the orbicularis lies flat against bone with minimal or no fat in between, effectively a ligamentous attachment.

Hong and colleagues put it about as plainly as anatomy ever gets put: the medial tear trough has tight muscular bands and no SOOF beneath the muscle, which makes deep fat-layer injection there fundamentally impossible. And they follow it with the sentence that should stop the argument. Practitioners may aim for the orbital bone, but the muscle is bound so tightly to it that there is almost no space to occupy — so in practice the product is going **into the muscle or above it**.

Read that again, because it is not a claim about bad technique. It is a claim about available space. You can have excellent hands, a well-chosen cannula and a confident narration, and still be depositing filler in a plane other than the one you are naming, medially, because the plane you are naming has no room in it.

So the honest medial lower-lid stack is: skin, a very thin subcutaneous layer, orbicularis, then the tear trough ligament and a tight suborbicularis interface — not a roomy fat pocket — then periosteum and maxilla. Laterally that changes, and SOOF appears between muscle and bone. Medially there is no comfortable space to arrive in; there is a ligamentous attachment. Medially you should assume the deep option is not available and plan accordingly — just above the periosteum within the muscle or at its boundary. Laterally, where SOOF genuinely exists, deep placement beneath the muscle is a real option, and that is where the deep work belongs. There is a matching terminology trap worth naming while we are here: the superficial **infraorbital fat** of the superficial-fat model sits *above* orbicularis and is a different structure entirely from SOOF, which sits below it. Two similar names, two different planes, endlessly conflated.

A word on what you can and cannot tell at the chairside. Start with the introducer, because it is the source of a lot of false confidence. The depth of

your entry nick does not set the plane the cannula ends up in. Two millimetres or three at the port tells you where the tip *started*; where it finishes, several centimetres later, depends entirely on the layer it has been tracking through since. Tissue thickness varies substantially between patients, and in this region supra-orbicularis, intramuscular and suborbicularis are separated by fractions of a millimetre. An entry depth is not a plane.

I used to say that if you can see the cannula tenting the skin, you must be above the muscle. That is not sound, and I would rather correct it here than have you repeat it. In this region the entire stack above SOOF is a couple of millimetres — skin around half a millimetre, a negligible subcutaneous layer, orbicularis around a millimetre — so a cannula sitting *beneath* orbicularis will still tent skin and muscle together and look much the same from outside. Tenting proves the tip is shallow and the overlying tissue is thin. In the lower lid, that is true of every plane you might be in. It discriminates nothing.

Which leaves an uncomfortable but useful conclusion: in the medial trough, feel and appearance cannot tell you which of three planes you occupy — above orbicularis, within it, or beneath it. What the anatomy *can* tell you is that the deepest of those three is largely unavailable medially. That is why the technique still works, and works well, without anybody reconstructing a deep compartment: you are smoothing a visible contour transition at the lid-cheek junction. A successful superficial correction does not prove the deficit was superficial, and a known deep change does not oblige you to replace it deeply. The principle again, in the most delicate region of the face.

If you want certainty rather than inference about your own habitual plane here, high-frequency ultrasound will give it to you in an afternoon. I would not teach fixed millimetre depths in this region — or in the preauricular region, for the same reason — to anyone who had not looked.

The Forgotten Layer — Dermal Hydration Once the volume is replaced with the cannula, the deeper hollow softens. But many patients still complain of tired skin, fine lines, or crepiness under the eyes. That is not volume — that is dermal thinning. Here is where we return to the surface. The skin above the trough is thin, but it still has a dermis.

Tiny micro-boluses of soft filler into this layer can dramatically improve skin quality — hydration, elasticity, and smoothness. Scan the QR code to watch the dermal blebbing technique:

Scan the QR code for a full clinical tear trough case walkthrough:

When performing dermal blebbing, stabilise your injecting hand. Do not let it free-float in the air in case the patient flinches. Enter the skin parallel to the surface, bevel up. This allows the needle to travel a few millimetres towards the site of injection, making it much easier to ensure you are truly in the dermis. The travel distance is minimal, and the volumes are microscopic — literally less than 0.1 ml for the entire tear trough and palpebromalar groove.

The Tyndall Effect The Tyndall effect is one of the most visually recognisable complications in aesthetic medicine. It appears as a subtle blue-grey hue when filler sits too superficially under thin skin. To understand it, you must understand light. When a hyaluronic acid gel sits too close to the skin surface, it behaves like a micro-prism.

The gel refracts, bends, and scatters incoming light. Shorter wavelengths — the blue tones — scatter the most. That is why we see a blue hue: the physics of light interacting with gel density, crosslinking patterns, and water content. The Tyndall effect is not a filler problem — it is a physics problem.

And physics does not negotiate. However, the type of filler you use matters immensely. Biphasic fillers (which contain HA particles suspended in a carrier gel) have a high light-scattering prism effect and a much higher risk of Tyndall if placed superficially. Monophasic fillers (smooth, cohesive singlephase gels) have minimal scatter and uniform refraction.

If you use a soft, monophasic filler (like Teosyal Redensity II, RHA-2, or Juvederm Volbella) and place it in the correct plane, Tyndall rarely appears.

Beginner Case Selection If you are new to treating the tear trough, case selection is everything. The Ideal Patient: • Young to middle-aged (25–45) • Good skin elasticity

- True anatomical hollowing (a visible dip) • No history of malar oedema

or morning eye puffiness The Patient to Avoid: • Significant skin laxity or festoons • Large prolapsed orbital fat pads (these need surgery, not filler) • Chronic morning puffiness or lymphatic drainage issues • Unrealistic expectations Start with the easy cases. Use a cannula. Use a soft, monophasic filler. Stay superficial to the bone but deep to the dermis. And remember the golden rule of the tear trough: less is more. You can always add 0.1 ml in two weeks. You cannot easily un-see a blue, puffy eye that you overfilled.

CHAPTER 26

The Cheek — The Power Below the Trough

The cheek is the kingmaker. It dictates everything that happens below it. The tear trough, the nasolabial fold, the jowl—they all take their orders from the midface. If you chase the folds without fixing the cheek first, you are just treating the symptom while ignoring the disease.

Central to this architectural integrity is the Ogee Curve, the elegant, S-shaped silhouette that defines a youthful, vibrant midface. Observe it in profile: a gentle convexity from the lateral zygoma, sweeping inward to a concavity beneath the orbital rim, then outward again to the fullness of the malar eminence.

This curve is the hallmark of vitality, reflecting light beautifully and creating a harmonious transition from the lower eyelid to the cheek. Its assessment is critical. Have the patient turn their head slightly, allowing the light to catch the contours. Does the curve flow smoothly, or is it interrupted by shadows and flatness?

With age, this graceful curve invariably disappears. The culprits are usually described as two: atrophy of the deep medial cheek fat pad and changes in the sub-orbicularis oculi fat (SOOF). Note the name — SOOF is *sub*-orbicularis, sitting beneath the muscle. It is not the superficial infraorbital fat that lies above orbicularis, and the two are confused constantly, including in print. The deep fat provides the structural support, and its loss leads to a flattening of the malar prominence.

The SOOF, once a youthful cushion, succumbs to gravity, creating a hollow beneath the eye and exacerbating the appearance of the tear trough. Filler, when

strategically placed, acts as a scaffold, restoring the lost volume and lifting the descended tissues, thereby re-establishing that coveted Ogee Curve and the youthful light reflection it brings.

What your cannula is actually travelling through Let me describe my own habitual cheek technique honestly, because naming the plane you are truly in is more useful than naming the plane you would like to be in.

I enter low, at the lower part of the mid-cheek groove, angle towards the medial canthus, and fan — first across the groove, then laterally over the cheekbone. If that cannula stays where I can palpate it easily beneath the skin, here is the stack I am working in:

Skin superficial fat compartments SMAS and the mimetic

muscles deep fat compartments periosteum bone.

Which means the first meaningful compartment I am inside is the **superficial medial cheek fat** — not the deep medial cheek fat. As I fan laterally I pass from superficial medial cheek fat into **middle cheek fat**, and further out into the **lateral temporal cheek fat**. If I sweep up towards the lid-cheek junction while staying superficial, I am in the **superficial infraorbital compartment**, not SOOF.

Below all of that — beneath the SMAS and muscle layer — sit the deep medial and deep lateral cheek fat, and, more superiorly, medial and lateral SOOF. I know they are there. I am usually not in them.

Now, why does such a superficial technique produce so large a visual change? Because the mid-cheek groove is not an empty valley between two pads. It is a zone of **zygomatocutaneous ligamentous tethering**, with superficial fat arranged around that fixation. Alter the volume and contour immediately beneath the skin around a tethering structure and you change the external geometry of the face directly. Superficial fat is intimately bound to the skin and the cutaneous retaining system; deep fat is more static and acts as underlying support. Modest change in the superficial plane moves what the patient sees.

One caution to go with it: being “over the cheekbone” does not mean you are on periosteum or in deep fat. If you deliberately descend beneath orbicularis and SMAS over the zygoma, you can enter lateral SOOF and the deep malar structures — but you have to mean it. Lateral is not the same as deep.

For the discerning injector, understanding midface anatomy is paramount. We can delineate the cheek into three critical injection zones, each with its distinct purpose and anatomical considerations: 1. Zygomatic Arch (Structural Zone): This is where we lay the foundation. The zygomatic arch provides the lateral projection and definition of the midface.

Injecting here, directly onto the periosteum, offers maximal lift and support, effectively pulling up the overlying soft tissues. It's the anchor point for the Ogee Curve. 2. Mid-Cheek (Volume Zone): This area encompasses the malar fat pad and the submalar region. It's where we restore the bulk of lost volume, addressing the deep medial cheek fat atrophy and contributing to the convexity of the Ogee Curve.

This zone requires careful consideration of depth to avoid a puffy appearance.

3. Anterior Cheek (Transition Zone): This zone bridges the gap between the mid-cheek and the nasolabial fold. It's a delicate area where volume restoration can subtly soften the nasolabial fold and improve the transition from the lower eyelid to the cheek, without directly filling the fold itself.

Precision in entry points and technique is non-negotiable. For the zygomatic arch, a lateral hairline entry is often preferred. Using a needle, we advance supraperiosteally, directly onto the bone. This deep placement minimizes the risk of vascular compromise and provides robust structural support. The goal here is lift and projection, not superficial puffiness.

For the mid-cheek fat pad, a mid-cheek groove entry with a cannula is generally safer and allows for a broader, more diffuse product placement. The cannula navigates through the subcutaneous plane, reducing the risk of piercing vessels and nerves. This technique is ideal for restoring volume and creating a smooth, natural contour.

Product selection is not a matter of preference; it's a science. The rheological properties of the filler must match the anatomical zone and the desired outcome. For the structural work along the zygomatic arch, you need a product with high G-prime – a robust, firm gel that can withstand external forces and provide significant lift.

Think of it as the rebar in concrete. For the mid-cheek, where volume restoration and a softer contour are desired, a medium G-prime product is more appropriate. This allows for a more natural integration with the surrounding tissues and a smoother aesthetic. Injection Zone Zygomatic Arch

Mid-Cheek Anterior Cheek Characteristics Product Examples (HA Fillers) Juvederm Voluma, High G-prime, firm Restylane Lyft Volift, Medium G-prime, softer Juvederm Restylane Defyne Volbella, Medium G-prime, flexible Juvederm Restylane Kysse Product Examp (Biostimulators Radiesse (dilute Sculptra (diluted - Volumes, like product selection, must be judicious. The aim is restoration, not exaggeration. For structural work along the zygomatic arch, a conservative approach is always best. You're building a foundation, not a mountain. For the mid-cheek, where more diffuse volume loss is being addressed, slightly higher volumes may be necessary, but always with an eye towards natural integration. Injection Zone Zygomatic Arch Volume per Side (Initial Treatment) 0.5 - 1.0 ml Mid-Cheek Anterior Cheek 0.5 - 1.5 ml 0.2 - 0.5 ml (as needed)

One of the most satisfying outcomes of the cheek-first philosophy is its profound effect on the nasolabial fold. Patients often present fixated on these folds, demanding direct treatment. However, the astute injector understands that the nasolabial fold is often a symptom, not the primary pathology.

When you place 1 ml of filler strategically in the midcheek, you are not just adding volume; you are lifting the descended malar fat pad and the surrounding soft tissues that contribute to the fold. This upward and outward vector of lift effectively softens the nasolabial fold from above, creating a more natural and harmonious result than directly injecting 0.5 ml into the fold itself.

Direct injection into the fold can often lead to a heavy, unnatural appearance, whereas midface support addresses the root cause, providing a more elegant and lasting improvement. It's about understanding the mechanics of facial aging and working with them, not against them. Finally, the conversation with the patient.

The dreaded, "I don't want bigger cheeks, Doctor." This is where your communication skills are as crucial as your injection technique. Patients often equate cheek filler with the overfilled, unnatural look they've seen on social media. Your role is to educate, to differentiate between augmentation and restoration.

Explain that the goal is not to create new, prominent cheeks, but to restore the youthful volume and support that has been lost over time. Use analogies: "We're not trying to make your cheeks bigger, we're simply putting things back where they used to be." Show them photos of their younger selves, if available, or use subtle before-and-after examples of your own work that demonstrate

natural restoration.

Emphasise the Ogee Curve and how restoring it will improve their overall facial harmony, softening other areas of concern without creating an artificial appearance. Reassure them that a conservative, staged approach is always an option. This conversation builds trust and manages expectations, ensuring that the patient understands and appreciates the subtle, yet transformative, power of a well-treated midface.

The Ogee Curve — The S-Curve of Youth If you want to understand the cheek, you have to understand the Ogee curve. It is the double-curved, S-shaped silhouette that runs from the lateral orbital rim, down through the fullness of the cheek, and gently tapers towards the nasolabial fold. It is the defining architectural feature of a youthful midface.

How do you assess it? You don't look from the front. You stand to the side. A youthful face has a beautiful convexity at the cheekbone and a gentle concavity below it. As we age, the deep medial cheek fat atrophies, the SOOF descends, and the zygoma resorbs.

That elegant S-curve flattens into a straight, miserable line. How do we fix it? We don't just pump volume blindly. We use a supraperiosteal injection at the zygomatic arch to restore the upper convexity. We use a cannula in the mid-cheek fat pad to restore the transition.

The S-curve reappears. The ultimate clinical test? Photograph the patient from a 45-degree angle. The Ogee curve— or its tragic absence —is undeniable at 45 degrees.



CHAPTER 27

Nasolabial Folds — The Smile's Shadow

Let us set the record straight on the nasolabial fold. It is one of the most misunderstood and overtreated areas in aesthetic medicine. The nasolabial fold is not a wrinkle. It is an anatomical structure.

A groove created by the attachment of the skin to the underlying muscle and the descent of the fat pads above it. You cannot iron it out. You cannot fill it away.

You can only understand what is causing it — and treat that. The Fold and the Crease Are Not the Same Thing This is the distinction that separates a competent injector from a great one. The nasolabial fold is the shadow cast by the cheek descending over the upper lip.

It is a volume problem. The fat pads of the midface — specifically the medial cheek fat, the middle cheek fat, and the nasolabial fat pad — have deflated and descended. The skin above is now hanging over the junction between the mobile upper lip and the relatively fixed cheek.

The fold is the result of that overhang. The nasolabial crease is the fine line that sits within or just below the fold. It is a dermal problem.

Years of smiling have repeatedly folded the skin at the same point. The dermis has thinned. The crease is etched in. Volume loss from the fat pads above makes it more visible, but the crease itself is a separate issue. Treating the fold without understanding this distinction is how you end up with a patient who looks heavy, overfilled, and unnatural — because you stuffed the fold instead of lifting the cheek.

The Fat Pads That Drive the Fold The nasolabial fold is primarily driven by the deflation of three fat compartments: The medial cheek fat pad sits directly above the fold. It is the first to deflate in most patients and the most directly

responsible for the fold deepening.

When this pad empties, the skin above the fold loses its support and the shadow deepens. The middle cheek fat pad sits lateral and slightly superior to the medial pad. Its deflation contributes to the general flatness of the midface and the loss of the ogee curve — the Sshaped contour of a youthful cheek.

The nasolabial fat pad itself sits within the fold. It is smaller than the other two but its deflation contributes to the crease becoming more visible. The sub-orbicularis oculi fat (SOOF) and the deep medial cheek fat sit deeper, closer to the periosteum. Their deflation causes the more structural midface collapse seen in older patients — the kind where the cheekbone appears to have disappeared entirely.

The Injection Plan — Two Phases Treating the nasolabial fold correctly requires two separate assessments and, often, two separate treatments. **Phase One: Restore the Cheek Volume** The entry point for the cheek fat pads is the mid-cheek groove. This is found along an imaginary line drawn from the corner of the lip to the earlobe.

Somewhere along that line, you will feel and see a subtle depression — a dip in the cheek surface. This dip corresponds directly to the nasolabial fold below it. Where the cheek is hollow, the fold is deep. Use a blunt cannula — a 22G or 23G is appropriate here.

The product should be a high Gprime, structural filler: Juvéderm Voluma, Restylane Lyft, Teosyal Ultra Deep, or equivalent. In younger patients where the goal is hydration and soft volume rather than structural lift, even a softer product like a lip filler can work beautifully — the tissue is more forgiving and the result more natural.

Insert the cannula at the mid-cheek groove entry point and advance it towards the dip you have identified. You are working in the deep subcutaneous plane, above the periosteum. Deposit the filler into the hollow.

As you inject, you will see the dip fill and the cheek surface rise. The nasolabial fold above will visibly soften as the support beneath it is restored. If the patient already has good cheek volume — if the mid-cheek is full and the fold is still deep — then the problem is not the medial fat pad.

It is the cheekbone projection. In this case, enter from the hairline, following the cheek silhouette, and advance one cannulalength towards the zygoma. You are now going under the facial retaining ligaments — the zygomatic and masseteric ligaments — and you will feel significant resistance as the cannula

passes beneath them.

This is expected. Do not force it. Work slowly, feel the plane open, and deposit your filler supraperiosteally at the cheekbone. As you inject, you will see the cheekbone rise and the cheek silhouette restore. The further the filler is from the fold itself, the less direct effect it will have on the fold — but the structural improvement to the overall face will be greater.

This is the trade-off. Know it before you start.

Phase Two: Treat the Crease — If It Remains After the cheek has been restored, reassess the nasolabial fold. In many patients, particularly younger ones, the fold will have significantly improved and no further treatment is needed. In patients with a long-standing, deep crease — one that has been etched into the dermis by years of smiling — the crease will persist even after the cheek has been lifted.

This is the crease, not the fold. And it requires a different approach entirely. For the crease, use a soft, low-G-prime filler with a fine needle. Teosyal RHA2, Belotero Balance, or Juvéderm Volift are all appropriate choices. These products come with a 30G needle and are designed to integrate into the dermis without creating lumps or the Tyndall effect.

The technique is intradermal or deep dermal injection, parallel to the skin, retrograde threading along the crease. Small aliquots. The goal is to hydrate and subtly lift the dermis at the crease line — not to fill the fold. If you are depositing more than 0.1 ml in the crease itself, you are probably overinjecting.

The Decision Framework Before you pick up a syringe, ask yourself two questions: Is this a fold or a crease? If the fold is deep and the cheek is flat, treat the cheek first. If the cheek is already full and only the crease remains, treat the crease directly.

What is the patient's age and skin quality? In a younger patient with good skin elasticity, restoring the cheek fat pads will often resolve the fold entirely. In an older patient with thin, inelastic skin, the crease may require direct treatment regardless of how well the cheek is restored.

The Cannula in the Crease — The Simplest Approach If you are using a cannula rather than a needle for the crease itself, the technique is straightforward: thread the cannula along the crease in the deep subcutaneous plane, depositing small retrograde aliquots. This is the most accessible approach for practitioners who are still developing their intradermal needle technique, and it produces a reliable, natural result. The more technical approach — and

the more precise one — is the 30G needle in the mid-dermis, threading retrograde along the crease line. Both work. The needle gives you more control over depth and deposit size. The cannula is safer and more forgiving. The practical advantage of the cannula approach is this: whatever is left in the syringe after treating the crease goes directly into the cheek.

You are using one entry point, one cannula, and one syringe to address both the crease and the fold that is causing it. This is efficient, elegant, and logical.

[VIDEO: Nasolabial crease treatment via cannula — demonstrating the crease-first approach with the remainder of the syringe used to restore cheek volume.] Watch: <https://youtu.be/t4tkVwgA7aA> Volume Allocation — Where the Filler Actually Goes This is something patients need to understand before treatment, and something injectors need to internalise before they pick up a syringe.

The nasolabial crease can only absorb 0.1 to 0.2 ml of filler. That is it. Any more than that and you are not treating the crease — you are stuffing the fold, and the result will look heavy, immobile, and unnatural. The crease is a fine, shallow structure.

It does not need volume. It needs hydration and a small amount of dermal support. The rest of the filler — the majority of what you are injecting in this treatment — goes to the cheek. This is where the volume belongs.

This is where the cause of the fold lives. Addressing Patient Apprehension About Cheek Volume Almost every patient who presents with nasolabial folds will say some version of the same thing: “I don’t want bigger cheeks. I don’t want to look puffy.” This is a reasonable concern.

They have seen the results of overfilled cheeks. They have seen the pillow face, the apple cheeks, the unnatural roundness that signals filler from across the room. The answer is not to avoid the cheeks. The answer is to explain what you are actually doing.

You are not adding volume. You are replacing volume that was already there. Every millilitre you inject into the cheek fat pads is replacing a millilitre that the patient had ten years ago — and lost. The goal is not augmentation.

It is restoration. The face should not look different. It should look like itself, at an earlier point in time. When patients understand this distinction — that you are restoring, not adding — the apprehension usually dissolves. They are not getting bigger cheeks.

They are getting their cheeks back. The Mistake Most Injectors Make Most injectors go straight for the fold. They fill it. The patient smiles. Everyone is happy — for a month. Then something starts to feel off.

The smile does not move quite right. The mid-face looks heavy. There is volume, but no vitality. That is because they did not lift. They stuffed. They treated the symptom instead of the cause. The nasolabial fold is the punctuation mark of the human face.

It is not a problem to be erased. It is a sign to be read. Read it correctly, treat the cause, and the result will be a face that looks younger, lighter, and more alive — not a face that looks filled.

The Jawline and Jowls — Rejuvenation from the Bottom Up

Most injectors love the midface — the cheeks, the tear trough — and they stop there. But if you only lift from the middle, you create imbalance. You get young, plump cheeks sitting on an old jawline with sagging corners, tired marionette folds, and aged lips.

That is how the “pillow face” happens. When we rejuvenate from the jawline upward, the effect is undetectable. The result looks like good genetics, not good filler. The jawline is the foundation of youth — the base upon which the rest of the face rests.

Once that base is restored, everything above naturally follows.

The Logic of the Lift The first thing to understand is that the face does not age vertically — it ages diagonally. Loss of support in the lateral temporal-cheek fat pad allows the overlying tissue to slide forward and downward. This drags the superficial jowl fat pad over the mandibular retaining ligament, forming the visible jowl bulge.

Jowls do not form from the bottom up. They form from the top down. That is why we do not inject the jowl itself. We correct it from a distance — lifting it from above (outer cheek) and supporting it from below (mandible and pre-chin sulcus).

Hormones, Bone, and Muscle In women, oestrogen decline from the early thirties reduces osteoblastic activity. Bone resorption starts around the mandibular angle and anterior chin, leading to flattening and loss of structural lift. At the same time, muscular habits play a role. Those who chew gum excessively or grind their teeth show temporary masseter hypertrophy, which

keeps the jawline looking defined.

However, once that muscle relaxes (for example, after a Botox treatment), the underlying bone loss becomes suddenly apparent. In bruxers, youth is borrowed from the muscle — but the debt is paid by the bone.

Why We Avoid the Superficial Jowl Fat Pad We do not inject the superficial jowl fat pad. Not because of vascular or nerve risk, but because of aesthetic design. It is a descended, heavy compartment; adding filler makes it heavier. I once watched a highly respected plastic surgeon perform a facelift. He made the incision, lifted the SMAS, pulled the tissue taut, and sutured it into place. The result was beautiful.

Then, almost as an afterthought, he took the tiny piece of excised fat from the procedure and tucked it back into the pre-jowl sulcus to smooth the transition. Within six months, that patient had a jowl again. Not because the facelift failed, but because he had added weight to the exact compartment that gravity targets first.

We don't fill the jowl to hide it. We lift the face so the jowl has nowhere left to fall.

The Skeletal Context Before you treat a jawline, you must assess the patient's skeletal class. The jawline does not exist in isolation — it is the lateral extension of the chin. • **Class I (Ideal):** These patients have good natural projection. You are simply restoring the bone they have lost to age.

Focus on the mandibular angle and the pre-jowl sulcus. • **Class II (Retrognathic):** These patients have a weak, recessed lower jaw. Their jowls appear earlier and more severely because the soft tissue has no bony ledge to drape over. You cannot fix their jawline until you have projected their chin.

- **Class III (Prognathic):** These patients have a strong, protruding lower jaw. They rarely complain of jowls, but they may complain of a harsh, masculine lower face. Your goal here is softening the transitions, not adding projection.

Two Methods. One Decision.

Before you pick up a cannula, you need to decide what you are actually doing. There are two fundamentally different goals in jawline treatment, and they require different approaches. **Method 1: Lift and Restore** is what the vast majority of patients need. They have lost volume, lost bone, lost structural support.

The face has descended. You are putting it back. Method 2: Widen and Masculinise is what a small minority request. Usually younger patients, usually male, who want a broader, more angular lower face. This is not a lift.

This is a shape change. Here is the important clinical point: almost everyone who comes to you with a jawline concern wants Method 1 first. Even men who eventually want masculinisation usually have some volume loss to address before you can talk about widening. By the time most patients sit in your chair, the lower face has already descended.

Masculinisation becomes a secondary conversation — if it comes up at all. Scan the QR code to watch a demonstration of jawline contouring:

Method 1: Lift and Restore — The Tragus Vector The goal is to restore the sharp ‘L’ shape of the mandible. When the ‘L’ becomes an ‘I’, the face looks tired. When you restore the ‘L’, the eyes lift again — even if you never touched them. The Entry Point You make the entry point approximately two-thirds of the way down the mandible, onethird away from the angle of the jaw, close to the anterior border of the masseter muscle on the mandible.

The needle faces towards the ear — towards the tragus. That is your vector.

As you advance the cannula towards the tragus, you will feel some resistance as you approach the ligament just anterior to the ear. Then it pops — you are now in the outer temporal fat pad compartment. This is where you deposit 0.2 ml of ultra-deep, high-Gprime filler.

Withdraw slightly, fan the cannula inferiorly by a couple of centimetres, and deposit another 0.2 ml. Fan again, slightly more inferior, and deposit a third 0.2 ml along the mandible itself. Three deposits.

Before you run that vector, know what is underneath it. From the surface down, the preauricular and lateral cheek stack is: **skin subcutaneous fat, including the lateral temporal cheek compartment SMAS parotid fascia parotid gland**

parotidomasseteric fascia masseter mandible.

If you introduce at the mid-jawline, travel posterolaterally towards the ear, and keep the cannula easily palpable immediately beneath the skin, you are most likely running through **superficial lateral cheek fat into the lateral temporal cheek and preauricular superficial fat** — superficial to the parotid, not under the SMAS. That is a legitimate and well-described plane;

there is published work treating this lateral superficial compartment specifically in the subcutaneous plane, extending towards the mandibular angle and the preauricular region.

Here is the part that should slow you down. **The preauricular subcutaneous layer can be extraordinarily thin.** Ultrasound and anatomical work put typical thickness in this region at around two to four millimetres, and in some people at **less than one millimetre** before you are into SMAS and the parotid region. Filler has been identified, unintentionally, inside the parotid gland. So the philosophy is sound — stay superficial in the lateral face — but the margin between “superficial lateral temporal cheek fat” and “too deep, into the parotid” is smaller here than almost anywhere else you work.

This is one of the two regions in this book — the medial tear trough is the other — where I would genuinely want ultrasound on my own technique before teaching anybody exact depths.

0.2 ml each. That is your standard lift for a patient in their twenties with mild volume loss. If There Is Bone Resorption If you can see a resorption sign along the angle of the mandible — a visible flattening or concavity where the bone has retreated — you can treat up to three centimetres of the mandibular border directly.

Place 0.1 to 0.2 ml of ultra-deep filler along that stretch. You are replacing the scaffold that has disappeared. Scaling the Volume Double these volumes for older patients or those with significant volume loss. The entry point and the vector stay the same. The quantities scale with the degree of descent.

The Product You need a high-G-prime, bone-like filler. Teosyal Ultra Deep, Stylage XXL, or Juvederm Volux. These fillers do not just fill — they sculpt. The Anterior Continuity Using the same entry point, redirect the cannula anteriorly towards the pre-chin sulcus. Add small threads or micro-boluses to smooth the transition between the jawline and the chin.

This lifts the marionette zone and makes the contour continuous. You are creating support from three pillars: the outer cheek for upward tension, the jawline for lateral support, and the pre-chin sulcus for forward projection.

Method 2: Widen and Masculinise This is a completely different injection. If Method 1 is about lifting the tissue behind the masseter, Method 2 is about depositing filler on the masseter muscle itself. Filler placed on the masseter surface makes the face look wider. It does not lift — it broadens.

This is the masculinisation effect. But understand this clearly: placing filler behind the masseter (posteriorly) lifts the jawline upward. Placing it on the masseter (superficially) widens the lower face. These are not interchangeable.

Doing Method 2 on a patient who needed Method 1 does not look masculine. It just looks like a wide face.

Masculinisation is also never just the jawline. To achieve a genuinely masculine lower face, you need to look at the jaw and chin together, the cheekbones, and — if you are being thorough — the temple. The jawline filler is one component of a wider architectural conversation. For most patients, that conversation starts with Method 1.

The Art of Feeling When you are in the subcutaneous space with a wide-bore cannula, you do not need to see. You just need to feel. Your cannula should glide like a paintbrush, not a plough. • Smooth glide: Correct plane, safe zone. • Soft give: Fat layer — ideal area.

- Click / pop: Ligament — change vector slightly. • Rough or gritty: Too deep — periosteum. • Resistance with blanching: Too superficial — dermis.

A Note on “Migration”

During your treatment, the filler will spread and integrate, as it should. But social media loves the word “migration.” Let’s be honest — filler doesn’t migrate. It was put in the wrong plane. What people call migration is just misplaced filler that followed gravity and fascia.

Filler doesn’t migrate — injectors do. Do not blame the product. Blame the layer, the vector, and the lack of respect for anatomy. When you inject with structure and logic, the face stays sculpted, natural, and harmonious. We don’t chase what fell — we restore what held it up.



CHAPTER 29

The Chin — The Anchor of the Face

The chin is the anchor. It is the period at the end of the sentence. You can build the most beautiful jawline in the world, but if the chin is weak, the whole lower face collapses into the neck. It is the most structurally important, least requested treatment in the clinic.

Patients never walk in and ask for a chin. They walk in and complain about their jowls, their double chin, or their neck. Your job is to look at their profile and realise that the jawline isn't the problem — the anchor has disappeared.

The Five Layers of the Chin To inject the chin safely and effectively, you must understand the five layers. The chin is a highly mobile, muscular structure sitting over bone, and injecting the wrong layer leads to migration, unnatural movement, or vascular disaster. 1.

Skin: Thick, sebaceous, and tightly bound to the underlying muscle. 2. Subcutaneous Fat: The superficial fat layer. This is thin in the chin, but it is where the mentalis muscle inserts into the dermis to create the “button” effect. 3.

Muscle (The Mentalis): A paired, cone-shaped muscle that originates from the mandible (incisive fossa) and inserts directly into the chin skin. It elevates the lower lip and wrinkles the chin. 4.

Deep Fat: A small, deep fat compartment lying beneath the mentalis muscle, sitting directly on the periosteum. 5. Bone (The Mandibular Symphysis): The structural foundation.

The Ageing Sequence The chin does not age in isolation. It ages through a combination of bone resorption, muscle hyperactivity, and fat atrophy. 1. Bone Loss: The mandible rotates and the symphysis resorbs. The chin loses forward projection and begins to rotate upwards. 2.

Mentalis Hyperactivity: As the bone shrinks, the mentalis muscle has to work harder to close the mouth (achieve oral competence). This chronic hyperactivity leads to a permanent “pebbled” or “golf ball” appearance on the surface. 3.

The Witch’s Chin: The deep fat pad atrophies, and the retaining ligaments (the mandibular septum) weaken. The soft tissue envelope slides off the receding bone, drooping downward to create the classic “witch’s chin” (ptosis of the chin pad).

The Skeletal Class Roadmap You cannot treat every chin the same way. The starting point dictates the strategy. Before you pick up a syringe, you must classify the patient’s underlying skeletal structure. **Class I (The Ideal Foundation)** The upper and lower jaws are aligned. The most anterior point of the chin sits approximately 2–3mm posterior to the lower lip vermillion.

The Strategy: These patients only need age-related restoration. Small, precise periosteal boluses to replace lost bone, or superficial cannula work to smooth the pre-jowl sulcus. Do not over-project them.

Class II (The Retrognathic Chin)

The lower jaw is set back relative to the upper jaw. The chin is visibly weak, the neck angle is poor, and the mentalis muscle is usually hyperactive because it is constantly straining to pull the lower lip up to meet the upper lip. **The Strategy:** This is the most common presentation in aesthetic clinics.

These patients need significant structural support. 1. Relax first: You must treat the mentalis with botulinum toxin two weeks before filler. If you inject filler against a hyperactive mentalis, the muscle will crush the product or push it out of place. 2.

Project second: Use a high-G-prime filler (like Volux or Radiesse) placed deep on the periosteum to drive the chin forward. 3. **Blend third:** Soften the transition into the pre-jowl sulcus to prevent the chin from looking like an isolated, disconnected ball. **Class III (The Prognathic Chin)** The lower jaw protrudes past the upper jaw.

The chin is strong, often dominant. **The Strategy:** Do not project. Never add anterior volume to a Class III chin. If they present with ageing concerns, focus entirely on the lateral aspects — widening the jawline to balance the prominent chin, or smoothing the marionette shadows without adding forward projection.

Projection vs. Width (The Gender Axis)

When considering chin augmentation, it is crucial to differentiate between projection (forward) and width (lateral). This is the primary gender axis of the lower face. • **Feminisation:** A female chin should be narrow, tapering to a soft point that aligns with the medial canthus of the eyes.

You want projection, not width. • **Masculinisation:** A male chin should be wide and square, aligning with the oral commissures (the corners of the mouth). You want projection and lateral width.

A common pitfall is addressing perceived width issues when the underlying problem is a lack of anterior projection. Broadening a female chin will instantly masculinise the face. If a woman needs projection, keep the product central.

The Technique For chin augmentation, my preferred technique involves a supraperiosteal needle injection at the central chin. This is one of the few areas where I still advocate for direct on-bone injection. The midline of the chin (between the mental foramina, which sit laterally in line with the second premolars) is a vascular desert. It is incredibly safe. 1. The Entry: Using a 27G needle, advance perpendicular to the skin, straight down to the bone. 2. The Tap: A gentle tap confirms you are on the periosteum. 3.

The Bolus: Deposit a precise, slow bolus of high-G-prime product. The supraperiosteal plane minimizes the risk of vascular compromise and ensures the product remains fixed, providing a robust and long-lasting structural enhancement. Multiple small boluses can be layered if significant projection is required, always ensuring even distribution.

Product and Volumes Product selection is critical. For significant projection, a high G-prime product is indispensable. My go-to choices include Volux or Radiesse, both of which offer the necessary structural integrity and lift capacity to effectively augment the bony chin. These products resist deformation and maintain their shape, providing a crisp, defined mentum.

Regarding volumes, for typical projection cases, I generally use between 0.5 to 1.5 ml of product. The exact volume is dictated by the degree of recession. It is always better to under-correct and add more later than to over-correct and face the challenges of reversal.

Start conservatively, assess the profile, and build up if necessary. Finally, always consider the chin-jawline relationship. These two structures are inextricably linked. A beautifully projected chin, if not accompanied by a reasonably defined jawline, can appear disconnected or create the illusion of a more prominent submental area.

Conversely, a strong jawline without adequate chin projection makes the face appear heavy. Treat the anchor, but blend it into the ship.



CHAPTER 30

The Temple — A Quiet Sculptor's Field

Patients never walk into a clinic and ask for temple filler. They don't look in the mirror and think, "My deep temporal fat pad has deflated." Instead, they pull the skin at the side of their eyes upward and say, "My brows are dropping." Or they complain that their face looks skeletal, gaunt, or "peanut-shaped." The temple is the quiet architect of the upper face.

When it is full, it provides a smooth, convex transition from the forehead to the cheek, framing the eye and supporting the lateral brow. When it hollows, the entire upper face collapses inward. Treating the temple is one of the most satisfying procedures in aesthetic medicine.

It is also one of the most technically demanding.

The Anatomy of the Hollow The temple is not a single space. It is a layered sandwich of fascia, muscle, and fat, sitting over the temporal bone. From superficial to deep, the layers are: 1. Skin 2.

Subcutaneous fat 3. Superficial temporal fascia 4. Loose areolar tissue 5. Deep temporal fascia 6. Temporalis muscle 7. Deep temporal fat pad 8. Periosteum/Bone When the temple hollows, it is usually a combination of bone resorption, muscle atrophy, and the deflation of the deep temporal fat pad.

But remember the rule of the architect: we do not have to replace the volume exactly where it was lost. We have to choose the plane that safely and predictably restores the surface contour.

The Danger Zone The temple is heavily vascularised. The superficial temporal artery runs through the superficial fascia. The deep temporal arteries run deep, near the bone. If you are sloppy in the temple, you will cause a vascular occlusion. If you inject too much volume too quickly in the wrong

plane, you can compress the venous drainage and cause severe, long-lasting swelling around the eye.

This is not a region for guesswork. You must know exactly which plane you are in.

The Two Techniques There are two primary ways to treat the temple: deep and superficial. Both have their place. Both require precision.

1. **The Deep Periosteal Shot (The Gunshot Technique)** This is the classic technique. The goal is to place a bolus of high-G-prime filler directly onto the bone, underneath the temporalis muscle, to push the entire complex outward.

- **The Tool:** A sharp needle (usually 27G).

- **The Entry:** One centimetre up and one centimetre out from the lateral canthus (the tail of the brow).
- **The Execution:** Insert the needle perpendicular to the skin. Push straight through all the layers until you tap the bone. You must feel the hard stop.

Aspirate. Wait ten seconds. If it is clear, inject a slow, steady bolus of 0.5 to 1.0 ml.

- **The Result:** The filler acts like a wedge, lifting the muscle and fascia outward. It is a powerful, structural lift.

2. **The Superficial Subcutaneous Wash (The Cannula Technique)** If the patient has a wide, shallow hollow, or if you want to smooth the transition into the lateral cheek, the deep shot may look like a localized lump. In these cases, we act as architects and use the superficial subcutaneous plane to camouflage the deficit.

- **The Tool:** A 22G or 25G cannula.
- **The Entry:** Lower down, near the zygomatic arch or the hairline.
- **The Execution:** Glide the cannula into the superficial subcutaneous fat, just beneath the dermis but strictly above the superficial temporal fascia (where the artery lives).

You are creating a thin, even wash of softer filler across the entire hollow.

- **The Result:** A smooth, airbrushed finish that seamlessly blends the forehead into the cheek.

Product Selection Your technique dictates your product. If you are going deep onto the bone, you need a firm, cohesive filler that will hold its shape and lift the heavy muscle above it. Think Juvederm Voluma, Teosyal Ultra Deep, or Restylane Lyft. If you are doing a superficial cannula wash, a high-G- prime filler will look lumpy and unnatural.

You need something softer that integrates smoothly into the subcutaneous fat. Think Juvederm Volift, Teosyal RHA 3, or a slightly diluted biostimulator.

The Art of the Temple The temple requires restraint. The goal is not to make the temple bulge outward. A youthful temple is flat or very slightly convex. If you overfill it, the patient will look alien. Treat the temple, and you will notice something magical happen. The tail of the eyebrow will lift.

The transition from the cheekbone to the hairline will soften. The patient won't know exactly what you did, but they will know they look rested.

That is the hallmark of a quiet sculptor.

The Temple and the Brow: An Inseparable Relationship

The lateral brow is not held up by magic. It is held up by the soft tissue volume in the temporal region. When the deep temporal fat pad deflates, the lateral brow descends. The upper eyelid becomes heavier. The eye looks smaller, more tired, more aged.

This is one of the most common presentations in aesthetic practice, and it is one of the most commonly misdiagnosed. The patient presents complaining of heavy upper eyelids. They want blepharoplasty. They want a brow lift. They want Botox in their forehead to “open up” the eyes.

But the problem is not the eyelid. The problem is the temple. Restore the temporal volume, and the brow lifts passively. The eyelid lightens. The eye opens. No surgery. No brow lift. No aggressive Botox that leaves the patient looking perpetually surprised.

Always assess the temple before you treat the brow. The two are anatomically inseparable.

Complications to Know

The temple is one of the highest-risk regions in aesthetic medicine. The superficial temporal artery is large, palpable, and runs directly through the treatment zone. Inadvertent intra-arterial injection here can cause skin necrosis along the distribution of the artery, and in the most catastrophic cases, retrograde embolisation towards the ophthalmic artery and blindness.

The rule is simple: aspirate before every deep bolus injection in the temple. Use a sharp needle for the deep periosteal technique, not a cannula — the cannula cannot reliably reach the periosteal plane through the temporalis

muscle, and a cannula in the wrong plane in this region can still lacerate the artery. Inject slowly. Watch the skin. If you see blanching, stop immediately.

For the superficial cannula wash, the risk of arterial injection is lower, but the risk of venous compression is real. Do not inject large volumes in a single pass. Distribute the product in small aliquots, massaging between passes, and watch for any periorbital swelling.

The temple rewards the careful practitioner and punishes the careless one. Treat it with the respect it deserves.



CHAPTER 31

Lips — Knowing When to Stop

Lips were never supposed to be complicated. Years ago, lip treatment was simple. You restored hydration, corrected minor asymmetries, enhanced the Cupid's bow when appropriate, and stopped. Patients looked refreshed.

Friends noticed they looked well. Nobody noticed they had filler. Then social media arrived. Suddenly, lips became measurements, trends, and techniques. Every month a new method promised a more dramatic shape, a sharper border, or a larger pout. Yet the more aggressive the treatment became, the less natural the result often appeared.

I teach lips last. Not because they are the most technically difficult procedure in aesthetic medicine, but because restraint is the hardest skill to learn. Almost every practitioner can inject more filler. Very few know when to stop.

Why Lips Matter The lips occupy a surprisingly small area of the face, yet they command enormous attention. They are central to expression, communication, attraction, and identity. A smile is interpreted largely through the lips. Confidence, warmth, sensuality, sadness, and displeasure are all communicated through subtle changes in lip position and movement.

Throughout history, full, healthy lips have been associated with youth, fertility, vitality, and beauty. It is therefore unsurprising that lip enhancement remains one of the most requested aesthetic procedures worldwide. The challenge is that beautiful lips are rarely the biggest lips. Beautiful lips are balanced lips.

Understanding Lip Anatomy Before injecting a single drop of filler, understand the structure you are treating. The coloured portion of the lips is known as the vermilion. Unlike normal facial skin, the vermilion is thin and

highly vascular, allowing the underlying blood vessels to contribute to its characteristic colour.

The vermilion border is the transition line between the vermilion and surrounding facial skin. It frames the lips and contributes greatly to lip definition. The Cupid's bow forms the central contour of the upper lip, while the philtral columns create the vertical ridges extending from the upper lip towards the nose.

The lips are not smooth structures. They contain natural elevations known as tubercles. Typically, three tubercles are present in the upper lip and two in the lower lip. These natural projections create much of the lip's youthful shape and must be respected during treatment. Ignoring them produces the artificial lumps and irregularities commonly seen in poorly performed lip augmentation.

Beneath the surface, the lips are composed of several layers: • Epidermis • Dermis • Submucosal tissue • Orbicularis oris muscle Understanding these layers is critical because successful filler placement depends primarily on depth control. Injection anatomy is depth.

Everything else is secondary. [DIAGRAM: Lip Anatomy — vermilion border, Cupid's bow, philtral columns, tubercles (three upper, two lower), Glogau-Klein points, lateral commissures, upper and lower vermilion borders, upper and lower lip body.] The Dermis is the Training Ground Let us go back to first principles.

The product is called dermal filler. It was originally designed to be injected with a needle into the dermis. The viscosity of the gel, the gauge of the needle, the extrusion force required — everything was engineered for intradermal placement. Yet, somehow, we have a generation of injectors who have never mastered the dermis.

They go straight for deep volume. They go straight for the lips. And their lips look terrible. Why? Because injecting the lips requires speed, stability, and profound depth control. You have to put the needle in swiftly, find the exact plane instantly, inject retrograde with perfectly consistent pressure, and get out.

The longer you linger in the lip, the more it swells, the more it bruises, and the more the patient suffers. When a beginner takes too long in the lip, it is because they are lost. They don't know their depth.

They don't have the muscle memory to control the extrusion pressure while withdrawing the syringe. Their hand is not stable. If I am going to teach someone to inject lips properly, I do not start them on the lips. I start them in

the dermis around the lips.

I make them treat perioral lines. I make them treat the cutaneous upper lip. I make them execute full-needle-length, superficial retrograde injections in the deep dermis over and over again. Why? Because the dermis is unforgiving. If you are too superficial, you see a lump. If you are too deep, the line doesn't lift. You are forced to develop absolute precision. Once you have mastered the deep dermis — once your hand is stable, your depth perception is automatic, and your retrograde extrusion is perfectly smooth — then you are ready for the lips.

Because the lip is nothing more than a series of progressively thicker, softer tissue planes. When your hand has the muscle memory from the dermis, the actual hydration and augmentation of the lip becomes a swift, elegant, almost effortless procedure. The Layers of the Lip From an injector's perspective, the lip is not simply "skin and muscle." It is a highly stratified environment.

The total thickness from the skin surface to the wet oral mucosa is usually only 8 to 12 millimetres. Within that tiny space, everything happens. | Layer | Average Thickness | Clinical Relevance | — | — | — | | Epidermis | 0.05–0.10 mm | Extremely thin. Never inject. Topical anaesthetic works here. | | Papillary Dermis | 0.15–0.30 mm | Ideal for fine superficial wrinkles (smoker's lines). Very soft HA, tiny aliquots. | | Reticular Dermis | 0.5–1.0 mm | The structural dermis. Excellent plane for white roll enhancement and Cupid's bow definition. | | Subcutaneous Tissue | 1–4 mm | Contains fat and vessels. Excellent plane for volume support, lip body augmentation, and soft contouring. Cannulas travel extremely well here. | | Orbicularis Oris Muscle | 3–5 mm | The functional sphincter muscle. Most traditional lip fillers lie superficial to, within, or deep to this muscle. Offers surprisingly little resistance.

Deep Areolar Plane Variable Oral Mucosa 0.3–0.6 mm The vascular danger zone. Contains the superior and inferior labial arteries. Slow injections, small boluses. Cannulas reduce but do not eliminate risk. The wet lip. Rarely injected directly.

(Note: The total combined thickness of the dermis is only about 0.7 to 1.3 millimetres — roughly the thickness of a credit card. This surprises many injectors.) The White Roll is Not a Ligament Let us dispel a common anatomical myth. The white roll is one of the most misunderstood structures in the face.

It is not a separate anatomical ligament. It is an optical effect produced by dense collagen within the superficial dermis, changes in dermal thickness, light

reflection, and the junction between the cutaneous skin and the vermilion. Injecting immediately beneath this dermal structure — in the mid-to-deep reticular dermis — can restore crisp definition to the border without causing the dreaded “duck lip” excessive projection. Practical Injection Planes Precision in the lip comes not from finding an exact microscopic layer, but from knowing which tissue compartment you are in, how it behaves, and where the critical vascular structures lie. • White roll definition: Mid-to-deep reticular dermis. Restores the border without excessive projection.

- Vermilion border enhancement: Superficial subcutaneous (immediately deep to the dermis). Defines the edge while maintaining softness.
- Lip body augmentation: Subcutaneous tissue or superficial orbicularis oris. Adds volume with a natural contour.
- Central pout (tubercle): Subcutaneous tissue. Increases projection while preserving shape.
- Structural support in ageing lips: Deep subcutaneous plane, staying strictly superficial to the deep areolar (arterial) plane. Maximises support while reducing vascular risk.

The lip functions as a continuum of compliant soft tissue. Because HA is a soft gel, it diffuses within the surrounding connective tissue and behaves as a three-dimensional volume rather than a sharply defined line. Master the dermis first, respect the layers, and the lip will follow.

The Three Lip Types Before planning treatment, determine which lip type is sitting in front of you. Type One: The Small Elastic Lip These lips are tight, compact, and highly elastic. When filler is introduced, they resist expansion and often recoil like stretched rubber. Attempting dramatic augmentation in a single session usually produces excessive projection rather than attractive volume.

These lips require training over time. Patience creates beauty. Aggression creates distortion. Type Two: The Ageing Lip These patients once had attractive lips. Their lips have lost hydration, support, projection, and definition through ageing. The upper lip may lengthen and invert. The lower lip may become thinner.

The corners begin to descend. The vermilion border softens. The Cupid’s bow gradually fades. These patients rarely want transformation. They want restoration. Type Three: The Naturally Full Lip These lips already possess significant volume. Here, even a small amount of filler can disrupt proportion.

One careless fraction of a millilitre may convert a naturally attractive lip into an obviously treated one.

In these patients, treatment is rarely about volume. It is about refinement.

Lip Ageing Ageing affects the lips far beyond simple volume loss. Collagen declines. Fat pads diminish. Muscle support weakens. The vermilion gradually loses vascularity and colour. Structural changes occur throughout the face: • The upper lip lengthens and inverts • The lower lip rolls inward • The commissures descend • Philtral columns flatten • The vermilion border thins

- Perioral lines become more apparent • Dental support decreases •

Bone resorption reduces lip projection These changes explain why youthful lips cannot simply be recreated by injecting volume alone. A youthful lip is a structural relationship. Not a volume measurement.

The Consultation Most patients arrive carrying photographs. They have saved images from Instagram, TikTok, or celebrity profiles. They have rehearsed descriptions of exactly what they believe they want. A degree of selective amnesia can be useful. Listen respectfully.

Then assess the anatomy. Patients frequently request excessive volume when what they truly need is hydration, support, or subtle restoration. My preference for first-time patients is conservative. A small amount of filler is placed throughout the lower lip, followed by modest treatment of the upper lip.

The result is then reassessed. At that moment, I hand them a mirror. Watch carefully. Often their words remain cautious. Their pupils do not. The eyes frequently reveal satisfaction before the patient consciously realises it. The sympathetic nervous system cannot suppress a happy response. The pupils give you the truth that the words cannot.

The Eight-Segment Framework This is the most important section of this chapter. Most practitioners think of the lips as two structures — upper and lower. That is too crude. It leads to uneven results, missed volume, and the classic “sausage lip” that announces bad filler from across the room.

The lips are eight segments. The Lower Lip — Four Segments Draw an imaginary line from the oral commissure to the midline of the lower lip. That gives you the halfway point. • Segment 1: Oral commissure to the halfway point, left side • Segment 2: Halfway point to midline, left side • Segment 3: Midline to halfway point, right side • Segment 4: Halfway point to oral commissure, right side Each segment receives its own thread.

Each segment is treated as an independent unit. The Upper Lip — Six Segments The upper lip is more complex than the lower because of the Cupid's bow. It has six segments, not four.

Four of them run horizontally along the vermilion border. Two of them run vertically, connecting the peaks of the Cupid's bow to the body of the lip. The Four Horizontal Border Segments: • Segment 1: One needle-length from the left oral commissure towards the left peak of the Cupid's bow • Segment 2: From the left peak of the Cupid's bow to the midline (the central dip) • Segment 3: From the midline to the right peak of the Cupid's bow • Segment 4: One needle-length from the right peak of the Cupid's bow towards the right oral commissure The Two Vertical Body Segments: • Segment 5: From the top of the left Cupid's bow peak downward to the midpoint of the upper lip body • Segment 6: From the top of the right Cupid's bow peak downward to the midpoint of the upper lip body These two vertical segments are what give the upper lip its three-dimensional fullness.

Without them, the border is defined but the body is flat. With them, the lip has depth, projection, and the soft central pout that makes a treated lip look natural rather than outlined. Plain Text LEFT PEAK RIGHT PEAK

[Seg 1] [Seg 2] [central]

[Seg 3] [Seg 4] [Seg 5] [Seg 6] (lip body midpoint — no injection at midline)

The central dip of the Cupid's bow — the midline — is left alone in all segments. You do not inject here. Injecting into the central dip flattens the Cupid's bow and erases the most architecturally important feature of the upper lip. "The Cupid's bow is not a problem to solve. It is a landmark to protect."

Volume — The Numbers That Matter The target volume per segment is 0.05 millilitres. Ten segments. Ten threads. Ten times 0.05 ml. That is a total of 0.5 ml for the entire mouth — four segments in the lower lip, six segments in the upper lip. This is the ideal volume for a first treatment in most patients.

It hydrates. It softens. It creates a subtle, natural improvement that patients love and nobody else can identify. Most beginners will place 0.1 to 0.2 ml per segment. That is normal.

Precision takes time. As your technique develops, your deposits will become smaller, more controlled, and more evenly distributed. The goal is 0.05 ml per segment. The reality for most beginners is 0.1 to 0.2 ml per segment. Both are acceptable.

The direction of travel is what matters. Volume Reference Table Treatment Goal Hydration only (first treatment) Subtle augmentation Moderate augmentation Total Volume 0.5 ml 0.6–0.8 ml 1.0 ml Significant augmentation 1.0 ml + review Per Segment 0.05 ml 0.075–0.1 ml 0.125 ml Never exceed 1.0 ml in one session Do not exceed 1.0 ml in a single session. The tissue cannot accommodate more without distortion. If the patient wants more, bring them back in four weeks.

Injection Anatomy and Technique The ideal plane for most lip augmentation lies within the submucosal layer. Too superficial and the filler becomes visible, irregular, or palpable. Too deep and the product may interfere with mobility, producing stiffness and an unnatural feel. The goal is controlled placement beneath the lip surface while preserving natural movement. A practical way to appreciate depth: when the lip is gently tented, the needle should remain visible beneath the surface. When the tissue relaxes, the needle should no longer be clearly seen. That is usually the correct plane.

The Technique: Linear Threading with Slow Withdrawal Use a 27G or 30G needle. Insert from the corner of the lip and advance through the segment to its full length. Then deposit the filler during withdrawal — slow, steady, retrograde. The filler follows the needle out of the tissue, creating a smooth, even thread.

The key word is slow. Quick withdrawal deposits filler unevenly. Slow withdrawal distributes it precisely. Quick injection creates boluses. Slow injection creates threads. Boluses lump. Threads smooth. The mantra is: inject in, slow withdrawal. The Entry Point For the lower lip: enter at the oral commissure, advance medially along the segment, deposit on withdrawal.

For the upper lip: enter at the oral commissure for the lateral segment, advance towards the Cupid's bow peak. For the medial segments, you may need a second entry point near the philtral column. Do not enter through the vermilion border. Enter through the wet-dry line or from the commissure.

What to Avoid The central dip of the Cupid's bow. Leave it alone. Every time. The vermilion border in young patients. If the border is defined, do not touch it. Injecting into a healthy border creates a shelf — an unnatural ridge that shouts filler. The lateral corners of the lower lip.

This is the most commonly missed area. The lateral lower lip loses volume early with age and contributes significantly to lip balance. Treat it. Large boluses anywhere. A bolus is not a thread.

A bolus is a lump waiting to happen.

Excessive volume in one session. The tissue has a limit. Respect it.

A Word on Vertical Lip Techniques Every few years a new lip trend appears. Russian lips. Tent lips. Vertical lifts. Most are visually impressive immediately after treatment because swelling exaggerates the effect. The lip appears taller, sharper, and more dramatic. Unfortunately, swelling is not the result.

The healed tissue is the result. Once swelling resolves, excessive vertical techniques often produce rigidity, reduced mobility, overemphasised borders, and an unnatural upper lip contour. They prioritise geometry over physiology.

The lips were designed to move. Any technique that compromises that movement should be approached with caution.

The Vermilion Border The vermilion border deserves respect. In older patients who have genuinely lost border definition, careful restoration can be transformative. Small amounts of soft filler can recreate structure and improve lip aesthetics without obvious augmentation. In younger patients, however, the border is frequently already well defined.

Injecting filler into a healthy border simply because it can be done is unnecessary. Treat the border when it has been lost. Leave it alone when it remains beautiful.

Vascular Considerations The lips possess an exceptionally rich vascular network. The superior and inferior labial arteries, branches of the facial artery, provide the primary blood supply. Numerous anatomical variations exist, and vessels may not follow predictable pathways. For this reason, practitioners should assume arteries may be present almost anywhere within the lip. Several principles improve safety:

- Maintain appropriate depth
- Inject slowly

- Use retrograde linear threading
- Avoid large boluses
- Continuously observe tissue response

Understanding anatomy reduces risk. Respecting anatomy reduces it further.

Complications Most lip filler treatments are uncomplicated. Common side effects include swelling, bruising, and tenderness. More significant complications include infection, nodules, granuloma formation, filler migration, vascular compromise, and tissue necrosis. Serious complications remain uncommon when treatment is performed thoughtfully and conservatively. The overwhelming majority of problems I encounter are not caused by anatomy. They are caused by excess. Too much filler. Too quickly.

In the wrong patient.

The Lip in Summary — Everything You Need to Know and Nothing You Don't Let me save you from yourself. The lip is 8 to 12 millimetres thick. That is it.

From the skin surface to the oral cavity, you have less than a centimetre of tissue to work with. Inside that centimetre, you have the epidermis — which you never touch — the papillary dermis, the reticular dermis, the subcutaneous tissue, the orbicularis oris muscle, the deep areolar plane where the labial arteries live, and the oral mucosa.

The entire dermis — both layers combined — is about the thickness of a credit card. Let that sink in. Most injectors who have been doing this for years have never truly appreciated how thin the dermis is. They talk about “intradermal” injections as if they are navigating a vast landscape.

They are not. They are navigating a credit card. Precision is not optional. It is the only thing. Here is what each layer is actually for. The papillary dermis is for smoker's lines and fine perioral rhytids. Tiny amounts of the softest HA you own, placed with a needle, retrograde, with the lightest possible touch.

If you see a white ridge, you are too superficial. If the line doesn't lift, you are too deep. There is no middle ground. There is only right or wrong.

The reticular dermis is for the white roll, the Cupid's bow, and border definition. This is where you restore the frame of the lip without adding volume to the body of it. Most people who want “definition” actually want this layer treated. Not more filler in the body.

More precision at the border. The subcutaneous tissue is where the volume lives. The body of the lip. The tubercles. The pout. The cannula glides here. The HA diffuses here.

This is the forgiving layer — relatively speaking. The orbicularis oris is the muscle that does everything. It speaks. It smiles. It kisses. It eats. It is in constant motion.

Filler placed here will be squeezed, compressed, and redistributed every single day. Respect it. Do not overfill it. The deep areolar plane is where the labial arteries run. This is the layer you do not want to be in. If you are here, you are one misplaced bolus away from a vascular event.

Slow down. Small volumes. Aspiration is unreliable in the lip. Your best protection is not technique — it is restraint. Now. The white roll. It is not a ligament.

It is not a separate anatomical structure. It is an optical illusion produced by dense collagen, changes in dermal thickness, and the way light bounces off the junction between the cutaneous skin and the vermilion. Injecting immediately beneath it — in the mid-to-deep reticular dermis — restores the definition without the duck.

Injecting into it, or above it, produces a ridge that looks like someone drew a line around the lip with a marker. And the vermilion itself. It is not normal skin.

It has almost no keratin, no hair follicles, no sweat glands. It is thin, highly vascular, and entirely unforgiving. The colour you see is the underlying vasculature. Treat it with the same reverence you would treat the tear trough. The practical rules are simple.

If you want the border, work in the dermis. If you want the body, work in the subcutaneous tissue. If you want the pout, work in the subcutaneous tissue at the tubercle. If you want to support an ageing lip, work deep subcutaneous — but stay above the arteries.

If you want to treat the oral commissures, work deep subcutaneous at the corners. And if you are not sure which layer you are in? You are probably too deep.

The lip does not reward guessing. It rewards repetition, restraint, and the kind of quiet confidence that only comes from having done the same thing correctly a thousand times before you attempted it on a face that matters.

Master the dermis. Learn the layers. Earn the lip.

Final Thoughts The secret to beautiful lips is surprisingly simple. Respect anatomy. Respect ageing. Respect movement. Respect tubercles. Respect proportion. Most importantly, respect restraint. Patients rarely regret the syringe you did not inject. They often regret the one you did. The best lip treatment should leave people wondering why the patient suddenly looks healthier, happier, and more attractive. Not wondering where they had their lips done.

CHAPTER 32

Everything That Moves — The Muscles of Expression

The muscles of the face are not like the muscles of the body. They do not move bones. They move skin. They are attached, at one end, to bone, and at the other end, directly to the dermis. This is why, over time, those movements etch themselves into the skin as lines and wrinkles.

But there is a more fundamental difference. In the body, muscles exist in opposing pairs that pull against rigid bone across a hinge joint. Flexion and extension. Bicep and tricep.

The face uses opposing forces, but the mechanics are entirely different. Instead of pulling against a rigid bone in a strict back-and-forth plane, facial muscles typically originate from bone or fascia and insert directly into the skin. When one muscle contracts, it actively pulls the skin in one direction, while its antagonist must relax to allow that movement.

Dropping an eyebrow is not simply the frontalis relaxing and letting gravity take over. The frontalis ceases its upward pull, but the actual dropping motion is an active contraction of the depressors (the procerus, corrugators, and orbicularis oculi). Pouting and smiling involve complex, coordinated antagonism.

The zygomaticus major pulls the corners of the mouth up and outward, while its antagonist, the depressor anguli oris (DAO), pulls them downward. When you smile, the zygomaticus fires and the DAO relaxes. When you inject botulinum toxin, you are not just paralysing a muscle.

You are altering the balance of a dynamic, interconnected system of opposing forces. You are a conductor. You choose which section of the

orchestra to quieten so that another can be heard more clearly.

A word about depth, before we touch a single muscle

There is a piece of vocabulary this whole field uses, and it is quietly wrong.

The four injection planes, by approximate depth. Categories, not rigid boundaries.

We say “deep” and “superficial” as though they were opposite ends of a long road. On the face, they are usually not. On much of the face the difference between a deep injection and a superficial one is two to four millimetres — the thickness of a stack of a few coins. A practitioner picturing “deep” as something like a centimetre down is already lost before the needle goes in, because a centimetre, in the upper face, is through the muscle, past it, and into a plane you did not intend to be in. So I am going to ask you to stop thinking in two words and start thinking in four planes. They are not rigid categories — the whole point of this section is that they are not — but they give you a vocabulary that matches the tissue instead of misleading you about it.

Plane 1 — intradermal. Roughly 0.5–1.5 mm. Within the dermis itself, often with a visible bleb. Micro-toxin, the most superficial lower-eyelid work, skin-level modulation.

Plane 2 — subdermal, or superficial muscle. Roughly 1–3 mm. Just beneath the dermis, or into a thin mimetic muscle sitting right under it. The frontalis lives here. So does the orbicularis oculi, the orbicularis oris, the platysma, the lateral tail of the corrugator.

Plane 3 — standard intramuscular. Roughly 3–6 mm. Into the body of a muscle that sits a little deeper. The procerus, the corrugator body, the DAO, the nasal elevators.

Plane 4 — deep intramuscular. From roughly 5 mm down to bone, and in some regions a great deal further. The medial corrugator origin, the mentalis, and — in a different league entirely — the masseter and temporalis, which are measured in centimetres rather than millimetres. That is the map. Now here is the thing the map cannot tell you, and it is the most important sentence in this chapter.

Depth is relative to the patient, not to the textbook

Every number I have just given you, and every number in the table that follows, is an average. And an average face does not walk into your clinic. A specific one does, with its own skin thickness, its own fat, its own build, its own history.

Here is what actually rescues you from the tyranny of the average: **on a single patient, the muscles keep their relationships to each other**

even when their absolute depths change.

Take a thin patient with fine skin and little subcutaneous fat. Their procerus is not at the textbook ~5–6 mm. It is at four, because there is simply less tissue on top of it. And the moment you know that, you know something about the rest of that face — because the same thinness that put their procerus at four millimetres has put their corrugator, their mentalis, their nasal elevators proportionally shallower too. It is the same skin, the same fat, the same build across the whole face. Read one muscle honestly and you have calibrated the entire patient.

The reverse is just as true. A heavier face with thick, sebaceous skin will run deeper than the average at every point, in proportion. Their frontalis is not where the diagram says; it is a millimetre or two lower, and so is everything else.

So the practitioner's real skill is not memorising a depth for each muscle. It is establishing this patient's *offset* from the average — shallow, standard, or deep — usually within the first one or two injections of a treatment, and then carrying that offset across the whole face. The table gives you the relationships. The patient in the chair gives you the multiplier.

This is why an experienced injector can work on a face they have never treated and be right about depth almost immediately, while a practitioner following fixed millimetre figures is wrong on half the muscles the first patient who is not average sits down. The experienced one is not remembering better numbers. They are reading the relationship and letting the patient set the scale.

The facial toxin depth map

Read this as a set of relationships to hold against each other, not a prescription to apply blind. Every figure assumes an average adult and describes the approximate depth of the deposit *from the uncompressed skin surface* — not how much needle you have buried, which is a different and less useful

measurement, for reasons I will come to.

Target	Approx. depth	Plane
Frontalis	1.5–3 mm	Superficial IM / subdermal
Medial corrugator origin	4–7 mm	Deep IM, often near periosteum
Corrugator body	3–5 mm	Intramuscular
Lateral corrugator tail	1.5–3 mm	Superficial IM
Procerus	3–5 mm	IM (not necessarily to bone)
Depressor supercilii	2–4 mm	Superficial-to-mid IM
Orbicularis oculi (crow's feet)	1–2.5 mm	Very superficial IM / subdermal
Infraorbital orbicularis	1–2 mm	Intradermal / immediately subdermal
Nasalis (transverse, bunny lines)	2–4 mm	Superficial IM
Levator labii superioris alaeque nasi	3–5 mm	Targeted IM
Gummy-smile elevator complex	3–6 mm	IM
Depressor septi nasi	3–5 mm	IM near columellar base
Orbicularis oris (lip flip)	1–2 mm	Very superficial / subdermal
DAO	3–5 mm	Intramuscular
Mentalis	4–7 mm	Deep IM, often towards bone
Depressor labii inferioris	2–4 mm	Superficial IM
Masseter	8–15+ mm	IM, varies hugely with bulk
Temporalis	8–15+ mm	IM, to bone then withdraw
Platysma bands / Nefertiti	2–4 mm	Superficial IM
Trapezius	10–20+ mm	IM, highly tissue-dependent

Two entries in that table deserve to be understood rather than memorised, because they are the ones that teach the principle.

The **corrugator** is the clearest lesson in the whole chapter: it is not one depth, because it is not at one depth. It originates deep, medially, against the superciliary arch — Plane 4 — and as it travels laterally it climbs, thinning and

rising until its tail is almost subdermal, Plane 2. One muscle, two planes, a couple of centimetres apart. A useful phrase for trainees: *the corrugator begins deep and finishes superficial*. And a safety consequence: a deep injection out at the lateral tail is not a slightly-too-deep corrugator injection, it is an injection heading towards structures deep in the orbit, which is how you produce a ptosis.

The **orbicularis oculi**, at the other extreme, is one of the thinnest muscles you will ever treat — the ultrasound literature puts both its thickness and its depth at ~1.5 mm, less than a millimetre in places. A four-millimetre injection here is not “a little deeper.” It is through the muscle and into somewhere you did not mean to be. When people ask why crow’s feet is where beginners cause trouble, this is the answer: there is almost no margin for error in the vertical dimension, because there is almost no vertical dimension.

What a stated depth does not mean

One last thing before the muscles, because it is the caveat that makes the numbers honest.

When I say a muscle sits at five millimetres, I do not mean “bury five millimetres of needle.” Those are different quantities, and confusing them is how a careful-looking injection lands in the wrong plane. A stated deposition depth is not a needle-insertion measurement, because: The needle compresses the skin as it enters — often by 1–3 mm. Push in five millimetres of steel and the tissue you have flattened springs back, and your bevel is deeper than you think.

An oblique entry travels further through tissue to reach the same perpendicular depth. The angle changes the arithmetic.

The bevel itself is around a millimetre long, so where the toxin actually leaves the needle is not quite where the tip is.

Pinching a muscle lifts it away from what lies beneath, changing the depth and the safety margin at once.

So the mental model to carry is not a ruler on the needle. It is this:

Depth is not how much needle disappears. It is the anatomical

layer in which the bevel comes to rest.

Hold that sentence, hold the four planes, and hold the fact that this particular patient may run shallow or deep across all of them together — and you can treat a face you have never seen before and be right about depth from the first muscle.

But depth is only half of it, and on its own it will still put your product in the wrong place.

Angle is the other half of depth

Here is the thing the depth table cannot show you, because a table has no third dimension.

From a single entry point, a perpendicular needle reaches the deep muscle while a shallow-angled one deposits into the muscle above it.

Angle converts a depth into a destination.

Two practitioners stand at the same patient, choose the same entry point, and advance the same five millimetres. One holds the needle at ninety degrees, straight down. The other holds it at forty-five. They have used an identical entry point and an identical length of travel, and they have deposited their product in two entirely different places — different depth, and often different muscle.

The ninety-degree needle goes down. Five millimetres of vertical travel is five millimetres of depth, and over the corrugator origin that puts you into the corrugator, near bone. The forty-five-degree needle, from the very same spot, travels those same five millimetres mostly *across* rather than *down* — it ends up perhaps three and a half millimetres deep and a few millimetres away horizontally, which over that same spot can place the product not in the corrugator at all, but up in the overlying frontalis. Same entrance. Same distance. A different muscle switched off. And if you did not intend the frontalis, you have just given a patient a heavy brow they did not ask for, from an injection you would have sworn was a corrugator injection.

So the instruction “enter here and go to five millimetres” is incomplete to the point of being dangerous. The full instruction is always three things together: **where you enter, how deep you go, and at what angle you travel** — because the angle is what converts a depth into a destination. Depth tells you how far the bevel is from the surface. Angle tells you which structure it has

arrived in.

This is why the same muscle appears in this book with different techniques. The procerus you enter at ninety degrees, straight to bone, because you want it *there* and nowhere else. A superficial frontalis modulation near the brow you approach shallow and flat, because a perpendicular jab risks carrying you deeper than the thin muscle you are aiming at. The corrugator you often enter perpendicular medially and change your approach as you move laterally, because the muscle itself is diving and climbing beneath you.

A few working rules that follow from this:

Perpendicular for depth, oblique for control. When you want to reach a deep target cleanly and keep the product in a tight spot — procerus, mentalis, medial corrugator — go in at or near ninety degrees. When you want to stay in a thin superficial muscle and spread along it rather than punch through it, go shallow and flat.

A shallow angle is how superficial injections stay superficial.

The commonest way to end up deeper than you meant is to inject a thin muscle perpendicularly. The needle wants to keep going. Lay the angle down and the tissue you are aiming at stays under the bevel instead of passing it.

Over a thin muscle sitting on a thicker one, angle decides

which you get. Frontalis over the deeper frown musculature;

orbicularis over the structures at the orbital rim. Perpendicular risks the deeper one; flat keeps you in the one you want.

State the angle when you teach or document, every time. “0.1

ml, procerus, 90° to bone” is a technique. “0.1 ml, procerus” is a hope.

Put the two halves together and you have the actual mental model. You are not aiming a needle at a depth. You are aiming a bevel at a *layer within a specific muscle*, and you get there by choosing three things at once — entry point, depth of travel, and angle of approach — on a patient whose whole face may run shallower or deeper than the average. Master that and the individual doses below become what they should be: the easy part.

That is the whole of it. Everything below is detail hung on that frame.

The Frown Complex

The frown is the anchor of the upper face. It is powered by the procerus in the centre and the corrugators on either side. The Procerus The procerus pulls down.

We inject this at a 90-degree angle, straight down to the bone. The standard volume is 0.1 ml of solution — which, depending on your dilution, equates to 5 units of Botox/Bocouture or 15 units of Azzalure. The Corrugators The corrugators pull down and in.

This is where your non-injecting hand becomes your most important tool. You inject from one side. Your non-injecting hand rests firmly on the patient's face. Your thumb and your index finger sit literally on the eyebrows, and your palm rests over the nose. It is a very accurate, stable axle to make this grip.

From this position, you can inject the procerus and the corrugators medially and laterally with total control. While the procerus is injected at 90 degrees (quite vertical), the rest of the injections are angled away from the eye. You are directing the needle and the product away from the orbit to protect the levator palpebrae superioris.

In the lateral tail of the corrugator, I use what I call a “dash.” A dash is barely calculable. It is a volume of 0.025 ml — half of a standard 0.05 ml unit dose. It is just enough to quiet the tail without freezing the brow.

Relativity Over Recipes There is no standard dose for a frown. There is no universal protocol for a forehead. Every face is different. Every muscle has a different strength, a different size, a different relationship to the other muscles around it. The dose that gives one patient a beautiful, natural result will give another a heavy, dropped brow.

You must observe the patient. Watch them speak. Watch them smile, frown, and laugh. See the dance of the muscles. Understand the logic of the movement. You will know exactly where to place your needle.

The Danger of the Depressor Anguli Oris

The depressor anguli oris — the DAO — is one of the most commonly treated muscles in the lower face, and one of the most commonly overtreated. It pulls the corners of the mouth downward, creating the “sad mouth” appearance that ages the lower face dramatically. When you relax it with toxin, the corners lift. The patient looks happier, more approachable, more rested.

But the DAO is anatomically variable. It overlaps with the depressor labii inferioris and, critically, with the mentalis. If you inject too medially, you will catch the depressor labii and the patient will develop an asymmetric smile. If you inject too deep, you will catch the mentalis and the patient will lose the ability to purse their lower lip.

If you inject too much, the patient will look like they have had a stroke. The DAO is not a forgiving muscle. Inject it laterally, at the mandibular border, with a conservative dose. Watch the patient smile before you inject. Watch them smile after. The correction should be subtle. If it is obvious, you have done too much.

The Platysma: The Forgotten Muscle

The platysma is the broad, flat muscle that runs from the chest up through the neck and inserts into the lower face. As we age, it loses its tone. The muscle bands become visible as vertical cords running down the neck. The jawline blurs as the platysmal bands pull the lower face downward.

Treating the platysma with botulinum toxin — the Nefertiti lift — is one of the most underutilised techniques in aesthetic practice. By relaxing the downward pull of the platysmal bands, you allow the elevators of the lower face to work unopposed. The jawline sharpens. The jowls lift slightly. The neck looks longer and more defined.

The technique is simple: identify the platysmal bands by asking the patient to clench their teeth and pull the corners of their mouth downward. The bands become visible. Inject along each band with small aliquots of toxin, spaced approximately two centimetres apart. The dose is conservative — typically two to four units per injection point.

The risk is dysphagia if the product diffuses to the deep neck muscles. Inject superficially, into the muscle only, and stay lateral to the midline. The platysma is a thin muscle. You do not need to go deep.

Reading the Face in Motion

The most important skill in botulinum toxin practice is not knowing where to inject. It is knowing where not to inject.

Every face has a compensatory pattern. When one muscle is weak, another muscle overworks to compensate. When you inject the procerus and

corrugators, the frontalis may overcompensate, raising the brows too high. When you inject the crow's feet, the zygomaticus may overcompensate, pulling the cheek upward and creating a bunny line.

You must read the face in motion before you touch it. Ask the patient to frown. Ask them to smile. Ask them to raise their brows. Watch what happens. Identify the dominant muscles and the compensatory patterns. Then design your treatment plan around the entire system, not just the individual muscle.

A toxin treatment is a conversation with a system of muscles. You are not silencing one voice. You are rebalancing a choir.



CHAPTER 33

Stop Guessing — The Upper Face, Precisely

The upper face is where most practitioners begin their Botox journey. It is where most of the early mistakes are made.

The frown, the forehead, the crow's feet. These are the classic three. They are treated as a system, not as three independent areas. The Frown The frown is the anchor. You treat it first.

The glabellar injections spread slightly upward and lighten the load on the lower frontalis. The procerus gets a single injection at its mid-point. The medial corrugators get a deep injection. Deep enough that the bubble you create moves when the patient frowns. This confirms you are in the correct muscle.

The lateral corrugators get a smaller, more superficial injection. The Forehead The forehead is treated second, but it requires far more nuance than a simple map. The traditional rule taught on basic courses is: Never inject the frontalis below the midheight of the forehead. This is not a rule.

It is a guideline for beginners. In reality, your approach depends entirely on the patient's age and the condition of their upper eyelids. If a patient over thirty already has visible upper eyelid hooding and they ask for "forehead only," you are walking into a trap.

Even if you treat high up, if you give a strong enough dose to relax the upper half, it can still cause ptosis. The frontalis is a single sheet, but it functions almost like two parts — the upper half and the lower half — which have complex, sometimes opposing effects on the brow position. This is why I have moved almost entirely to microdosing the forehead. Using a 32-gauge needle, I use very small amounts spread across a wider area. For a female patient, I might

use 10 to 15 units of Botox (or 30 units of Azzalure) spread across the whole forehead, including a few “dashes” (0.025 ml) over the lateral brow if they have the classic “Wi-Fi sign” (curved lines over the lateral eyebrow).

You have to look at hundreds of foreheads to understand where the dose should go. My minimum total forehead dose is usually 7.5 units of Botox; my maximum is around 15 units. For a man, I might go up to 20 units. But the crucial question is always: Am I treating the upper half or the lower half?

Which one is stronger? Am I giving an equal microdose across the whole muscle, or am I biasing the dose higher up? When in doubt, give less than you think you need. The traditional method is four injections across the frontalis. Take each injection point as the centre of a 2 cm radius.

Does that 2 cm circle cover the wrinkling? If yes, leave it there. If the forehead is tall and you see wrinkling way past your 2 cm radius over the lateral eyebrows, put a small dash in each eyebrow corner. For females, that works well. For males, you might just need to add two more standard injections. Whether those central injections sit above or below the lateral ones depends entirely on how big the forehead is and where the wrinkling is happening.

Look at the face, not the textbook map. The Crow’s Feet The crow’s feet are treated last. The standard approach is three or four points. Placed at least one centimetre from the lateral orbital rim, in a gentle arc following the natural crease pattern. The upper-lateral point gives a subtle but beautiful brow lift.

The lower-lateral point gives gentle support to the lower crow’s feet. It avoids the risk of the under-eye roll that comes from treating too close to the rim. The system behind the upper face is a triangle. Frown first, to stabilise the centre.

Forehead second, to control the lines and tone. Crow’s feet last, to lift and refine the edges. This sequence produces the most natural expression and the fewest complications.

When Aesthetics Accidentally Discovered Neurology — Botox and Migraine There is a recurring pattern in medicine where a treatment developed for one purpose quietly solves a completely different problem. Sildenafil was developed for angina. It did not work especially well for angina. But the trial participants were reluctant to return their unused tablets, which told the researchers something interesting.

Botulinum toxin followed a similar trajectory. In the late 1990s, aesthetic practitioners began noticing something unexpected. Patients who came in for

frown line treatment — glabellar injections, 20 to 30 units, standard cosmetic protocol — were coming back at their review appointments and mentioning, almost as an aside, that their migraines had improved.

Not a little. Significantly. Some patients who had suffered chronic headaches for years reported that the frequency had dropped by half. Nobody had planned this. Nobody had designed a trial around it. It was a side effect of treating a cosmetic concern, and it was too consistent to ignore.

In 2000, Binder published the first open-label study documenting migraine benefit from botulinum toxin. Allergan, the manufacturer of BOTOX, took notice and launched a clinical programme that would eventually produce the PREEMPT trials — the largest randomised controlled trial series ever conducted in chronic migraine prevention. What those trials found changed the way neurology treats one of the most disabling conditions in the world. Why the Aesthetic Dose Is Not the Therapeutic Dose Here is where it gets interesting.

The aesthetic practitioner who treats a frown uses 20 to 30 units across five or six points in the glabellar complex. The patient mentions their headaches improved. The practitioner thinks: I am treating migraines. They are not.

Not really. The PREEMPT protocol — the only regimen with robust randomised controlled trial evidence and regulatory approval for chronic migraine — looks nothing like a cosmetic treatment plan. Total dose Injection sites Aesthetic Approach 20–50 units 5–11 Muscle groups Frontalis, corrugator, procerus Frequency Target Evidence base Every 3–6 months Cosmetic wrinkle reduction Observational, case reports PREEMPT Protocol 155–195 units 31–39 7 muscle groups including occipitalis, cervical paraspinal, trapezius Every 12 weeks Chronic migraine prevention Phase 3 RCT (PREEMPT 1 and 2)

The neurologist is not being excessive. They are following the only evidence-based protocol that has been shown to reduce migraine frequency in chronic migraine patients. The seven muscle groups — including the back of the head, the neck, and the trapezius — are there because that is where the evidence pointed.

The aesthetic practitioner who sees migraine improvement after a frown treatment is observing a real phenomenon. But they are observing it in a different population (episodic migraine, not chronic), at a different dose (cosmetic, not therapeutic), and at a different set of sites (anterior only, not full

head and neck).

These are not the same thing. Who Actually Qualifies The licensing is specific and non-negotiable. OnabotulinumtoxinA (BOTOX) is licensed for chronic migraine only — defined as fifteen or more headache days per month for more than three months, with at least eight of those days having migraine features. NICE TA260 endorses this indication and defines the commissioning criteria for NHS treatment.

It is not licensed for episodic migraine (fewer than fifteen headache days per month). Multiple placebo-controlled trials in episodic migraine have been negative or inconsistent. The early paradoxical signal — where 25 units outperformed 75 units in one small trial — is now understood as likely reflecting placebo effects and random error rather than a genuine dose-response relationship.

This matters for aesthetic practitioners because the patients who mention migraine improvement after a cosmetic treatment are almost always episodic migraine sufferers. They feel better. The treatment is not doing what PREEMPT does. What is more likely happening is a combination of reduced muscle tension in the frontalis and corrugator, reduced trigger point activity, and a genuine but partial effect that does not replicate the full therapeutic benefit of the licensed protocol.

The Clinical Takeaway If a patient mentions migraine improvement after a cosmetic treatment, acknowledge it, document it, and refer them to a neurologist if their headache burden is significant. Do not expand your treatment plan to mimic the PREEMPT protocol without appropriate training, licensing, and clinical governance.

The PREEMPT protocol is a therapeutic intervention, not a cosmetic one. It requires a different clinical framework, different patient selection criteria, and a different consent process. What you can say, honestly, is this: the discovery that botulinum toxin has any effect on migraine at all came from aesthetic medicine.

The patients who sat in cosmetic clinics and mentioned their headaches were the original signal. The neurologists took that signal, ran the trials, and built the evidence base. Aesthetics asked the question.

Neurology answered it. For a full clinical review of the PREEMPT protocol, dosing evidence, and the chronic versus episodic migraine debate, read the HSI Journal article at the QR code below.[QR Code — HSI Journal:

OnabotulinumtoxinA for Migraine Prevention]<https://www.harleystreetinstitute.com/journal/botox-migraine-preventionpreempt-protocol-review> The Forehead — Treating the Elevator Without Dropping the Brow The frontalis muscle is the only thing keeping your patient's eyebrows off their eyelids.

It is the sole elevator of the upper face.

If you over-treat it, the brow drops. The patient looks angry, tired, and Neanderthal. It is the most common, most entirely avoidable botox complication in the upper face. The Assessment Before you touch a needle, look at the brow position. The ideal female brow arches above the orbital rim.

The ideal male brow sits right at the orbital rim. The Rule of the Lower Third Never, ever inject the lower 2 centimetres of the frontalis. That is the danger zone.

That is the tissue doing the heavy lifting. You inject only the upper two-thirds. The Technique Four to six injection points in the upper frontalis. Two to four units per point.

Use a 30G needle. If you inject more laterally than medially, you create a lateral brow lift (the infamous 'Spock brow' if you overdo it). Symmetrical injection maintains the natural arch.

Dosing is usually 8-15 units total for women, and 15-25 units for men because their muscle mass is heavier. Always review forehead botox at two weeks. The brow position is the only outcome that actually matters. When addressing the upper face with botulinum toxin, the forehead presents a unique challenge and, frankly, the most common pitfall: the dreaded brow drop.

Understand this fundamental truth: the frontalis muscle is the sole elevator of the brow. Over-treat it, and gravity, along with the depressor muscles, will inevitably win, pulling the brow downwards. This isn't just an aesthetic faux pas; it's a functional concern and a significant source of patient dissatisfaction.

Your primary directive here is to relax the forehead lines without compromising the natural, or desired, brow position. Before you even consider drawing up a syringe, a meticulous assessment of the brow position is non-negotiable. The ideal female brow typically arches gracefully above the supraorbital rim, creating an open, refreshed appearance.

In contrast, the ideal male brow tends to sit directly at or just below the supraorbital rim, conveying a more masculine, defined look. These are not hard and fast rules, but crucial benchmarks. Always assess the patient's natural

resting brow position, any compensatory elevation, and their dynamic expressions before marking a single injection point.

Ignoring this step is akin to flying blind. Now, for the golden rule, etched in the annals of upper face botox: the rule of the lower third. Never, under any circumstances, inject the lower 2 cm of the frontalis muscle. This critical zone is directly responsible for brow elevation.

Violating this boundary is a direct invitation for ptosis. Your injection strategy must focus exclusively on the upper two-thirds of the frontalis. This allows for effective treatment of horizontal forehead rhytides while preserving the muscle's ability to maintain brow height. Think of it as a no-fly zone for your needle. Regarding technique, precision is paramount. For the frontalis, aim for 4 to 6 injection points, strategically placed across the upper two-thirds of the muscle. Each point should receive a conservative dose, typically 2 to 4 units, depending on the patient's muscle mass and the depth of their rhytides.

Always use a fine 30G needle, ensuring superficial intramuscular placement. The goal is a subtle, even relaxation, not paralysis. Consider the desired brow shape. Injecting more laterally than medially can induce a subtle lateral brow lift, often desired by patients seeking a more 'open' eye appearance.

However, be warned: overdoing this can lead to the infamous 'Spock brow' – an unnatural, exaggerated arch that is anything but aesthetic. Conversely, injecting symmetrically across the frontalis helps to maintain the patient's natural arch, which is often the safest and most predictable approach for a harmonious result.

When it comes to dosing, a nuanced approach is essential. For female patients, a total of 8 to 15 units is generally sufficient to achieve a smooth forehead without causing brow descent. Male patients, with their typically heavier muscle mass, will often require a higher dose, ranging from 15 to 25 units in total.

Always err on the side of caution; you can always add more at the review, but you cannot easily subtract. Finally, the 2-week review is not optional; it is an indispensable part of forehead botox treatment. Always schedule a follow-up at 14 days. The primary outcome to assess is, unequivocally, the brow position.

Look for symmetry, natural arch, and the absence of ptosis. Address any residual lines or asymmetries with touch-up injections, but remember, the brow's integrity is your ultimate measure of success. When addressing the

upper face with botulinum toxin, the glabella and crow's feet demand a meticulous approach, prioritizing precision over aggressive volume.

The glabella, that often-furrowed region between the eyebrows, is primarily influenced by two key muscles: the corrugator supercilii and the procerus. The corrugator muscles originate from the medial orbital rim and run obliquely upwards and laterally, pulling the eyebrows medially and inferiorly to create those characteristic vertical “11 lines.” The procerus, a smaller muscle, sits centrally at the root of the nose, pulling the nasal skin superiorly and contributing to horizontal glabellar lines.

Understanding this anatomical interplay is crucial for effective treatment. For the glabella, a standard 5-point injection pattern is typically employed to effectively relax these muscles. This involves a central injection into the procerus muscle at the midline, and two injection points per corrugator muscle, positioned bilaterally.

Each injection point usually receives a dose of 2-4 units of botulinum toxin, leading to a total dosage ranging from 10-20 units for the entire glabellar complex. The goal is a smooth, relaxed appearance without compromising natural eyebrow movement.

However, the glabella is not without its perils. The “danger zone” here is defined by the course of the supratrochlear artery, which emerges from the superior orbital rim and ascends into the glabella. To mitigate the risk of intravascular injection, which can lead to serious complications such as skin necrosis or even vision impairment, injections in this area must always be superficial and lateral.

Never inject deeply into the medial glabella. A superficial injection ensures the toxin is delivered into the muscle belly without impinging on deeper vascular structures. Moving laterally, crow's feet are caused by the contraction of the lateral orbicularis oculi muscle fibres. These fan-shaped wrinkles radiate from the outer canthus of the eye, becoming more prominent with smiling or squinting.

For effective treatment, inject 3 points per side in a fan pattern, staying strictly lateral to the orbital rim to avoid diffusion into the levator palpebrae superioris, which could cause ptosis. Each point typically receives 2-4 units, with a total of 6-12 units per side.

The aim is to soften the lines without creating an unnatural, frozen appearance or affecting the patient's natural smile. Finally, a cardinal principle

in botulinum toxin administration, particularly in these delicate areas, is the undercorrection principle. It is always better to undercorrect a patient and offer a top-up at a follow-up appointment, typically around two weeks post-injection, than to overcorrect.

An undercorrection allows for fine-tuning and ensures patient satisfaction, as additional units can easily be administered. Conversely, an overcorrection, which might lead to an undesirable aesthetic outcome or functional impairment, necessitates a waiting period of up to three months for the effects to naturally wear off, a situation that is far less manageable and can lead to patient dissatisfaction.

Precision and conservative dosing are the hallmarks of successful and safe aesthetic practice.



CHAPTER 34

The Bottom Third — Shape, Softening, Symmetry

Twice a year, a woman I will call Eugene arrives with masseters like river stones.

She is a trader, late twenties, British, and entirely undramatic about her jaw. She does not want a slimmer face. She has never once used the word contour. She grinds her teeth in her sleep and clenches through the day, and by the time she books in, the muscle is hard enough that you can see the outline of it from across the room and she has a headache most afternoons.

The appointment takes about ten minutes. The conversation takes considerably longer, because Eugene talks markets, and I have learned more about currency positioning from that chair than from any newspaper. She tells me what the desk is worried about. I nod as though I am going to act on it. Neither of us pretends this is why she is here, and neither of us minds.

What interests me clinically about Eugene is her timing. She does not come back when the toxin wears off. She comes back when the market does something unpleasant. Her treatment interval is set by her stress cycle, not by the product's duration, and if you plotted her appointments against her working year you would learn more about her life than about her masseter.

That is worth holding onto as you read this chapter. The lower face is where the body puts tension it cannot otherwise express. You will treat a great many masseters in your career, and a proportion of them will not be aesthetic cases at all — they will be people whose jaws are carrying something, and the injection is the smallest part of what you are being asked to do.

One practical warning before the anatomy. For several years the majority of my masseter work was for a patient group whose muscle bulk was consistently

greater than what I saw elsewhere in my list, and the doses that worked comfortably for one patient were frankly ineffective in the other. The lesson is not about any population. It is that you must dose the muscle in front of you, assessed by palpation on clench, rather than the number you were given on a course. A masseter you can grip between finger and thumb and a masseter that stands out like a river stone are not the same target, and treating them with the same units is how you end up with a patient who tells you the treatment did nothing.

The lower face is a warzone. It is where gravity, muscle tension, and bone loss all collide. You cannot treat it piecemeal. If you hit the masseter but ignore the chin, or fill the jawline but ignore the jowl, you lose the war. You have to think in triangles.

At the core of this framework lies The Lower Face Triangle. This foundational concept defines the overall shape and structure of the lower third of the face. Its three anchor points are the two mandibular angles and the chin. The lines connecting these points delineate the lower facial contour, and their strength and position are paramount to a youthful and aesthetically pleasing appearance.

A well-defined triangle, with clear angles and a proportionate chin, signifies vitality and structural integrity. Conversely, a weakened or poorly defined triangle often contributes to the perception of aging, characterised by a loss of jawline definition and the emergence of jowls. Understanding the masseter-chin-jawline relationship is critical, as these three elements are inextricably linked. A hypertrophied masseter muscle, often due to bruxism or genetic predisposition, can broaden the lower face, making the chin appear narrower and less prominent than it truly is. This imbalance can create a disproportionate facial shape.

Furthermore, a weak or recessed chin significantly exacerbates the appearance of jowls, as it fails to provide adequate anterior support for the overlying soft tissues. Treating these components in isolation is a common pitfall; a truly effective approach necessitates their simultaneous assessment and a coordinated treatment plan.

They are not solo acts but rather a symphony, where each instrument must play in harmony for the overall composition to succeed. Sequencing in the lower face is not merely a suggestion; it is a directive for optimal outcomes. When addressing the lower face, a precise order of operations minimizes complications and maximizes aesthetic improvement.

The first step, if indicated, is to address masseter hypertrophy with botulinum toxin. Reducing the bulk of an overactive masseter can subtly slim the lower face, allowing for better appreciation of the underlying bone structure and reducing the strain on the jawline. Following this, the mandibular angle should be augmented to restore definition and projection, re-establishing the posterior anchor points of the lower face triangle.

Next, the pre-jowl sulcus, a common area of volume loss, should be carefully filled to smooth the transition from the jawline to the chin. Finally, the chin itself is treated for projection and contour, completing the anterior anchor point and refining the overall V-shape or squareness depending on the patient's gender and aesthetic goals.

Deviating from this sequence often leads to suboptimal results or the need for corrective treatments. The feminisation vs. masculinisation axis dictates entirely different treatment goals and, consequently, different approaches. For the female lower face, the ideal is typically a narrow, pointed, and V-shaped contour, emphasizing delicacy and elegance.

The mandibular angles should be subtle, and the chin should project gracefully, contributing to a heart-shaped or oval facial silhouette. In contrast, the male lower face is ideally wide, square, and sharply defined. Strong, prominent mandibular angles and a broad, wellprojected chin are desirable, conveying strength and masculinity.

Treating a female patient with a masculinizing approach, or vice-versa, is a cardinal sin in aesthetic medicine, leading to unnatural and dissatisfying outcomes. The practitioner must always align their treatment strategy with the patient's inherent gender characteristics and desired aesthetic. Selecting the appropriate product is paramount, and the product rationale for the lower face hinges on understanding rheological properties, particularly G-prime.

For structural support and definition at the mandibular angle, a high G-prime filler is essential. This robust product provides the necessary lift and firmness to sculpt a sharp, defined angle that mimics bone. Similarly, for chin projection, a high G-prime filler is required to achieve significant anterior advancement and maintain its shape against dynamic forces.

However, for the more delicate task of softening the pre-jowl sulcus, a medium G-prime filler is more appropriate. This allows for a smoother integration with the surrounding tissues, avoiding a lumpy or overfilled appearance while still providing adequate volume replacement. Using the

wrong G- prime in the wrong area is a recipe for disaster, leading to either insufficient correction or an unnatural, palpable result.

Finally, let's address the common mistake: directly filling the jowl. This is akin to trying to fix a leaky roof by painting over the water stain; it addresses the symptom without touching the cause, and often makes the problem worse. The jowl is not an independent entity to be filled; it is a consequence of volume loss in the midface, laxity of the skin and ligaments, and a weakened lower face triangle.

Directly injecting filler into the jowl area invariably adds weight to an already sagging structure, making the lower face appear heavier, more prominent, and ultimately older. The correct approach is to lift the jowl by treating everything around it: restoring midface volume, strengthening the mandibular angle, and projecting the chin.

By providing structural support and tension to the surrounding tissues, the jowl is naturally attenuated and lifted, achieving a far more harmonious and youthful result without adding unnecessary bulk.



CHAPTER 35

The Toxin's Other Job — Medical Applications

A television doctor once asked me to inject a man's navel on camera.

He worked out of a practice below ours, we knew each other reasonably well, and the programme was one of those that invites the public to bring in the things they have been too mortified to show anybody. The patient had a persistently wet umbilicus. Sweating, presumably. Would I come and treat it?

I would like to claim that I declined on lofty clinical grounds. In truth my first reaction was straightforwardly personal: I did not want to do it, and the person feeling embarrassed in that arrangement was going to be me rather than the patient.

But the reason I actually said no, once I had thought about it for longer than a second, is the reason this chapter opens the way it does.

Nobody had established why the navel was wet.

A persistently damp umbilicus is not automatically hyperhidrosis. It can be a urachal remnant. It can be an umbilical sinus or fistula, occasionally communicating with bowel. It can be an infected pilonidal- type sinus, a chronically macerated deep umbilicus in a patient with a high BMI, or discharge that the patient has reasonably interpreted as sweat because sweat is the only explanation available to a layperson.

Several of those need imaging. One or two need a surgeon. None of them improve because you have paralysed the local eccrine glands, and one of them gets considerably worse, because you have now delayed a diagnosis in exchange for a temporary improvement in the symptom that was the only thing pointing at it.

Botulinum toxin is exceptionally good at abolishing a symptom. That is precisely what makes it dangerous outside its familiar territory. On the face you are treating an appearance and the diagnosis is usually self-evident. Everywhere else in this chapter, the symptom you are being asked to remove may be the only evidence anybody has.

Treat the diagnosis. If there is no diagnosis, that is the thing to obtain, and no amount of enthusiasm from a producer changes it.

What hyperhidrosis actually looks like in practice

Textbook teaching on hyperhidrosis concentrates on axillae and palms, because those are the licensed and best-studied indications. Real practice is broader and considerably less dignified, and the patients who arrive at your door have usually exhausted everything else first.

I have treated the inframammary fold in women whose skin beneath the breast was permanently macerated and repeatedly infected, and for whom no amount of antiperspirant or careful drying had ever worked. The area is straightforward to map with a starch-iodine test and responds well, though the fold itself needs care: you are injecting intradermally over a curved, mobile surface, and it is easy to drift too deep or too lateral into pectoral fibres.

I have treated a model with severe inguinal hyperhidrosis. Her clothing was visibly patched by mid-morning, she had rearranged what she wore and what work she accepted around it, and she described the whole of her social life in terms of managing it. She had been told by more than one doctor that it was not a medical problem.

It was consuming her life, which is as medical as a problem gets.

And I treated scrotal hyperhidrosis some years before the media discovered the subject and gave it a nickname. That case is worth a paragraph on consent, because it is the model I still use for anything off-label in a sensitive region. There was no meaningful literature on fertility outcomes.

I could not tell the patient the risk was zero, because nobody knew. So I told him exactly that — that the evidence did not exist, that on mechanistic grounds significant systemic effect was not expected, and that I could not promise more than that — and consented specifically for the theoretical possibility of an effect on fertility.

He was in his thirties and had not completed his family. He chose to proceed, understanding what was and was not known.

That is the standard. Not “it is probably fine.” Not a raised eyebrow and a signature. You say out loud where the evidence ends, you write down that you said it, and you let an adult make an adult decision.

Facial blushing

Blushing belongs in the same chapter because it is the same idea running in reverse: instead of interrupting cholinergic signalling to a sweat gland, you are attenuating the cutaneous vascular flush, and in practice you are treating the same population — young, often high-achieving, socially constrained, and frequently in a state of considerable distress by the time they find somebody willing to help.

I want to be careful here, because facial blushing is the treatment in this book that has caused me the most trouble, and the story of how is in Chapter 43.

Most practitioners learn botulinum toxin through the cosmetic lens. Frown lines. Forehead. Crow's feet. That is the beginning. Not the whole picture. Botulinum toxin is one of the most versatile therapeutic tools in medicine. Its mechanism — temporary, reversible, dose-dependent inhibition of acetylcholine release at the neuromuscular junction — applies to any condition driven by overactive muscle contraction, overactive glandular secretion, or chronic pain from muscular hypertonicity.

The aesthetic practitioner who understands this is not just a cosmetic injector. They are a clinician with a remarkably broad toolkit. What follows are five applications that sit at the intersection of aesthetics and medicine. Each one has a strong evidence base, a clear clinical rationale, and a patient population that is actively looking for treatment. Migraine Prevention — The PREEMPT Protocol Aesthetic doctors discovered the migraine application by accident.

In the late 1990s, patients treated for glabellar lines were coming back to their review appointments and mentioning, almost as an aside, that their headaches had improved. Not a little.

Significantly. Some patients who had suffered chronic migraines for years reported that the frequency had dropped by half. Nobody had planned this. It was a side effect of treating a cosmetic concern, and it was too consistent to

ignore. In 2000, Binder published the first open-label study documenting migraine benefit from botulinum toxin.

Allergan launched a clinical programme that produced the PREEMPT trials — the largest randomised controlled trial series ever conducted in chronic migraine prevention. The PREEMPT Protocol The licensed protocol looks nothing like a cosmetic treatment plan. Total dose Injection sites Cosmetic Approach 20–50 units 5–11 Muscle groups Frontalis, corrugator, procerus Frequency Indication Every 3–6 months Cosmetic PREEMPT Protocol 155–195 units 31–39 7 groups: frontalis, corrugator, procerus, temporalis, occipitalis, cervical paraspinal, trapezius Every 12 weeks Chronic migraine only The licensing is specific. OnabotulinumtoxinA is licensed for chronic migraine only — defined as fifteen or more headache days per month for more than three months, with at least eight of those days having migraine features. It is not licensed for episodic migraine. The neurologist treating chronic migraine is not being excessive with 155–195 units.

They are following the only protocol with robust RCT evidence. The seven muscle groups — including the back of the head, the neck, and the trapezius — are there because that is where the evidence pointed. The aesthetic practitioner who sees migraine improvement after a cosmetic frown treatment is observing a real phenomenon.

But they are observing it in a different population, at a different dose, at a different set of sites. These are not the same thing. The Clinical Takeaway If a patient mentions migraine improvement after a cosmetic treatment, document it and refer them to a neurologist if their headache burden is significant. Do not expand your treatment plan to mimic PREEMPT without appropriate training, licensing, and clinical governance. What you can say, honestly, is this: the discovery that botulinum toxin has any effect on migraine at all came from aesthetic medicine.

Aesthetics asked the question. Neurology answered it. Scan the QR code for the full HSI Journal clinical review of the PREEMPT protocol. <https://www.harleystreetinstitute.com/journal/botox-migraine-preventionpreempt-protocol-review> Masseter Botox — Bruxism, TMJ, and Facial Slimming The masseter is one of the strongest muscles in the human body relative to its size. It is also one of the most commonly undertreated sources of facial pain, headache, and jaw dysfunction in the adult population. Bruxism — the involuntary grinding and clenching of teeth — affects an

estimated eight to thirty percent of adults, with the majority undiagnosed.

The masseter hypertrophies in response to chronic overuse, producing a squared, heavy lower face and a cycle of muscular tension that drives headaches, jaw pain, tooth wear, and disturbed sleep. The Dual Indication Masseter botulinum toxin serves two purposes simultaneously, which is clinically elegant. The first is therapeutic: reducing the force of involuntary contraction relieves the muscular tension driving bruxism-related pain, protects the dentition from further wear, and often significantly reduces headache frequency in patients whose headaches originate from jaw tension rather than migraine.

The second is aesthetic: a hypertrophied masseter creates a square, masculine lower face. As the muscle reduces in bulk over three to six months, the lower face narrows and softens, creating a more oval facial contour. In East Asian patients in particular, this is one of the most requested aesthetic outcomes.

Mapping the Muscle — The Earlobe-to-Lip Line Before you inject anything, you need to know exactly where you are. Draw a line from the corner of the earlobe to the corner of the lip. This line defines the inferior border of your safe injection zone.

Everything below this line is too low — you are approaching the depressor anguli oris and the marginal mandibular nerve territory.

Now palpate the masseter actively while the patient clenches. The muscle belly will harden and become clearly palpable. Mark the anterior border and the posterior border of the muscle as it contracts. You will notice the muscle runs superomedially — it is not a vertical structure.

The injection box sits within these borders, above the earlobe-to-lip line, and below the zygomatic arch. Within that box, mark your injection points approximately one centimetre apart along the greatest bulk of the muscle. This is typically three to four points in a gentle arc following the muscle's contour.

The Posterior Rule — Why You Do Not Inject the Bulge Directly This is the detail that separates a safe masseter injection from an unsafe one. The fascia of the masseter muscle overlaps with the fascia of the risorius — the muscle responsible for the lateral pull of the smile.

They share a fascial plane at the anterior border of the masseter. A superficial injection at the point of greatest muscle bulk can diffuse anteriorly onto the risorius, weakening the smile. The solution is simple: inject slightly posterior to the point of greatest bulge. Half a centimetre posterior is sufficient.

This keeps you within the safe posterior zone of the masseter and away from the risorius fascial overlap. Depth — Full Needle Depth The injection must be deep. Use a 30-gauge yellow needle.

Insert to three-quarters of the needle length — approximately five to seven millimetres. You are aiming to be close to the periosteum of the mandible. At this depth, you are in the deep muscle belly, well away from the superficial fascial planes where diffusion causes problems.

A practical technique: insert the needle and then push slightly so the skin tents downward. This confirms you are in the muscle belly and not superficial to it. At this depth, the injection is intramuscular and the product stays where you put it. The Temporalis — When the Masseter Is Not Enough Some patients clench primarily with the temporalis rather than the masseter.

Others have both muscles involved. As part of every bruxism assessment, palpate both the masseter and the temporalis while the patient clenches. Feel which side is more hypertrophied.

Feel which muscle is tighter. Ask the patient which side they are aware of grinding on — grinding is typically unilateral, and the dominant side will be visibly and palpably larger. If masseter treatment alone does not resolve the patient's symptoms, add temporalis injections. Two to three injection points of three to five units each, placed into the temporal fossa along the anterior and middle fibres of the temporalis, will address temporal clenching effectively. Before injecting the temporalis, always palpate the temporal artery. It runs through the temporal region and must be identified and avoided. It is palpable as a pulsatile cord running vertically through the temporal fossa.

Mark it and stay clear. The T-Shirt Sizing System Assess the masseter like a T-shirt. Small, medium, large, or extra large. A small masseter is barely palpable on clenching — a modest thickening of the lower face. A medium masseter is clearly palpable but not visually prominent.

A large masseter is visually apparent at rest, with a noticeable squaring of the lower face. An extra-large masseter is unmistakable — when viewed from the front, the muscle angles outward, widening the lower face significantly beyond the width of the cheekbones. The size determines the dose and the treatment interval.

Dosing — Female Patients Muscle Size Small Medium Large Extra Large
Starting Dose (per side) 15 units 20 units 25 units 30 units
Dosing — Male Patients Add 5–10 units per side to the female dose at each size category. Male

masseters are consistently larger and more resistant to treatment. The Treatment Interval Schedule The interval between treatments is not fixed. It follows the muscle.

An extra-large masseter at first treatment will typically reduce to large by the next appointment. Large reduces to medium. Medium reduces to small. As the muscle reduces in size, the interval between treatments lengthens. Starting Size Extra Large Large Recommended Return Interval 3 months 4 months Medium Small 5 months 6 months This graduated schedule means that patients who start with a severely hypertrophied masseter will need more frequent early treatments, but as the muscle reduces, they naturally transition to a maintenance schedule of every five to six months. Set this expectation at the first consultation. It avoids the patient feeling like they are being overtreated.

Key Safety Points The parotid gland sits anterior and superior to the masseter. Injecting too high or too anterior risks parotid diffusion, which can cause temporary facial weakness or altered salivation. Stay in the lower two-thirds.

Stay posterior. Palpate the muscle before every injection. The smile muscles — zygomaticus major and risorius — run superficially over the anterior masseter. Injecting too superficially or too anteriorly risks weakening the smile. The injection must be deep, into the muscle belly, not superficial. What to Tell Patients Results are not immediate.

The muscle reduces gradually over three to six months. Pain relief from bruxism often begins earlier — within two to four weeks — as the resting muscle tension reduces even before significant atrophy occurs. Set the expectation clearly: the aesthetic slimming effect takes time. The pain relief comes first.

For a full hands-on practical course on masseter Botox including live patient demonstrations, scan the QR code below.[QR Code — HSI Masseter Botox Masterclass]<https://www.harleystreetinstitute.com/masseter-botox-course/> Hyperhidrosis — Axillary, Palmar, and Plantar Hyperhidrosis is the medical term for excessive sweating beyond what is physiologically necessary for thermoregulation. It affects approximately two to three percent of the population. The majority never seek treatment because they do not know effective treatment exists. This is an opportunity to change a life.

Before treating any new client, ensure they meet the diagnostic criteria for primary focal hyperhidrosis. The onset is typically under the age of 25 (often in

adolescence), it is bilateral and relatively symmetrical, it impairs daily activities, it occurs at least once a week, there is a family history in many cases, and crucially — sweating stops during sleep.

If a patient is sweating profusely at night, or if the onset is sudden in later life, you must exclude secondary causes like thyroid dysfunction, infection, or malignancy before proceeding. We document severity using the Hyperhidrosis Disease Severity Scale (HDSS), a simple 1-to-4 scale that helps you understand how significantly the condition impacts their life, and provides a baseline to measure your treatment success against. Axillary Hyperhidrosis — The Central Concentration Technique The standard teaching often recommends the iodine-starch test to map the sweating area. I do not use it. It is messy, and if an area highlights less initially, give it another five minutes and the anxiety will trigger it, turning that area blue too.

Given the doses we use, we can easily cover the necessary area without needing to map and potentially undertreat less active zones. Instead, map a boundary around the hair-bearing patch. This forms a visible triangle where the hair grows. Divide this area horizontally: the centre, a top quarter, and a bottom quarter.

The central region contains the highest density of sweat glands. Therefore, half of your total dose goes into the centre. A quarter goes to the top section, and a quarter goes to the bottom section. For dosing, I use 50 units of Botox (or equivalent, such as 150 units of Dysport) per axilla for females.

For males, who typically have a larger surface area and higher gland density, I increase this up to 75 units per axilla. Injection depth is critical. It must be very skin deep — intradermal. Too superficial, and it stings significantly more. You want a nice little bleb, similar to the depth you use for crow's feet or standard forehead injections.

Place your injection points one centimetre apart. You can mark dots to guide you. If you have extra dose left for a specific section (centre, top, or bottom), come back and reinject those dots to double the dose in that location rather than spreading it too thin elsewhere.

Palmar Hyperhidrosis — Managing the Hands Palmar hyperhidrosis is technically demanding. In my experience, you will see the hands start sweating the moment you do the first couple of injections — the anxiety triggers it immediately, showing you exactly where the active zones are. The dose is typically 50 to 60 units per hand.

Let us assume 60 units for a standard treatment. Divide the dose strategically. I allocate 30 units for the fingers. In each digit, I place one injection of 5 units. Along the hypothenar eminence (the fleshy edge below the little finger), I halve the dose per point.

I do not give 5 units; I give 2.5 units per point. I will place four or five points along the hypothenar, one or two over the thenar muscles (the thumb base), and some in the central palm. Turn the hand around and inject in between the web spaces.

For the dominant hand — the shaking and writing hand — I try to give a bit more to the ulnar edge of the fingers as well. The Functional Trade-off in Palmar Treatment You must warn patients about temporary functional weakness. It is unavoidable if you are treating the palms effectively.

The web space injections will decrease finger adduction power. The thenar injections will decrease power for fine motor movements requiring thumb opposition — specifically, actions like buttoning a shirt or picking up a coin. Almost 100 percent of patients will notice and mention this weakness. However, in my practice, no one has ever asked me to decrease the dose for their next treatment because of it. The relief from the sweating far outweighs the temporary clumsiness.

The weakness lasts two to four weeks. I have treated pianists, guitarists, and people in highly manual jobs, and they do not complain about it once they understand it is temporary. For patients who lift weights at the gym, reassure them that this treatment does not significantly affect their lifting capacity, because the primary muscles required for heavy lifting are located in the forearm, not the intrinsic muscles of the hand. Plantar Hyperhidrosis Plantar hyperhidrosis follows similar principles to palmar treatment. It is less commonly requested but highly effective. The plantar surface is extremely sensitive, and regional anaesthesia or nerve blocks are strongly recommended before attempting treatment here.

Trigger Point Injections — Chronic Myofascial Pain Trigger points are discrete, hyperirritable spots within a taut band of skeletal muscle that produce local and referred pain on compression. They are extraordinarily common. Chronic neck pain, shoulder tension, tension-type headache, and upper back pain are frequently driven by active trigger points in the trapezius, sternocleidomastoid, levator scapulae, and suboccipital muscles.

Conventional trigger point injections use local anaesthetic, saline, or dry needling. Botulinum toxin offers a longer-lasting alternative — the mechanism is the same (disrupting the abnormal acetylcholine release that sustains the taut band), but the duration of effect is weeks to months rather than hours to days.

Technique Identify the trigger point by palpation — the taut band is palpable as a cord-like structure within the muscle, and direct pressure on the trigger point reproduces the patient's familiar pain pattern (the “jump sign”). This reproduction of familiar pain is the key diagnostic criterion.

If you cannot reproduce the pain, you have not found the trigger point. Inject 5–10 units directly into the trigger point. For multiple trigger points in the same muscle group, distribute the dose across all active points. The trapezius commonly has three to five active trigger points in chronic sufferers.

Total doses per session vary widely depending on the number of sites and the muscles involved. For upper trapezius and cervical paraspinal trigger points, 50–100 units per session is typical.

Important Distinction Trigger point botulinum toxin is not the same as the PREEMPT protocol. PREEMPT targets specific anatomical sites regardless of trigger point status. Trigger point injections are guided entirely by palpation and symptom reproduction. The two approaches can be combined but should not be confused.

Trapezius Tox and the Barbie Neck The trapezius tox — colloquially known as “Barbie Botox” or “trap tox” — has become one of the most requested treatments in the past two years. Aesthetically, it elongates the neck and reduces the bulky, masculine shoulder silhouette caused by hypertrophied upper trapezius muscles. Therapeutically, it relieves chronic neck and shoulder tension. The technique requires mapping the upper trapezius belly. Have the patient shrug against resistance. Mark the anterior and posterior borders of the upper trapezius. The safe injection zone is the muscular bulk between the neck and the acromion. Do not inject too medially (risk to cervical paraspinal muscles and neck weakness) or too anteriorly (risk to the apex of the lung).

Standard dosing is 40 to 50 units per side, distributed across four to five points in the thickest part of the muscle. **The CNS Pain Flare — Why Patients Get Worse Before They Get Better** There is a critical neurophysiological phenomenon you must warn your patients about when treating chronic trapezius tension: the temporary pain flare.

If you do not warn them, they will think you have injured them. When a patient has chronic muscle tension — months or years of the trapezius being locked in a state of rigid contraction — the central nervous system (CNS) adapts. The constant bombardment of nociceptive signals from the tense muscle into the spinal cord dorsal horn causes a phenomenon known as “wind-up.” The NMDA receptors in the spinal cord become hypersensitive.

The brain and spinal cord literally recalibrate their baseline to accommodate this chronic pain state. This is called central sensitization. The CNS essentially turns down its own volume dial so the patient can function despite the constant muscular tension. When you inject botulinum toxin, you suddenly paralyze the muscle.

The chronic tension is abruptly removed. The peripheral nociceptive input drops to zero. But the central nervous system is still calibrated for chronic pain.

With the heavy muscular tension gone, the CNS suddenly loses the structural “splinting” it had adapted to. The descending pain modulation pathways are temporarily thrown out of balance. Because the spinal cord is still in a state of central sensitization, normal sensory inputs are suddenly perceived as painful.

The brain, confused by the sudden silence from the trapezius, amplifies surrounding signals. The clinical result? Two to three weeks after the injection, the patient will often experience a flare of pain. Their neck might ache more.

Their shoulders might feel heavy, tired, or deeply sore. This is not a complication. This is the CNS recalibrating. You must explain this physiology during the consultation. Tell them: “Your brain is used to your muscles being tight. When we relax them, your nervous system has to adjust to the new normal.

About two to three weeks from now, you may actually feel more ache or fatigue in your shoulders. This is temporary. By week four or five, the brain figures it out, the pain flare subsides, and you will feel the true relief.” When you set this expectation, the patient feels informed and secure.

When the pain flare hits at week three, they think, “Ah, my CNS is recalibrating, just like the doctor said.” If you fail to warn them, they will leave a one-star review claiming you ruined their neck. Dosing Summary for Medical Botulinum Toxin Applications Indication Chronic migraine (PREEMPT) Masseter (bruxism/slimming) Axillary hyperhidrosis Palmar hyperhidrosis Trigger points Upper trapezius (trap tox)

Indication	Typical Dose	Sites	Frequency
Chronic migraine (PREEMPT)	155–195 units	31–39	Every 12 weeks
Masseter (bruxism/slimming)	20–40 units per side	3–4 per side	Every 4–6 months
Axillary hyperhidrosis	50 units per axilla	10–15 per axilla	Every 6–12 months
Palmar hyperhidrosis	100–150 units per palm	30–40 per palm	Every 6–9 months
Trigger points	5–10 units per point	Variable	Every 3–4 months
Upper trapezius (trap tox)	50–75 units per side	4–6 per side	Every 3–4 months

The practitioner who masters these applications is not just an aesthetic injector. They are a clinician who happens to work in aesthetics. That distinction matters. It changes how patients perceive you, how you perceive yourself, and ultimately, what kind of practice you build.

The face is where most of us start. It is not where the story ends. When addressing bruxism, it's crucial to understand the distinct yet complementary roles of the aesthetic injector and the dentist. As injectors, our focus with botulinum toxin is primarily on mitigating the excessive force exerted by the masseter muscle, thereby reducing the intensity of grinding and clenching.

The dentist, on the other hand, is concerned with the dental sequelae of bruxism—the wear and tear on the teeth themselves, and the structural integrity of the oral cavity. We are not in competition; rather, we are two sides of the same coin, working in concert to provide comprehensive patient care.

It is imperative to recognise the signs that necessitate a dental referral. During your consultation, pay close attention to indicators such as visibly worn tooth surfaces, evidence of cracked teeth or restorations, patient complaints of jaw pain upon waking, persistent headaches, or heightened tooth sensitivity.

These are red flags that suggest significant dental damage or ongoing issues that fall squarely within the dentist's purview. The referral conversation requires a delicate touch. The goal is to inform, not to alarm. Frame it as a proactive measure to ensure the longevity of their dental health.

You might say, “While the Botox will help reduce the muscle activity causing your grinding, I’ve noticed some signs that suggest your teeth could benefit from a dental assessment. A dentist can check for any wear or damage and ensure your bite is optimally protected.” This positions the referral as an enhancement to their overall treatment plan, rather than a cause for concern.

Patients often inquire about night guards, and it’s important to clarify their role. A night guard, typically provided by a dentist, acts as a physical barrier to protect the teeth from the forces of grinding. It does not, however, reduce the muscle’s ability to clench.

Therefore, the use of a night guard does not diminish the efficacy of botulinum toxin; they operate via different mechanisms. The botox addresses the source of the force, while the night guard manages the impact of any residual force. Indeed, the combined approach of botulinum toxin and a night guard often yields the most superior outcomes.

Botox effectively reduces the sheer force of grinding and clenching, alleviating muscle hypertrophy and associated pain. The night guard then steps in to protect the teeth from any remaining, albeit reduced, occlusal stress. Together, they offer a synergistic benefit that is far more effective than either treatment modality employed in isolation, providing both symptomatic relief and structural protection.

When referring a patient, a brief, professional letter to the dentist can facilitate seamless communication and ensure the patient receives appropriate follow-up care. A template such as the following can be highly effective: Plain Text To Whom It May Concern, This patient, [Patient Name], is receiving botulinum toxin to the masseter for b Sincerely, Bruxism — When to Refer to the Dentist Let’s get one thing clear: you are an aesthetic injector, not a dentist. When a patient comes to you with masseter hypertrophy and bruxism, your botox treats the muscle. It stops the excessive force.

But it does not fix the damage already done to the teeth. The Signs Look inside their mouth. Are the tooth surfaces worn flat? Are there cracked teeth?

Do they complain of jaw pain on waking, chronic headaches, or severe tooth sensitivity? If yes, you need to refer them. The Conversation Don’t alarm them. Just be a professional.

“The botox will relax the muscle and stop the grinding force, but I can see some wear on your teeth. I want you to see your dentist to protect the enamel.” The Combined Approach The dentist will likely provide a night guard. Some

injectors think a night guard makes botox redundant. Nonsense. They are complementary. The botox reduces the force; the night guard protects the teeth from whatever force remains. Together, they are a fortress. Write a brief letter to the dentist: “This patient is receiving botulinum toxin to the masseter for bruxism. I would be grateful if you could assess for dental sequelae and consider a night guard if appropriate.” It shows you know what you are doing, and it builds your referral network.

PART V

The Patient

“They tell me they look in the mirror and do not recognise the person standing there. They expected someone else.”

Why Now? — The Three Questions

By the third year I was seeing thirty to forty patients a day.

The website was at the top of Google for hundreds of keywords. At the aesthetic conferences, the IT people had started using it as a case study — this is what a clinic website ought to look like — which meant they were, in effect, selling my competitors a copy of me. I minded less than I should have.

I was good at the work. I want to be accurate about this rather than modest: I could build a lip in a few minutes that other injectors would spend an hour on and still not get right. Chin, jaw, the whole architecture of a face. It came easily. It had come easily almost from the start.

And I was not happy.

I could not have told you why. Nothing was wrong. The diary was full, the results were good, the patients came back. But I did not relate to what I was doing. It felt like a trade I had wandered into rather than a thing I believed in, and I could not shake the sense that the whole industry was slightly absurd and that I was now part of it.

So I went to see my mother and said more or less that. I'm very good at this. I don't think I want to do it.

She thought about it for a moment and said that if she were young again, she would have her lips done.

I said, what?

She told me she had spent her whole life overlining her lips. That she had always been conscious of them. That it had simply never occurred to her that there was anything that could be done about it.

My mother. Decades of it. She had never mentioned it once — not to me, and as far as I know not to anyone. And I had been injecting lips for years, thirty and

forty a day, in the same city, and had never thought to ask.

That is the moment the second half of my career started.

It had not previously occurred to me that everybody walking into my clinic was carrying something like that. I had been treating faces. I had never asked why anybody wanted their face treated, because the answer seemed obvious, and the answer was never obvious.

So I started asking. Three questions, and I still ask them in the same order.

What do you want done. Why do you want it done. And why now.

The first question they have rehearsed. The second one slows them down. The third one is the one that tells you everything, because nobody has an accident of timing — something has happened, and it happened recently.

What came back, over years, was almost never about the face.

A divorce, six months old, sometimes six weeks. Children who had grown up and left, and a woman who had not looked at herself properly since they were small. A parent in decline, being cared for by a daughter who had stopped being cared for by anybody. Stress with no end date attached to it. And a very large number of people who simply wanted to give themselves something back, and did not have a vocabulary for that, so they booked in for lips.

The sentence I have heard more than any other, in nearly twenty years, in some form or another, is this: I look in the mirror and I don't recognise the person there. I expected someone else.

That is not vanity. I want to be extremely clear about that, because the entire public conversation about this industry is built on the assumption that it is. Vanity is wanting to look better than other people. This is something else. This is the gap between the face you carry internally and the one that appears in the glass, and it widens by a millimetre a year until one day it is wide enough to make somebody pick up the phone.

Our job is not to close that gap by making people look twenty-five. It is to make the reflection legible again.

Some years ago I started asking a second question, and I ask it mostly of older patients — not the ones in their twenties, who have not yet accumulated the gap.

When you dream about yourself, how old are you?

Nobody has ever had to think about it. They smile — every single one of them smiles, which is the part I did not expect — and they tell me an age. It is always their prime. Thirty-two. Twenty-eight. Forty, the year everything was going well. Not one person in nearly twenty years has answered with the age they actually

are.

So there is a version of each of us that the mind holds on to and refuses to update. It is the one that appears in dreams. It is the one that is quietly expected in the mirror every morning. And every year the reflection drifts a little further from it, without permission and without announcement, until the distance becomes large enough to make somebody pick up a telephone and book an appointment with a stranger.

That is what we are actually treating. Not a fold, and not a line. The distance between the dreamed self and the reflected one.

Everything in this part of the book follows from that.

The three questions as clinical method

Chapter 13 covers the mechanics of a consultation: the structure, the examination, the documentation. This chapter is about the part that determines the outcome, and it takes about ninety seconds.

What do you want done.

They have rehearsed this. It will be delivered fluently, often with a gesture towards the area, and it is frequently borrowed language — a phrase from a screen rather than from their own head. Take it at face value and write it down verbatim. The exact words matter later, because “I want my lips done” and “I want my lips to look like they used to” are two different requests, and only one of them can be satisfied.

Why do you want it done.

This is where the fluency stops, and the pause is the information. Most patients have never been asked. Some will answer immediately and superficially — because it bothers me, because I look tired — and that is fine; not everything has a depth to it. Others will stop, look slightly startled, and start again in different language. When somebody restarts their sentence, listen to the second version.

The answers cluster. They look tired and are tired. They have been told they look angry when they are not. Something in a photograph. Somebody said something. A comparison with a sibling, a colleague, a former version of themselves.

And why now.

This is the question that earns its place, and I ask it of everybody.

Nobody arrives by accident of timing. The line has been there for four years; the appointment was made on Tuesday. Something happened between the line appearing and the phone being picked up, and that something is the actual presenting complaint.

What the answers predict

Over nearly twenty years, the answers to *why now* fall into a small number of groups, and each carries a different prognosis.

A dated event — a wedding, a reunion, a photograph, a birthday.

These are the easiest patients in medicine. The motivation is external, bounded, and honest. The only clinical issue is timing: never treat close to the event, always leave room for a review and a correction, and be explicit that you will not be rushed into a same-week result.

If the date is too near, decline and offer them the next one.

A transition — divorce, separation, children leaving, a new job, the end of a long period of caring for somebody. These are the majority, and they are usually good patients, but they need a slower approach. Somebody six weeks out of a marriage is not in a settled relationship with their own face.

Do less than they ask. Stage it. Book the review before they leave. Almost nobody in this group regrets a conservative result, and a meaningful number regret a dramatic one.

Exhaustion — the caregiver, the person under prolonged stress, the patient who says they simply want to feel like themselves again.

This group is easy to under-serve, because their complaint is diffuse and they will accept whatever you suggest. They often benefit more from skin work and a genuine consultation than from volume, and the risk is that you treat a face when what they came in with was a life. **A comment.** Somebody said something. A partner, a colleague, a stranger, occasionally a child. Ask who. If the answer is a partner, and the tone shifts when they say it, slow down considerably — you may be looking at a request that did not originate with the patient at all, and treating on somebody else's behalf is a consent problem before it is anything else.

Nothing. The patient cannot identify anything that changed. Sometimes this is simply true and unremarkable. Occasionally it means the preoccupation has been continuous and long-standing rather than triggered, which is one of the signals discussed in Chapter 39.

When why-now is a contraindication

Three answers should stop you, at least for that appointment.

To save a relationship. That is Chapter 37.

Because somebody else wants me to. The patient in front of you must be the one who wants this. If they are not, you do not have consent in any meaningful sense, regardless of what has been signed.

Because I cannot go out / cannot work / cannot be photographed. Functional impairment is a different order of complaint. Take it seriously and take it slowly, because you may be looking at something that a syringe will not touch.

Write down the motivation

Documentation in aesthetic medicine concentrates almost entirely on what was injected. Record what they wanted and why, in their words.

It takes one line and it does three things. It gives you a baseline you can return to at review — *you came in because you were told you looked angry; do you still get that?* — which is a far better outcome measure than a photograph.

It protects you, because a patient's account of what they asked for can drift considerably over eighteen months. And it makes you ask the question, which is the real point, because the temptation on a busy afternoon is to skip straight from the request to the needle.

When the correct treatment is none

The most useful consequence of asking *why now* is that it occasionally produces an answer that ends the consultation.

There is a particular kind of appointment where somebody books for something small, sits down, and talks for forty minutes. The stated request is trivial and they know it is trivial. What they wanted was an hour with somebody who was looking at them attentively and had no stake in the conversation.

You can treat that person. The treatment will be technically fine and it will not touch what brought them. Or you can finish the conversation, do very little or nothing at all, charge them for the consultation or not, and book them in for three months' time.

I have done that a reasonable number of times and I have never once regretted it. Almost all of those patients came back — some for treatment, some not — and several of them have been with me for over a decade.

The rest of this Part is about the patients for whom the answer to *why now* is more complicated than it first appears.



CHAPTER 37

The Procedure That Is Meant to Fix Something Else

Some patients are not buying a treatment. They are buying an outcome that the treatment cannot deliver, and they have not said so out loud, possibly not even to themselves.

This is the most common serious error in aesthetic medicine and it has nothing to do with technique. You can perform a flawless procedure — correct plane, correct volume, symmetrical, safe, beautiful — and produce a profoundly unhappy patient, because the result was never the point.

How it presents

It does not announce itself. Nobody sits down and says they would like their nasolabial folds treated in order to stop their husband leaving. What you get instead are signals, and they are consistent enough to be worth listing.

The complaint is disproportionate to the finding. You are looking at something minor and they are describing something catastrophic. That mismatch is the single most reliable indicator that the face is standing in for something else.

The expected outcome is described in terms of other people. Listen for the grammar. *I want to look less tired* is about them. *I want him to notice* / *I want them to take me seriously* / *I want to be able to walk into that room* is about an audience, and audiences do not respond predictably to two millilitres.

There is an unrelated crisis in the room. They mention the separation, the redundancy, the rejection — usually in passing, as context rather than as reason. It is the reason.

The timeline is urgent and the deadline belongs to somebody else. He is back in a fortnight. The interview is on Thursday. The reunion is Saturday and *she* will be there.

They ask what you would do. Not out of humility — out of a wish to hand over responsibility for a decision they cannot make, because the actual decision is not about their face.

Why it matters clinically

Because you cannot satisfy this patient, and the attempt tends to escalate.

The treatment is done, it looks good, and the marriage is still ending. The obvious inference — from inside that patient's experience — is that it was not enough. So they come back and ask for more. If you supply it, you are now a participant in something that has stopped being medicine, and the endpoint of that road is a face that has been progressively altered in pursuit of an outcome that was never available. These are also, disproportionately, the patients who become dissatisfied in ways that are difficult to resolve, because the dissatisfaction is real and cannot be addressed by anything you have in the fridge.

What to actually say

You are not required to make a psychiatric assessment, and you should not attempt one. What you are required to do is be honest about the limits of what you sell, which is a much smaller and more achievable task.

The most valuable sentence in the consultation is some version of this:

I can make this better. I want to be clear that it will not do the other thing you are hoping for.

Then stop talking. The silence after that sentence does more work than anything else in the appointment.

What generally happens is one of three things. Some patients say *no, I know, I just want it done* — and mean it, and are fine, and you proceed with a conservative plan. Some patients look at you and the whole conversation changes, because nobody has named the thing yet and they are relieved rather than offended. And a small number get annoyed, which is itself informative.

A few practical notes on delivery:

Do not diagnose the underlying situation. You are a doctor treating a face, not their counsellor, and speculating about their marriage is both presumptuous and clinically pointless. Name the limit of the treatment, not the nature of their life.

Never say “this won’t fix your relationship.” Say what your treatment does and does not do. The distinction sounds pedantic and is not: one is a comment on the product, the other is a comment on them.

Offer time instead of refusal. *Let’s do something small now, and look again in three months* is almost always the right answer. It is not a no, it prevents the irreversible, and three months is long enough for the crisis to resolve itself in one direction or another.

Write down the expectation. In their words, in the notes, alongside the fact that you addressed it.

The version that is fine

I want to guard against over-reading this, because the profession’s instinct when it learns about psychological complexity is to become suspicious of everybody.

A great many patients come in around a life event and they are perfectly well. A woman who has come out of a long marriage and wants to look like herself again is not deluded; she is doing something sensible and slightly brave, and treating her carefully is a genuine kindness.

The distinction is not whether there is a life event. It is whether the treatment is expected to *resolve* the life event.

I am starting again and I want to feel good is a patient. If I fix this, he will stay is a warning.

The first one you treat, gently and with a review booked. The second one you talk to, and then you decide.

CHAPTER 38

The Man Who Will Not Say Why He Is Here

Male patients arrive with a cover story, and the cover story is almost never the reason.

The literature on this is thin to the point of absence. There are papers on male anatomy, male dosing, the differences in muscle mass and skin thickness, and how to avoid feminising a face. There is almost nothing on the consultation itself, which is a shame, because with male patients the consultation is where the whole thing is won or lost.

The pretext

He will give you a reason, and it will be external, practical, and slightly deniable.

My wife booked it. There was a photograph at a work event and I looked awful. Someone at the office had it done and said I should. I'm not bothered really, but since I'm here.

Every one of those is a way of arriving without having admitted to wanting to arrive. The distinction matters, because a man who has framed his attendance as somebody else's idea has also built himself an exit: if the conversation becomes uncomfortable, none of it was his in the first place.

This is not evasiveness for its own sake. It is a vocabulary problem. Most men have simply never been given language for talking about how they look that does not sound either vain or unwell, and a great many of them are genuinely embarrassed to be sitting in your chair at all.

The room is often designed for somebody else, the magazines are for somebody else, and the practitioner asking them how they feel about their

appearance is asking a question they have spent forty years not answering.

Conduct the consultation sideways

The technique that works is not the one from the previous two chapters. *Why now* asked directly of a man in this state produces a shrug.

Start with function, not feeling. *Are you sleeping? How's work? Are people telling you you look tired, or is it you that thinks it?* Function is a permitted topic. Feeling is not, yet.

Let the mirror do the asking. Hand him a mirror and ask him to show you what he means, rather than asking him to describe it. Men who cannot say *I feel old* will point at a frown line without difficulty. Pointing is not admitting.

Use the third person. *Most men who come in about this say it's the frown — they get told they look angry when they're not.* This gives him a category to belong to, which is far easier to accept than an individual confession. A surprising number of men will then supply the real reason unprompted, because you have established that it is a normal thing to have.

Do not push for the underlying story. With many patients, the emotional content is the material. With this group, insisting on it will end the consultation. You do not need to know that his younger colleagues have started deferring to him differently, or that he saw himself on a video call and did not recognise the man on the screen.

You need to know what to treat, and how much, and whether he will come back.

Be brisk and technical. Explain the anatomy. Use the numbers. Tell him what the muscle does and what the injection does to it. Men who are uncomfortable being treated as aesthetic patients are usually entirely comfortable being treated as engineering problems, and the same information delivered technically is received without difficulty.

What they actually want

Two things, almost universally, and both are worth stating plainly.

Nobody must know. The result must be attributable to sleep, a holiday, or nothing at all. A man who is asked *have you had something done?* has, in his own terms, had a bad result — regardless of how it looks. This constrains everything: dose conservatively, avoid the freeze, keep some movement, never chase

symmetry into stillness, and stage anything volumetric over months rather than weeks.

It must not feminise. This is anatomical rather than psychological, but it is felt as identity. Preserve the flat, lower brow; do not lift the tail. Respect the wider, squarer lower face.

Avoid apex-high cheek projection. Treat the jaw as a structure to reinforce rather than soften. Men who are unhappy after treatment often cannot say what is wrong — they will say they look *odd*, or *not right* — and what has usually happened is that somebody has applied a feminine aesthetic template to a masculine face.

The follow-up problem

Men do not report complications and they do not come back to say something is wrong.

The pattern is consistent: the same reluctance that made it hard to arrive makes it much harder to return and admit that something has gone awry, and there is an additional layer of not wanting to make a fuss. A man with a mild ptosis will often simply wait it out for three months rather than telephone you.

So make the review appointment before he leaves, put it in the diary rather than offering it, and tell him explicitly that most men do not ring and that you would like him to be an exception. Say what specifically to ring about.

Then have somebody from your practice contact him at two weeks, which converts *him telephoning you about a problem* into *you asking him a routine question* — a much lower barrier, and the difference between catching something early and hearing about it at the next appointment.

One more thing

The male share of this market has grown considerably and will keep growing, and the practices that do well with it are not the ones with the darker paint and the different logo. They are the ones where a man can walk in, be dealt with straightforwardly, have the thing explained in terms he recognises, and leave without ever having been required to say the word *beautiful*.

The Refusal — Saying No and What It Costs

She was nineteen and she wanted lips.

I told her I did not think she was ready. Not that she could not consent — she was an adult and she could — but that her face had not finished settling, that I was not confident the wish was hers rather than borrowed, and that a lip at nineteen tends to become a larger lip at twenty-two and a different face entirely by thirty. I said it as kindly as I know how, which at that stage of my career was probably not very.

She wrote a review. A long one. Careful, articulate, and furious, and it contained a line I have never forgotten:

I am old enough to fight for my country and die for it, but he did not think I was mature enough for lip filler.

It is a good argument. It sounds unanswerable. I will come back to why it is not.

She went elsewhere. Several other reputable clinics turned her down too, which I did not know at the time and which I have thought about a great deal since. Eventually somebody said yes.

She came back to me some time later with necrosis. Filler in an artery, tissue dying, the damage tracking upwards from the lip towards the nose. We managed it, and the outcome was as good as that outcome gets, which is not the same as good.

I did not feel vindicated. I felt sick.

Because here is what a chain of correct individual decisions had produced. Every practitioner who refused her had made a defensible clinical judgement. Not one of them changed what she wanted. All they did was move her down the

list, and the list ends with whoever has the least judgement and the most availability. We did not prevent that lip.

We selected the person who would inject it.

That is the uncomfortable arithmetic of refusal, and no course I have ever attended mentions it.

What we actually did

Because a story about necrosis that does not describe the management is not much use to anybody, here is what happened after she sat down. The principle in an established occlusion is to flood. You are not trying to place hyaluronidase neatly into the offending bolus, because you do not know precisely where it is and the filler is no longer only where it was put. You are trying to get enzyme into the vascular territory in sufficient quantity that it reaches the obstruction and travels the pathway the filler travelled, reaching the distal vessels that are actually the reason the tissue is dying.

We used high-dose hyaluronidase — in the region of 1500 units in that case — with that aim.

And I want to be honest about the aim, because this is where the teaching in this field is often too clean. Getting enzyme to follow the exact path of an embolus into every distal branch is close to impossible. You cannot cannulate the problem.

What you are really doing is saturating the region and relying on diffusion, on repeat dosing, and on the fact that hyaluronidase works on hyaluronic acid wherever it eventually reaches it. So we did not treat it as a single heroic injection. We infiltrated locally and repeatedly across every area showing compromise, reviewed, and did it again.

Alongside that: aspirin, steroids, warmth, massage, and the rest of the standard protocol, with review at intervals short enough that deterioration would be caught rather than reported.

The tissue survived. The result was as good as that outcome gets, which is not the same thing as good.

Two things I took from it. High-dose does not mean single-dose — the practitioners who lose tissue are frequently the ones who gave a respectable quantity of hyaluronidase once and then waited to see. And the protocol is only as good as your access to it, which is the subject of a later chapter and the reason

I no longer treat anybody anywhere that a fridge and a colleague are not within reach.

You should hold your own protocol against current published guidance rather than against this paragraph. Mine is what I did, in a real room, on a patient who came back to me after I had turned her away.

What I do now, and why you should not do it yet

I changed my policy after that. My door is open. If somebody wants something I consider excessive, we negotiate — I will meet you halfway, we will do it in stages, and you will come back and we will look at it together before we go further. I work with patients other practitioners will not see, including people with significant psychological complexity around their appearance.

I am now going to tell you not to copy me.

That policy works because of everything sitting behind it: two decades of consultations, hyaluronidase in the building rather than in a fridge across town, a complications protocol I have used in anger, staff who know what a blanched lip looks like, and enough experience of these conversations to know within a few minutes whether the person in front of me is anxious or genuinely unwell. Remove any one of those and the same open-door policy becomes reckless.

The correct position for a practitioner in their first years is closer to this: refuse more than you want to, understand that refusal has a downstream cost you cannot see, and treat the ability to safely take on difficult patients as a late-career skill you are working towards rather than a philosophy you adopt on day one.

The point of turning people away early is not that it protects them. Often it does not. The point is that it protects them *from you*, at a stage when you are the greater risk.

That will cost you money. It is supposed to.

The distinction that matters most

There are two populations here and the literature blurs them constantly. **The red-flag patient.** Anxious, over-researched, has seen three or four practitioners, arrives with a folder of photographs, is difficult to satisfy and quick to compare you to somebody else. These patients are demanding, not untreatable. They respond well to structure: slow down, do less than asked, stage everything, review before proceeding, and document what was agreed. The halfway negotiation is designed exactly for them.

Body dysmorphic disorder. This is a psychiatric diagnosis, not a difficult personality. The defining feature is a preoccupation with a perceived flaw that others cannot see or consider trivial, occupying hours of the day and causing genuine functional impairment. The evidence here is consistent and it is not on our side: cosmetic intervention does not reliably improve the symptoms, satisfaction after treatment is poor regardless of how technically good the result is, and a proportion of patients deteriorate.

Treating a patient with untreated BDD well is not a demonstration of skill. It is the wrong intervention performed accurately.

The practical distinction is not how insistent they are. It is what the complaint is doing to their life. Ask how much time they spend thinking about it. Ask whether it stops them doing things — work, leaving the house, being photographed. Ask what they expect to change afterwards, and listen for whether the expected change is about the face or about their life.

A patient who says “I would stop noticing it in photographs” is describing an aesthetic complaint. A patient who says “I would be able to go back to work” is describing something else, and no syringe delivers it.

When you meet the second kind, your job is not refusal in the sense of a closed door. It is redirection: naming what you are seeing without pathologising them, declining the treatment clearly, and giving them somewhere else to go. Refusal without a route onward is what sent a nineteen-year-old down a list until she found the wrong clinic.

The thing I got wrong with her

She was correct that being refused felt like a judgement on her as a person. That is why she wrote a thousand words about it. I had declined a treatment; she had heard me decline *her*.

Those are separable, and most practitioners never learn to separate them. The words that do it are unglamorous — telling somebody what you *would* be willing to do, giving them a timeframe rather than a verdict, and making the next appointment before they leave. “Not this, not yet, come back in a year and we will look again” is a refusal. It just is not an insult.

And her argument, finally. She was old enough to consent, and nobody disputed it. But capacity to consent and suitability for treatment are two separate assessments, and conflating them is the error at the centre of how this entire industry discusses age. We are permitted to decline treatments we can lawfully perform. That is not paternalism. It is the whole of clinical judgement.

CHAPTER 40

What “Natural” Actually Means

Almost every patient I have ever treated has used the word natural. I have yet to establish that any two of them meant the same thing by it.

It is the most common word in the aesthetic consultation and the least useful, because it functions as a reassurance rather than a description. When a patient says *I want it to look natural*, they are usually saying *please do not make me look like the thing I am frightened of looking like* — and the thing they are frightened of varies enormously.

Treating that word as if it carried a fixed meaning is one of the most reliable ways to produce a technically good result and a disappointed patient.

The meanings hiding inside the word

In practice, *natural* is used to mean at least these things, and you will meet all of them:

Nobody should be able to tell. The most common. Detection is the failure, not the appearance itself.

I don’t want to look like the people I have seen. A negative definition, usually with specific reference points — a celebrity, a friend, a category of face.

I don’t want to be a different person. A fear about identity rather than aesthetics. Frequently in patients who have been talked into more than they wanted somewhere else.

I want it to look like nothing was done, but I want a big change. Internally contradictory and extremely common. This one has to be surfaced, because it cannot be delivered and both halves are sincerely meant.

I want to look like I did. A restoration request. Often accompanied by a photograph.

I don't want to look done, but I don't mind if it's obvious

there's something. More permissive than it sounds — this patient will accept a visible result provided it is not readable as treatment.

I want to look like the version of me other people see when I'm happy. Rare, articulate, usually the easiest patient in the list.

Seven meanings. One word.

How to find out which one you are dealing with

Do not ask them to define it. Definitions produce more of the same vocabulary.

Ask what they do not want. *What have you seen that you'd hate?* This is answered far more precisely than its positive equivalent, and it will be answered with specifics — a person, a feature, an effect.

Ask about detection. *If a good friend said "you look well, have you done something?" — is that a good outcome or a bad one?* This single question separates most of the categories above, and it takes ten seconds.

Ask for photographs. Both directions. One of themselves at a time they liked, and — if they will — an example of a result they would not want. The second is more informative than the first.

Ask who else is going to see it. A patient whose whole social circle has had work done means something quite different by *natural* than one whose circle has not and would disapprove.

Write the answer down in their words. *Natural* in the notes is worthless. *"Doesn't want anyone at work to notice; would be upset to be asked"* is a treatment plan.

The variable you cannot control

There is one further complication and it is the reason this chapter sits where it does.

The same physical outcome does not produce the same event in two different people.

In Chapter 25 I described a patient I called Steven, who had years of good work dissolved from one side of his face in pursuit of a diagnosis that turned

out to be wrong, and who came back cheerful, unbothered, and asking me to put it back. That week I also saw a patient whose brow had settled a millimetre or two after forehead toxin — an ordinary, expected, entirely temporary reduction in resting tone — who was in genuine distress and believed something irreversible had happened to her.

Same physics. Same practitioner. Opposite events.

This is not a comment on either patient. It is the central practical fact of this work: **the severity of a result is determined by the person receiving it, not by the millimetres.** A finding you can barely elicit can be the worst thing that has happened to somebody that year, as Chapter 43 describes at some length.

Which means temperament is a clinical variable, and you should be assessing it in the consultation alongside the anatomy. Some indicators, none of them definitive:

How they describe previous treatments, and whether previous practitioners are cast as villains Whether they can tolerate the idea of a two-week settling period without seeking daily reassurance How they respond when you tell them what could go wrong — engaged, or visibly destabilised Whether their satisfaction with a result seems to depend on other people's reactions to it The patient who worries you is not the demanding one. It is the one for whom a small imperfection would be intolerable, and who is asking for something in a region where small imperfections are common.

For that patient, do less. Stage everything. Treat areas with predictable behaviour before areas without. Choose reversible products. And say out loud, before anything happens, that a two-week period of asymmetry is normal and expected — because a complication you have predicted in advance is a very different psychological event from one that arrives as a surprise.

The most honest thing you can say

When a patient tells me they want to look natural, the answer I have arrived at, after twenty years, is roughly this:

Tell me what you'd hate, and I'll make sure we don't do that.

It sounds like a smaller promise than the one they asked for. It is in fact the only one anybody can keep.



PORTRAIT

Eugene

She is not, in any sense that would interest a textbook, a difficult case.

Twice a year, a woman I am calling Eugene comes in with masseters you can see from the doorway. She is a trader in her late twenties. She clenches through the working day and grinds through the night, and by the time she books, the muscle is hard under the fingers and she has a headache most afternoons.

Ten minutes of work, both sides, and she is fine for months.

What makes her worth a page is everything around the ten minutes.

Eugene does not want a slimmer face. In several years she has never once used the word contour, or jawline, or snatched, or any of the vocabulary that arrives with almost every other patient who sits down and points at the same muscle. She is not here about her appearance.

She is here because her jaw hurts and her sleep is poor, and somebody told her a doctor could help with that.

She talks markets for the entire appointment. What the desk is worried about, what is mispriced, what everyone is wrong about this quarter. I nod as though I intend to act on any of it. Neither of us pretends this is why she comes, and neither of us minds.

There is something in the arrangement that clearly works for her — fifty minutes with somebody who is competent, unimpressed, and completely outside her industry, who will not repeat any of it to anyone who matters.

The clinically interesting thing about Eugene is her calendar.

She does not return when the toxin wears off. She returns when the market does something unpleasant. Her intervals do not track the duration of the product; they track her working year. If you plotted her appointments against a chart of the last five years you would learn a great deal about her life and almost

nothing about her masseter.

I mention her because a certain number of the patients you treat in the lower face are not aesthetic patients at all. They are people whose jaws are carrying something that has nowhere else to go. The injection is genuine medicine and it genuinely helps, and it is also the smallest part of what is being asked of you.

She has never asked me how she looks. Not once. She is the only patient in this book who has never once mentioned the mirror, and I have come to think that is the same thing as her jaw: whatever most people put into their reflection, Eugene puts somewhere else.

PORTRAIT

Sarah

Sarah — not her name — has been coming to me for years, twice a year, for toxin and a modest amount of filler. She budgets somewhere in the region of several hundred pounds each time.

She has not worked in a long while. She raised her daughter on benefits and still lives on them. When she talks, which she does easily and at length, a good deal of it is about money: that the money does not stretch, that food is expensive, that the kitchen in the flat is too small to cook properly in so they end up eating things that are quicker and worse.

None of this is presented as a complaint against anybody. It is simply the weather she lives in.

She does not otherwise look after herself. I could dress that up and I am not going to. Her general self-care is poor in ways that are apparent within a few seconds of her sitting down.

And yet twice a year, reliably, she has found several hundred pounds and she is in my chair, having her frown softened and a small amount of volume replaced, and she leaves visibly pleased.

For a long time I did not know what to make of her.

The easy reading is that her priorities are disordered — that a person who cannot afford to eat well should not be spending on toxin, and that somebody ought to say so. I suspect a good number of practitioners reading this have had that thought about a patient of their own. I certainly had it about her.

I now think it is wrong, and that it is wrong in an instructive way.

Almost nothing in Sarah's life is subject to her decision. Not her income, not her housing, not the size of the kitchen, not the shape of her week. The list of things she can choose is extremely short, and this is on it. Twice a year she

makes a decision about herself, executes it, and sees the result in the mirror.

That is not a frivolous purchase competing with groceries. It is very nearly the only exercise of self-determination available to her, and it happens to take place in my clinic.

Read that way, she is not being irrational. She is allocating scarce resources to the one area where allocation still produces an outcome she controls.

Which leaves a question I am not going to resolve, because I do not think it resolves. Is a patient's ability to afford treatment any of our business? The standard answer is no — they are adults, the money is theirs, we are not their financial advisers.

That answer holds comfortably right up until you can see the rest of somebody's life, at which point most practitioners discover they have an opinion after all, and the opinion arrives dressed as concern.

I treat her. I do not upsell her, I have never suggested she come more often, and when she has asked about treatments I did not think were worth her money I have told her so directly. That is the whole of my policy and I am not certain it is right.

What I am certain of is this. When the invoice clients left London — the ones who never asked a price, who were settled at month end, who any consultant would have told me were the foundation of the business — Sarah kept coming. She was still there when they were not.

I had spent years reading the wrong signal.

PO R T R A I T

The Couple

They came to me for something close to thirteen years, together, and I never once worked out what they were for.

Both were in their sixties when they started, and very wealthy — properly so, in the quiet way that does not need to be established in conversation. They were not glamorous.

They were not extravagant. There was no maintained lifestyle around them, no discernible interest in being seen anywhere, no evidence at all of the vanity that the public assumes is compulsory in my patients. If you had met either of them at a bus stop you would have concluded they had never given a moment's thought to how they looked, and you would have been wrong in the most complete way possible.

Their treatments were modest and unfailingly conservative. Peels. A small amount of toxin. Filler in the amounts that make a face look rested rather than altered. Over thirteen years neither of them ever asked for anything I had to talk them out of, which is a sentence I can write about almost nobody else.

They came together. That was the fixed part. Same appointment, same afternoon, one after the other, and a certain amount of gentle needling of each other in the waiting room.

I used to assume, early on, that one of them was the driver and the other was being brought along. It took me an embarrassingly long time to understand that this was not the arrangement at all. It was simply something they did.

Like a standing lunch, or a walk on a Sunday — one of the small, repeating, unremarkable things that two people who have been together a very long time construct in order to keep having somewhere to be together.

Then one of them died.

The other continued for a while. Then the appointments got further apart, and then they stopped. Not out of grief exactly, or not only. When I asked, the answer was that there did not seem to be much point any more — that the face in the mirror had stopped belonging to anybody in particular now that the person who used to look at it was gone.

I have thought about that a great deal.

Thirteen years of treatment, and the treatment was never really the point. It was the occasion. What was actually being maintained was not two faces but a habit two people had built between them, and when one half of it went, the habit had nothing to attach to.

Somewhere in the public conversation about this industry there is an assumption that our patients are motivated by vanity, and that the wealthy ones are the vainest of all. I have now spent twenty years in a room with these people and I can tell you that the assumption is very nearly the opposite of the truth.

What most of them are doing is much smaller, much more ordinary, and much harder to mock: they are looking after something. Sometimes it is themselves. Sometimes, as it turns out, it is each other.

There is a broader version of this that I have had two decades to test. The profession has put me in rooms with people the public has strong opinions about — the wealthy, the famous, the ones who are assumed, by everybody who has never met them, to be a particular way.

Some of the most humble, curious, and genuinely kind people I have known came out of exactly that group, and they arrived carrying a stigma manufactured entirely by people who had never spent an hour with them.

I do not offer that as a rule either, because there is no rule. The point is the absence of one. Class and fame and money predict almost nothing about a person, in either direction, and the confident belief that they do is simply what each group does to the group it has never actually sat down with.

Everyone is wrong about the people they have not met. It is one of the few things I have learned in this work that has nothing to do with faces at all.

CHAPTER 41

The Case for Restraint — When Not to Treat

Most of what you get taught in this field is addition. What to inject, where, how much. A hundred ways to say yes. Almost nobody teaches you the harder half of the job, which is knowing when the answer is less, or nothing, or “not that, and not from me.”

Let me be clear about where I stand, because what follows can be misread as the grumbling of a man who thinks the whole industry has gone wrong. I do not think that. I have spent nearly twenty years doing this and teaching it and I believe in it. My objections are narrow. There are a handful of fashionable techniques, and a handful of recurring mistakes, that I think are genuinely harmful — and I want to tell you exactly which, and why, because the *why* is more useful than the list.

There are four things worth saying no to, and they come from four different principles.

Some damage outlasts the filler

The comforting line, repeated everywhere in this profession, is that hyaluronic acid is reversible. Wrong result? Dissolve it. And that is true — of the *product*. It is not true of the *tissue*, and the gap between those two things is where people get hurt.

The lip has a border. A fine one, anatomically and aesthetically. There is a whole family of fashionable lip techniques — the ones that inject into or across the vermilion border, that chase a shape bigger than the lip’s own architecture wants to be — and they share a problem. They stress the tissue, repeatedly, over

years, usually in young women who will be back for more. And tissue that is chronically stretched and repeatedly traumatised remodels. It fibroses. It stretches and does not spring back. You can dissolve every drop of filler you ever placed and the stretched, scarred envelope is still stretched and scarred.

That is why I treat crossing that border as a different *category* of decision, not just a bigger version of the same one. When you stay in the right plane and respect the border, a bad result genuinely reverses. When you don't, you are making a bet with the patient's tissue that the word "reversible" does not cover. I say it to trainees as a rule, and I will say it to you: fillers can be temporary, but the damage may not be. Any technique whose downside might be permanent has to clear a much higher bar than one whose downside dissolves on a Tuesday afternoon.

Over-projection does not restore a face, it rebuilds it into something else

The second one is doing too much in a single spot, and the commonest version is filler piled onto the cheekbone. The midface is dramatic — a tiny amount produces a visible lift — and that sensitivity is exactly the trap, because if a little does so much, surely more does more. It does not. Past a modest point you stop restoring the face and start building a different one: wider, flatter, catching the light in a way the eye reads instantly as *wrong*. It is one of the signatures of the over-done face, and the distance between "supported" and "inflated" is often less than a millilitre. Place the minimum that gives you the contour, then stop, and look — at rest, and moving — before you even think about more.

The commoner sin is not too much. It is not enough of the whole

thing

Now the necessary correction, because everything I have just said could be read as "do less," and that is not what I mean. In practice the *more* frequent fault I see is the opposite of over-treatment. It is incompleteness. Treating one part of a region and ignoring the parts that give it meaning.

The chin is the clearest example. Somebody projects the point of the chin and leaves the lateral walls of the chin alone, and the pre-jowl beside it alone, so now you have a chin that pokes forward but does not sit right, because the structures that would have given that projection context were never touched.

That is not the sin of doing too much. It is the sin of doing too little of the *whole*.

I file that deliberately as an error of omission, not of technique, because you fix it differently. The person who over-projects a cheek has to be taught to stop. The person who treats a chin without its walls has to be taught to *see the whole region before the first injection*. Opposite errors, opposite corrections. Restraint does not mean timid, and it does not mean treating less than the region needs. It means treating the right amount of the whole thing — which is often more complete, and more spread out, than the single fix the patient walked in asking for.

New is not the same as better

Last one. The market loves novelty, and every year there is a new injectable promising something HA supposedly can't do. I am cautious about most of them — the biostimulators, the semi-permanents — and the caution is on principle, not reflex.

HA has two things that matter more than being new. It is reversible, and it has a long, boring, well-understood safety record across a huge range of products, so a trained pair of hands can match a material to almost any plane and job. The newer agents usually give up the first of those — they don't dissolve — in exchange for an effect you could mostly have got from HA anyway. In twenty years I have not found an instant, accurate, reversible replacement for hyaluronic acid that makes giving up all of that worth it. When a product's main selling point is that it is new, and its main cost is that you cannot undo it, that trade rarely favours the person in the chair. That is not “never.” It is “the default should be the thing you can reverse and understand, and the new thing carries the burden of proving it does something genuinely necessary that HA can't.”

The whole thing, in one region: the nasolabial fold

All four of these meet in the nasolabial fold, which is why I treat it the way I do. It is tempting to fill the fold directly and heavily, because that is where the patient points. But in most faces the fold is not the problem — it is the *symptom* of the midface sliding down over it. And heavy filler packed straight into that fold, in an area that never stops moving, tends over the years to stiffen

and go hard.

So I do the opposite of what the patient expects. A little superficial dermal filler in the fold itself to soften it — not to erase it — and not much underneath. The real work is done above: cheek volume spread evenly around the bony frame of the malar apple, restoring the support whose collapse made the fold in the first place. Cause corrected above, symptom softened below. More complete than injecting the fold, and more restrained within it. That is the whole philosophy in a single crease: do more across the whole, less in the spot, and nothing at all where the honest answer is that the treatment on offer would cost the patient more than it gives.

The formal version of this argument — the four principles, with the evidence — is the fourth of the companion papers in the journal. This is the version I actually believe, said the way I would say it to you across the desk.

PART VI

The Practice

CHAPTER 42

Marketing — The Full Circle

A friend of mine, also in aesthetics, had an arrangement with several clubs in London.

The logic was sound. A large number of the women working there had breast implants that were approaching or past ten years old, and a proportion of those had developed capsular contracture — the tissue envelope tightening around the implant, hardening it, distorting the shape. For a woman whose income depends on how her body looks under stage lighting, that is not a cosmetic concern. It is a threat to her earnings. He could get them a good deal on revision, and he did.

One evening he took me along.

I had grown up with a bar of soap. Six months earlier I had been in a Chelsea dressing room failing to recognise a jar of face cream. Now I was in a club in the West End at eleven at night, being introduced as a doctor to a room of women who wanted to know, immediately and in considerable detail, what I could do about their faces.

They liked me. I still do not entirely know why. And within a few months a substantial number of them were my patients, which meant that a meaningful part of my early practice was built not by advertising but by a friend taking me somewhere and a room deciding it approved.

Two things came out of that which I have never forgotten.

The first is commercial and I will spend most of this chapter on it: closed communities do not respond to marketing. They respond to a verdict.

Dancers, models, traders, actors, anybody whose colleagues all do the same work within the same square mile — you get the whole group or none of it, and the decision is taken in a conversation you are not present for. Years later a model told me she had come because of the word on the street. That is the entire mechanism. It

cannot be bought and it cannot be rushed.

The second is clinical, and it belongs to the chapter on patients rather than this one, but I will state it here because it started that night. I had been treating appearance as self-image. For those women it was nothing of the kind. It was the means of production. The right question was never “what would make you happy” — it was “what does it need to do, and by when.”

I should also say plainly that the arrangement was his and not mine, and that referral relationships of that sort deserve more scrutiny than the industry gives them. Paying for access to a group of patients, or receiving a fee for delivering one, is a different thing from a colleague making an introduction, and the distinction matters more than the profession usually admits. I was introduced. I want that on the record before I spend a chapter telling you how practices actually grow.

The patients who could see me, and the ones who could not

Some years later I noticed something in the appointment book that I could not explain.

We were doing a great deal of masseter reduction and a surprising amount of calf reduction, and almost all of it was for Chinese patients. Not most. Almost all. Caucasian patients were booking these treatments at a rate close to zero, which made no sense, because masseter hypertrophy and heavy calves are not conditions that consult an ethnicity before appearing.

I assumed for a while that it was a difference in demand — that these were simply treatments one population wanted and another did not. That was half true at most, and it was not the interesting half.

The actual explanation was language.

Chinese patients were not searching in English. They were searching in Chinese, on Chinese-language sites, and a Chinese blogger had at some point written about her experience with me. That single post had put me at the top of results in a search market I did not know I was in, could not read, had never spent a penny on, and had done nothing to build.

An entire patient population could find me. Another one, standing on the same street, could not, because in their language I did not exist.

So we started writing about those treatments in English. Properly — what masseter reduction actually does, what calf reduction is and is not, who it suits. And the Caucasian patients arrived. The demand had been there the whole

time. It had simply never been addressed in a language those patients were searching in.

Two lessons, and I have never stopped using either.

The first is that a patient population can be invisible to you not because it does not want what you do, but because you are absent from the place it looks. Search is not one market. It is many markets, separated by language, and you are present in exactly as many of them as you have written for.

The second is more humbling. The single most valuable marketing asset my practice ever had was a blog post I did not commission, could not read, and did not know existed. I had been spending money on advertising at the time. None of it did what that one post did.

You cannot manufacture that. You can only be the kind of practitioner someone wants to write about, and then be findable when they do.

Intent is the whole of it

Everything above rests on a distinction that a surprising number of clinics never make, and it is the difference between a full diary and an expensive hobby.

Somebody typing *lip filler* and a place name into Google has already decided. They want the treatment, they want it near where they are, and the only remaining question is who does it. You are not persuading them of anything. You are simply being present at the moment they look, and the entire task is to be the most credible result on the page.

An advertisement on a social platform is the opposite transaction. It is shown to somebody who was not looking, was not thinking about it, and now has to be talked into a state of dissatisfaction before there is anything to sell. That is a cold audience, and it costs a great deal to warm one up.

Worse, the patient it produces is different in kind — persuaded rather than decided, more price-sensitive, less committed, and considerably more likely to arrive with expectations shaped by the advertisement rather than by anything you said.

In my first years the clinic became genuinely overwhelmed on search traffic alone, and I never spent meaningfully on cold advertising. The lesson has held for twenty years: **capture demand before you try to create it.** Creating demand is a legitimate business and there are people who do it well.

It is not where a clinical practice should start, and it is not what most practitioners think they are buying when they hand a media agency a monthly budget.

The doctors who leave

There is a coda to all of this that I have now watched play out several times.

Doctors who have worked in a busy clinic leave to start their own, and a proportion of them take a number of patients with them on the way out. I have never much minded that part; patients are not property, and a patient who prefers a particular practitioner should be able to follow them.

What is more interesting is what happens next, which is usually not very much.

When you work inside a practice that is already busy, the patients simply appear. There is a diary, and the diary is full, and it is extremely difficult from that position to see the machinery underneath — the years of content, the rankings that took a decade to build, the reviews, the accumulated word of mouth in a dozen closed communities, the reputation that means a blogger writes about you without being asked. None of that is visible from the treatment room. What is visible is a full diary, and full diaries look like a natural state of affairs rather than an achievement.

So you leave, you take a list of names with you, you treat them, and then the list runs out. And the question you have never had to answer arrives all at once: who makes it rain, and how.

I am not saying this to discourage anybody from going out on their own. I did exactly that, and it was the making of me. I am saying that the clinical skill is the smaller half of what a practice is, that the demand side takes years rather than months, and that the most common miscalculation in this industry is a very good doctor assuming that being a very good doctor is the business.

It is not the business. It is the thing you get to do if the business works.

The address

I started on Harley Street and I am still there, so take what follows as affection rather than cynicism.

Harley Street is easy to start on and hard to stay on. That is the whole of it, and almost nobody arriving is told.

Starting is straightforward because the model is built for it: rooms are let by the session, by the day, by the half-day, and a practitioner can hold a clinic there once a fortnight without committing to anything. Much of the building stock carries historic medical use consent, which is why there is so much of it and why the sessional market exists at all.

You can be on Harley Street next month.

Staying is a different proposition. The moment you outgrow sessional rooms — and you will, because a growing list needs continuity, storage, staff, equipment that does not travel, and the ability to see a patient the day something goes wrong — you are looking at a full-time lease in one of the more expensive square miles in Europe, in a building with restrictions on what you may do to it.

The step from part-time renting to full-time occupancy is the point at which most practices either commit properly or quietly retreat, and the cost of that step is not proportionate to the cost of the sessional room you were paying for the week before.

Then there is the other thing, which the profession is coy about.

The address is routinely mistaken for a qualification. It is not one. There is no examination, no register, no assessing body attached to the postcode — it is a street, and a lease is a commercial arrangement rather than a credential. A very large number of practitioners hold nothing there but a mailing address, used for branding and the business card, while the actual work happens somewhere else entirely and the Harley Street rooms are visited rarely, if at all.

I do not think that is dishonest in itself. It is marketing, and it works, because the public has been trained for a century to read the address as a proxy for competence. But it is worth naming plainly in a book aimed at practitioners, for two reasons.

If you are considering it, understand what you are buying: reputational borrowing, not capability, and patients who chose you for the address are choosing something you have rented. And if you are a patient, or a young doctor deciding who to learn from, know that the letterhead tells you nothing about the person, and that some of the best injectors in this country work in market towns nobody has written a song about.

One channel, done properly

The most common marketing mistake I see in new practices is not choosing the wrong channel. It is choosing all of them.

A practitioner opens, has a modest budget, and spreads it thin — a bit of paid search, a bit of Instagram, some leaflets, a local radio slot, an agency on a small retainer doing a little of everything. Every channel receives just enough money to fail, and none receives enough to teach them anything.

A year later the conclusion is that marketing does not work, when in fact nothing was ever tested.

Pick one. Search, or paid social, or leaflets in the right postcodes, or the local station — I am not going to tell you which, because it depends on your treatments, your area, and frankly your temperament. Then commit to it properly and long enough to be bad at it first.

That last part is the bit nobody says out loud. Your first attempts will not work. The first landing page will convert poorly, the first ads will be aimed slightly wrong, the first content will be written for the wrong reader. That is not evidence the channel is wrong; it is the tuition fee.

Every channel has a learning curve measured in months, and the practitioners who succeed are simply the ones who stayed on a single curve long enough to reach the top of it while everyone else was busy failing cheaply in six directions at once.

Fail properly, at one thing, and read the failure. That is the whole method.

Marketing is funny.

It goes in circles. When I started in aesthetics, the model was simple. You did good work. Patients told their friends. Their friends became patients. Nobody had a strategy.

Nobody had a funnel. You just had a reputation and a waiting list, and the two were directly connected. Then Google arrived and changed everything. Suddenly, patients were not asking their neighbour where to go. They were typing “Botox near me” into a search bar at eleven o’clock at night and booking the first clinic that appeared.

Search engine optimisation became the new battleground. If you ranked at the top of Google, your phone rang. If you did not, you were invisible. For nearly twenty years, that single shift transformed the industry. We invested heavily in educational content and SEO, and it helped build CosmeDocs into one of the most recognised aesthetic websites in the UK.

Then came reviews. Google Reviews. Trustpilot. Facebook recommendations. At first, they were genuinely useful. Patients relied on them because they reflected real experiences. Then people learned to game the system. Today, almost every clinic has 4.8 or 4.9 stars. Many clinics only ask their happiest patients to leave reviews.

Some remind patients repeatedly. Others incentivise feedback in subtle ways. The result is that reviews have become like restaurant ratings. You can find five restaurants within a mile of each other, all with almost identical scores. One serves unforgettable food.

Another serves something you would politely leave on the plate. From the reviews alone, you cannot tell the difference. When everyone has excellent reviews, excellent reviews stop being a differentiator. So something interesting has happened.

We have gone full circle. We are back to word of mouth. Only this time, word of mouth has evolved.

Build Ambassadors, Not Advertisements The most valuable marketing you will ever have is still another human being saying, “You have to see my injector.” Nothing has ever beaten that. Not Google. Not Instagram. Not AI. Not Facebook Ads. A trusted recommendation from someone whose appearance you admire is still one of the strongest influences on patient behaviour.

Think about where those people spend their time. Hairdressers. Make-up artists. Beauty therapists. Nail technicians. Personal trainers. Wedding planners. Photographers. These professionals spend hours talking to clients every single day. They build relationships, hear people’s insecurities, and are often the first person asked: “Do you know anyone good for Botox?” If you have treated them well — if they have experienced your work first-hand and genuinely believe in what you do — they become ambassadors for your clinic.

Not because you paid them. Because you earned it. Some clinics formalise this with referral programmes or commissions. Others simply build genuine professional relationships that naturally generate referrals. Whatever approach you take, transparency and compliance with local regulations are essential. But do not underestimate the power of a network of people who are proud to recommend you.

The best advertisement is not something you paid for. It is someone’s face.

Keep Your Eyes Open One of the biggest mistakes clinic owners make is thinking that marketing only happens online. It does not. Marketing opportunities are everywhere. You simply have to notice them.

When we opened our Derby clinic, we were looking for ways to make local people aware that we existed. Across the road was a David Lloyd health club. Thousands of people walked past the tennis courts every week. We rented a ninety-foot advertising banner that ran alongside the courts.

The agreement was for three months. Three months became six. Six became twelve. Eventually, the banner stayed there for almost two years because nobody ever came to take it down. For the price of a three-month campaign, we received nearly two years of continuous advertising to exactly the type of audience we wanted — people who cared about their appearance, their health, and their wellbeing.

One of the best returns on investment we ever had for that clinic. Not because it was a clever strategy. Because we noticed an opportunity that everyone else ignored. That is often the difference.

Good marketers do not necessarily spend more money. They simply notice more opportunities. Start looking around your own clinic. What is opposite you? Who is next door? What businesses already have your ideal patient walking through their doors every day? A luxury hair salon. A bridal boutique. A private gym. A golf club. A Pilates studio. A dental practice. A high-end café. Your next thousand patients may already be walking past your clinic every day. The question is whether you have given them a reason to notice you.

The Metrics That Actually Matter Most clinic owners track the wrong numbers. They track followers. Likes. Impressions. Reach. None of these pay the rent. The numbers that matter are simpler. How many new patients booked this month? What was the average spend per patient? How many patients came back within six months?

How many patients referred someone else? These four numbers tell you everything about the health of your practice. Follower counts tell you nothing. A clinic with five thousand Instagram followers and a half-empty diary is not a successful clinic. It is a well-photographed one.

A clinic with two hundred loyal patients who each return three times a year and bring a friend is a business. A real one. Build the second thing. The first one is a distraction.

The Long Game There is a version of this industry that is built on discounts, Groupon offers, and price wars. It exists. It is busy. It is also exhausting, poorly paid, and full of patients who will leave the moment someone offers them ten pounds less. Do not build that practice.

Build the one where patients come because they trust you. Where they pay your price because they know what they are getting. Where they refer their friends not because you gave them a discount code but because they are proud of the results and proud to be associated with you.

That practice takes longer to build. It is also the only one worth building. The marketing strategy for that practice is not complicated. Do exceptional work. Treat every patient as if they are your only patient that day. Follow up.

Remember their names. Know their faces. Be the kind of practitioner that people talk about at dinner parties — not because you are famous, but because you are good. The rest follows.

It always has. In the realm of aesthetic medicine, the “before and after” photograph is a powerful tool, yet one fraught with ethical and professional pitfalls if not handled with meticulous care. It serves as both a clinical record and a marketing asset, but these two functions demand distinct considerations.

First, let’s address the photography standards themselves. For any before and after comparison to hold clinical or ethical weight, the images must be captured under rigorously consistent conditions. This means identical lighting, precisely the same angle, and an unvarying distance from the subject. Crucially, no filters or digital editing are permissible.

The “before” photograph must genuinely precede any intervention, not merely be taken after a numbing cream has been applied, which can subtly alter skin texture or tone and thus misrepresent the initial state. Authenticity is paramount; anything less is a deception. Beyond the technicalities of image capture lies the critical domain of consent.

It is a nonnegotiable truth that explicit, written consent is required for every single photograph taken. Let me be unequivocally clear: consent for clinical record-keeping is entirely separate from consent for marketing purposes. A patient agreeing to have their images stored in their medical file does not automatically grant permission for those images to be used to promote your practice.

When seeking consent for marketing, the documentation must be granular, specifying precisely where and how these images will be utilized – whether on

your clinic’s website, across various social media platforms, or within educational materials. Ambiguity here is a professional liability and an ethical failing.

The ethics of social media amplify these concerns exponentially. The curated, often idealized, world of platforms like Instagram and TikTok can foster profoundly unrealistic expectations among prospective patients. While it is tempting to showcase only your most spectacular transformations, the best practitioners understand that this practice can be deeply misleading.

True professionalism dictates showing realistic results, encompassing the full spectrum of outcomes, rather than cherry-picking outliers that may be unachievable for the average patient. Transparency builds trust; hyperbole erodes it.

In the UK, the Advertising Standards Authority (ASA) rules are explicit and unforgiving on this matter. Advertising standards strictly prohibit misleading before and after images. The use of filters, retouching, or any form of digital manipulation that exaggerates results or conceals imperfections is simply not permitted. These are not mere guidelines; they are enforceable regulations designed to protect the public from deceptive advertising. Compliance is not optional; it is a professional imperative.

Furthermore, the patient’s right to withdraw consent is absolute. A patient must be able to revoke their consent for the use of their images at any time, for any reason, without penalty or prejudice. Your practice must have a clear, efficient, and well-communicated process for promptly removing any images upon such a request.

This isn’t just good practice; it’s a fundamental aspect of patient autonomy and respect. Finally, consider the professional standard: if you would not confidently present an image to the General Medical Council (GMC) as an accurate, unmanipulated representation of your work, then you absolutely should not post it anywhere for public consumption.

Your reputation, and the integrity of the entire profession, depend on upholding this standard. Before and After Photography — Standards, Consent, and Ethics We need to talk about the lies we tell on the internet. In aesthetics, the “before and after” photograph is your primary marketing tool.

But it is also a minefield of ethical failures. The Standards Consistent lighting. Consistent angle. Consistent distance. No filters. No editing. And for the love of god, take the “before” photo before you put the numbing cream on.

Numbing cream causes erythema and swelling; it changes the skin.

If your “after” photo is taken under a ring light and your “before” photo was taken in a dark closet, you are a fraud. Consent for clinical records is not consent for marketing. You need explicit, written consent to post a patient’s face on Instagram.

And the patient has the absolute right to withdraw that consent at any time, for any reason. If they ask you to take it down, you take it down. Immediately.

The Ethics of Social Media Social media has created a generation of patients with body dysmorphia, fuelled by injectors posting cherry-picked, unrealistic outliers. The ASA (in the UK) strictly prohibits misleading images. Here is the only professional standard you need: if you would be embarrassed to show the photograph to the medical council, do not post it on the internet.

In the realm of aesthetic medicine, the “before and after” photograph is a powerful tool, yet one fraught with ethical and professional pitfalls if not handled with meticulous care. It serves as both a clinical record and a marketing asset, but these two functions demand distinct considerations. First, let’s address the photography standards themselves. For any before and after comparison to hold clinical or ethical weight, the images must be captured under rigorously consistent conditions. This means identical lighting, precisely the same angle, and an unvarying distance from the subject.

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Your reputation, and the integrity of the entire profession, depend on upholding this standard.



CHAPTER 43

The Things You Cannot Control

Nothing in your training prepares you for the possibility that your practice can be reshaped by events on another continent, decided by people who have never been in a consultation room.

Twice in a decade, mine was.

The first was Brexit. The European patients went — not all at once and not dramatically, but steadily, over a couple of years, as the practical friction of coming here rose and the reasons to bother fell. These had been people who treated a trip to London as unremarkable: a morning appointment, an afternoon of shopping, an evening flight home. Then it stopped being unremarkable, and then it stopped.

The second was the war in Ukraine, and that one was faster. The Russian patients left London more or less at once.

I want to be precise about what that group represented, because the loss was not simply a number of appointments. They were the invoice clients. They did not pay at the desk on the way out; the clinic sent a bill at the end of the month and it was settled without discussion.

They did not ask the price beforehand. They did not compare us to anywhere else. In business terms this is the most desirable patient there is — high value, low friction, entirely uninterested in negotiating — and a practice that acquires enough of them starts, without ever deciding to, to organise itself around them.

Then they were gone, and it had nothing whatever to do with the quality of the work.

You could see the same thing happening across the neighbourhood. Small independent places that had depended on the same population — the coffee

shops I used, among others — did not survive it. None of those owners had done anything wrong either.

Concentration risk, which nobody in this field calls by its name

Here is the lesson, and it is not a comfortable one.

A patient base that feels like security is often the opposite. If a large share of your revenue comes from people who share a nationality, an industry, a visa status, or a single employer, you do not have a stable practice. You have a single point of failure that happens to be paying you very well at the moment.

The concentration is invisible while it works, because the money arrives reliably and reliability feels like resilience.

It is not. It is exposure that has not been tested yet.

The practitioners who came through those years best were not the most skilled. They were the ones whose patients had nothing in common with each other.

What excellence does not protect you from

I have spent this book arguing that technique matters, that anatomy matters, that judgement in the consultation is the difference between a career and an incident. All of that is true and none of it protects you from this.

There is no injection technique that survives a change in the movement of people. There is no result good enough to bring back a patient who no longer lives in the country. The single most consequential financial event in my professional life was decided by people who have never held a syringe, and my response to it — the only response available — was to accept that a chapter of the practice had closed and to go and find different patients.

That is worth sitting with early in a career rather than late. You will spend years becoming good at this, and being good at it is necessary. It is not sufficient, and it is not protective.

What actually stayed

The part that has stayed with me is who remained.

The patients who looked most permanent turned out to be the most contingent. They vanished on a news cycle. And a number of patients I would never have described as the backbone of anything — people budgeting carefully

for two appointments a year, people whose circumstances would make any accountant advise against relying on them — simply kept coming, exactly as they always had.

I have thought about that a great deal, and I do not think the explanation is loyalty. I think it is that **need predicts retention better than wealth does.** For the invoice clients, treatment was one line among many in a life full of options; when the life moved, the line moved with it.

For the patients still sitting in my chair a decade later, it was one of very few things they had chosen for themselves, and nothing about a change of government or a war in another country touched that.

You cannot tell from the outside which of your patients is genuinely committed. You spend your early career chasing the ones who look like money, and your later career discovering that you were reading the wrong signal the entire time.

When Things Go Wrong

In the early years I treated patients on aircraft.

Not routinely, and it was less glamorous than it sounds, but it happened: a private jet on the ground at a London airport during a refuelling stop, a ninety-minute window, a patient who was flying on to somewhere else that evening and who did not consider the schedule negotiable. You did the treatment on the aircraft. You got off. The aircraft left.

I did a number of those, and they went well, and I was pleased with myself in the way you are pleased with yourself at that stage of a career, when the aeroplane feels like evidence that something is going right.

It was a while before it occurred to me to conduct the following audit. If that patient had occluded a vessel at thirty thousand feet — or worse, on the tarmac with the doors about to close — what did I have?

A syringe. A pair of gloves. Someone else's aircraft.

The hyaluronidase was in a fridge in London.

Nothing happened. That is the only reason I can tell the story at the front of a chapter rather than somewhere much darker in it, and it is not a defence. Nothing happening is not the same as being prepared. I had confused competence with safety, which is the specific error this chapter exists to correct.

Preparedness is not a state of knowledge. You can recite the protocol perfectly and still be standing in a place where you cannot perform it. It is a question of geography: what is in the room, how far away is the thing you need, who else is present, and how long would it take you to get help. Answer those four questions honestly about every location in which you treat patients, including the ones that impress people.

The lightning strike

In my first year of practice I employed my first doctor. Excellent on paper — a European qualification at masters level, better formal training in aesthetics than I had at that stage — and the clinic was busy enough that I needed her.

In those days we treated deep glabellar lines with filler. Everybody did. We knew the region was dangerous and we believed we were being careful, and looking back at what careful meant in that period, we were naive. Thirty-gauge needles. Boluses in the region of 0.2ml under a single frown line. It seems an enormous quantity to me now, and at the time it was unremarkable.

A patient came back with what she and my colleague both described as a bruise.

It was not a bruise. It was tracking vertically up the glabella in a narrow, branching, unmistakably vascular pattern — a lightning strike, which is the only description I have ever found that does it justice, and once you have seen one you will never again mistake it for anything else. I knew what I was looking at within a second or two of walking into the room.

We ran the protocol. The skin went dusky, then blue-black, then scabbed, and in due course the eschar separated. At two months there was almost nothing to see. It was, in the end, as good an outcome as that event permits.

Two things about that day have stayed with me and neither is clinical.

The first is that when I finished examining her and walked out to find my colleague and tell her what had happened, she collapsed into my arms and cried. She was a competent doctor who had done nothing that the field at the time considered wrong, and she was devastated.

Nobody prepares you for that side of it — not for having a complication, but for supervising somebody through theirs.

The second is that I was, in every meaningful sense, on my own. There was no senior partner down the corridor. This work is stressful in a way that is easy to underestimate from outside, and it is markedly more stressful as a lone practitioner, which most of you reading this either are or will be.

What we changed, and what a decade of it looks like

We did not stop treating the glabella. Stopping is the response that feels responsible and teaches nothing. We changed how.

First, we moved to cannula for that region, for a period. No further events.

Then we capped the volume. A maximum of 0.05ml beneath any single frown line — a quarter of what we had previously considered normal.

Then we abandoned bolus placement entirely in favour of what I now call the invisible needle: very small deposits of hyaluronic acid placed in the mid-dermis, spread across the area, with a hard ceiling of 0.5ml in total regardless of how many lines there are or how deep they look.

The intention is no longer to fill a crease. It is to hydrate the dermis and stimulate collagen, and to let the line soften over weeks rather than disappear in the mirror while the patient is still in the chair.

We have used that technique for more than ten years. More than half of the patients we see with deep frown lines have it, which over that period is somewhere between three and four thousand people. In that time we have had no vascular compromise in the region.

I want to be careful about what that does and does not prove. It is a case series, not a trial, and no volume of uneventful cases makes any glabellar injection safe — the anatomy is what it is, the supratrochlear and supraorbital vessels are where they are, and the connection to the retinal circulation does not care how long your run has been.

What it does demonstrate is the principle underneath, which generalises well beyond the glabella:

The dose is the risk. Not the needle, not the product, not the practitioner's confidence. In the danger zones of the face, the single most protective decision available to you is to inject a smaller volume, more superficially, in more places, more slowly, and to accept a slower and less dramatic result in exchange.

Every one of the three changes we made was a reduction, and the technique we ended up with produces a quieter result than the one that caused the injury.

Two events in several thousand cases sounds like a low number. It is a low number. It is also two people whose skin died, and both of them were avoidable with what we know now.

The bruise the patient did not mention

A few years later the same doctor had her second event, and the shape of it is different in an instructive way.

She was treating a cheek — a small, superficial deposit intended to camouflage a shallow dip in the surface. The patient noticed a small bruise afterwards and did not report it, because patients do not report bruises. It darkened, went blue-black, and eventually separated, leaving an area of new pink skin roughly the size of a penny that took around six months to blend.

The patient came back for her next appointment as though nothing had happened. My colleague looked at her photographs, understood immediately what had occurred, and told her — explained what it had been, that it should have been reported, and what would have been done had she come back.

The patient was entirely unbothered. She was fond of the doctor, the area had healed, and as far as she was concerned that was the end of it. Some months later a letter arrived from a solicitor. One of the no-win- no-fee firms, seeking compensation.

My colleague procrastinated, as people do when a letter frightens them, and by the time she had worked out what to say the firm had not written again. It was never pursued.

I do not offer that as a strategy — it was luck, and the correct response to a letter of claim is to notify your indemnifier the same day and to say nothing to anybody until they tell you to.

What the episode teaches is this:

Patients do not report the early signs. A bruise is the most normalised finding in our field. Say explicitly, out loud and in writing, that a bruise which darkens, spreads, becomes painful or looks like a network of lines is a reason to contact you the same day, and that you would far rather see fifty normal bruises than miss one that is not.

A superficial injection is not a safe injection. She was placing a tiny amount, superficially, to correct a trivial contour. That is exactly the setting in which practitioners relax, and it produced skin loss.

Satisfaction and litigation are only loosely related. That patient was not angry. She was not dissatisfied. A letter arrived anyway, because somewhere between the appointment and the letter she spoke to somebody who told her she had a claim. You cannot manage that by being liked.

Honesty is still correct. My colleague's decision to explain what had happened, unprompted, to a patient who had not noticed, was the right one and I would want every doctor working for me to make it. It is also the reason there was a contemporaneous record of a competent, candid clinician — which is precisely what you want in existence if a claim is ever pursued.

The complication that nearly ended my career, and it was two millimetres

He was a student at Oxford, and he had facial blushing.

Not the ordinary kind. The kind that had organised his entire life around avoiding the circumstances that triggered it — seminars, presentations, being addressed unexpectedly. He had looked for help and found almost none, because in those years nobody was treating this, and he had found me through a forum where somebody else had mentioned my name. He was articulate, thoroughly researched, and quietly desperate.

I saw him for consultation, explained what was known and what was not, explained that this was an off-label use of the drug, and treated him on a separate day rather than the same one. Microdoses of Dysport across a wide area of the mid-face, the butterfly region either side of the nose — around thirty units in total, which is somewhere near ten units of the other brand, spread thinly over a large surface.

A week later, I was in Dubai attempting to have a holiday when my secretary rang. The patient's parents were on the phone and they were extremely angry. Their son had not left his room in college for a fortnight.

He had developed asymmetry.

This was before video calling was something you simply did, so I could not look at him. I arranged for a colleague to assess him in person, immediately. What my colleague found was a very mild restriction of the right-sided smile, visible only on maximal smiling — the sort of finding you have to ask a patient to produce before you can see it at all.

Two things were true simultaneously, and holding both is the whole of this section.

Clinically, this was a minor, self-limiting, entirely recoverable event. Toxin diffusing a few millimetres into a zygomatic fibre. It would resolve completely within weeks and it did.

And for a nineteen-year-old whose original complaint was that he could not bear being looked at, it was a catastrophe. He had come to me because his face betrayed him in public. I had given him a second, different way for his face to betray him in public.

The severity of a complication is not measured on your examination. It is measured against what the patient came in frightened of.

Then it escalated. The parents were extremely well connected — public figures, the sort whose displeasure travels — and they had their son assessed by a plastic surgeon near them, who told them that what I had done was borderline illegal.

That word did more damage to my week than the asymmetry did.

It was also wrong, and it is worth being precise about why, because you will meet this at some point in your career. Using a licensed medicine outside its licensed indication is not illegal. It is off-label prescribing, it is lawful, it is routine across the whole of medicine — a very large proportion of paediatric prescribing is off-label — and the obligations it carries are that the decision must be justifiable, the evidence must be honestly represented, an appropriately qualified prescriber must take responsibility, and the patient must be told and must consent.

All of which had happened, and all of which was documented in a consent form the surgeon commenting on the case had not read.

I did not know that with any confidence at the time. What I felt was that my career was over, and I spent several days in the particular kind of fear that only comes when you cannot tell whether you have made a clinical error or merely made an enemy.

So I did the only sensible thing available. I rang a colleague — considerably senior to me, professorial, someone with no reason to protect me — described what I had done, and asked him to tell me honestly whether it was defensible.

He said it was. He did not say very much beyond that, and he did not need to. That conversation is the reason I did not spend a year quietly frightened, and we have been close friends ever since.

What I would tell a younger version of myself about that fortnight:

The patient's fear defines the complication, not your

examination. A finding you can barely elicit can still be the worst thing that has happened to somebody that year.

Have a plan for being away. Not a colleague you could ask if it came to it — a named colleague who has agreed in advance, whose details your staff already have, who can put hands and eyes on your patient the same day. I did not have that. I improvised it under pressure and I was lucky.

Consent is not paperwork, it is armour. The only reason that episode ended in a friendship rather than a hearing is that everything I was accused of not disclosing was written down, dated, and signed, because I had explained it in a separate appointment and given him time.

Colleagues will comment on your work having seen none of it.

Some of them will be wrong, some will be confidently wrong, and a small number will be wrong in front of a patient's family. You cannot prevent that. You can make sure your notes describe a defensible decision, which is the only version of the story that survives the argument.

Ring the most senior person who will take your call. Not for reassurance. For an honest assessment while you still have time to act on it. Every practitioner needs one person like that, and if you do not have one, acquiring one is more valuable than any course you will attend this year.

Let us be honest about something most courses gloss over. Complications will happen. Not might happen. Will happen. If you inject enough faces over enough years, you will encounter problems. The question is not whether you will face a complication. It is whether you will recognise it in time and know exactly what to do when you do.

This chapter is not here to scare you. It is here to prepare you. The practitioners who panic in a complication are the ones who were never taught to expect it. The practitioners who manage it well are the ones who had already rehearsed the protocol in their minds long before they needed it.

There are three categories of complications that every injector must understand. The serious and rare (vascular occlusion). The common and manageable (lumps, bumps, and biofilm). The preventable but frequently missed (post-inflammatory hyperpigmentation and skin complications from peels). We will deal with each in turn.

Vascular Occlusion — The Complication You Cannot Afford to Miss Vascular occlusion is arguably the most feared complication in aesthetic practice, and for good reason. It demands immediate, decisive action. Hesitation, or worse, ignorance, can lead to devastating and irreversible consequences for your patient. This isn't a situation for 'wait and see'; it's a full-blown emergency requiring a clear, step-by-step protocol that you must know intimately.

A vascular occlusion occurs when blood is no longer able to pass through a vessel. It may be a complete blockage or a partial one that reduces blood supply to the tissue. When a blood vessel supplying the skin is compromised and the situation is not corrected quickly, the result is skin necrosis.

The death of tissue. This is the most feared complication in aesthetic medicine. The mechanism matters here. When filler material enters or compresses a vessel, three things can happen. The filler may directly embolise into the artery. It may compress the vessel from outside. Or it may trigger a vascular spasm that restricts flow through the surrounding anastomoses.

Of these, direct intravascular embolism is the mechanism best supported by the evidence. Hyaluronic acid, although well tolerated outside vessel walls, is highly inflammatory within them. It produces intense vessel wall inflammation and spasm.

The two areas of the face with the highest incidence of vascular occlusion are the glabellar region and the nasolabial fold/nasal tip/alar triangle. The glabella is supplied by the supratrochlear artery, the supraorbital artery, and cutaneous branches of the ophthalmic artery. Fifty percent of reported cases occur here.

The collateral circulation in this watershed area is poor. The nasal tip and alar are supplied by the angular artery. It turns sharply within the alar triangle and is prone to both external compression and inadvertent injection. The midline of the face deserves particular caution.

Recognition — The Five Signs The first, and often most critical, step is swift recognition. You must be acutely aware of the tell-tale signs that scream vascular compromise. The primary indicator is blanching, a sudden, stark paleness of the skin in the injected area or distal to it, indicating a lack of blood flow.

This is not merely the transient blanching from needle trauma; it persists and often spreads. Next, observe for pain disproportionate to injection. While

injections are never entirely painless, a patient reporting severe, escalating pain that feels ‘wrong’ should immediately raise a red flag. This isn’t just discomfort; it’s ischemic pain.

A livedo reticularis pattern—a mottled, net-like, purplish discoloration of the skin—is a classic sign of compromised microcirculation and demands urgent attention. Fourth, assess delayed capillary refill. Press firmly on the blanched or discolored skin and release; if the normal pink colour does not return within 2-3 seconds, blood flow is impaired.

Finally, if you are injecting in areas close to the eye, be vigilant for visual changes. Any report of blurred vision, diplopia, or sudden vision loss is an ophthalmic emergency, indicating potential occlusion of the ophthalmic artery territory, and requires immediate, aggressive intervention. The signs of vascular occlusion present on a timeline.

Knowing that timeline is what separates a managed complication from a catastrophic one. Pain is usually the first sign. Experienced immediately at the time of injection. However, if a local anaesthetic has been used, this warning sign may be absent or masked. Extraordinary pain during a soft tissue filler treatment is not a normal feature of the procedure.

Treat this as a vascular event until proven otherwise. Blanching follows. A pale, white, or dusky discolouration of the skin. This blanching may initially be transient and local. If unresolved, it will become reticulated or irregular. Adrenaline in local anaesthetics can mimic and even disguise blanching.

A livedo pattern — a mottled, net-like discolouration — may appear within minutes to tens of minutes. Delayed capillary refill time greater than three seconds is suggestive of vascular compromise. A blue-grey appearance develops over tens of minutes to hours. Skin breakdown and necrosis occur over days if the situation is not managed.

Here is the honest truth about vascular occlusion. By the time you see the signs, the damage has often already begun. You are not reversing the occlusion so much as performing damage control. Stopping further injury. Maximising collateral perfusion. Preventing the cascade from progressing to full necrosis. That reframing matters. It focuses your attention on the speed and completeness of your response.

Immediate Action — Stop Injecting Immediately This is non-negotiable. The moment you suspect vascular occlusion, your hand must come off the plunger. Stop injecting immediately. Do not, under any circumstances, inject

more filler. Adding more product to an already compromised vessel will only exacerbate the problem, pushing the occlusion further or increasing the volume of the blockage.

Your priority shifts from aesthetic enhancement to damage control. Remove the needle, apply gentle pressure, and prepare for the hyaluronidase rescue. The Hyaluronidase Rescue — Dose and Administration Hyaluronidase is your primary weapon against hyaluronic acid filler-induced vascular occlusion. There is no room for conservatism here.

The recommended dose is 1500 IU, dissolved in 1 ml of saline. This concentration allows for potent, localized degradation. Inject this solution liberally throughout the affected area, directly into the suspected occluded vessel and the surrounding tissue. The goal is to flood the area with enzyme, ensuring maximum contact with the filler.

Do not be conservative with your administration. The risk of under-treating, leading to tissue necrosis or permanent vision loss, is far greater than the risk of over-treating with hyaluronidase. You are in an emergency; err on the side of aggressive dissolution. Warm Compress and Massage — Encouraging Vasodilation Once hyaluronidase has been administered, supportive measures are crucial.

Apply a warm compress to the affected area. The warmth helps to encourage vasodilation, which can improve blood flow and aid in the dispersion of the hyaluronidase and the dissolved filler. Concurrently, gently but firmly massage the area. This physical manipulation can help to break up any remaining filler, distribute the hyaluronidase more effectively, and further promote blood flow through the compromised vessels.

Continue these measures consistently as you monitor the patient. Aspirin 300mg — Reducing Platelet Aggregation In addition to local measures, systemic support is vital. Administer 300mg of aspirin immediately.

Aspirin acts as an antiplatelet agent, reducing platelet aggregation and preventing further clot formation within the compromised vessel. This helps to maintain any remaining blood flow and prevent the occlusion from worsening. Ensure the patient has no contraindications to aspirin before administration, but in an emergency, the benefits often outweigh the risks. Timeline — Reassess Every 20 Minutes Time is tissue. After initiating treatment, you must meticulously monitor the patient's response. Reassess the affected area every 20 minutes. Look for signs of improvement: return of normal skin colour,

reduction in pain, improved capillary refill, and resolution of any livedo pattern.

If, after 60 minutes, the blanching or other signs of occlusion persist, or if the patient's condition deteriorates, you must escalate care. Call 999 (or your local emergency services) immediately and arrange for transfer to a hospital. This is beyond your immediate scope, and the patient requires specialist intervention.

Documentation — Photograph Everything Thorough documentation is paramount, both for medical-legal reasons and for learning. Photograph everything: the initial presentation of the occlusion, the area after hyaluronidase administration, and subsequent changes over time. Document the exact time of injection, the specific product used, the volume injected, the entry point, and critically, the precise time of hyaluronidase administration.

This detailed record will be invaluable for subsequent medical professionals and for your own audit and reflection. **The Most Important Rule — Hyaluronidase Must Be In The Room** Let me be unequivocally clear: hyaluronidase must be in the room before you inject a single unit of hyaluronic acid filler.

Not in the cupboard down the hall. Not in your car in the parking lot. It needs to be immediately accessible, on your treatment tray, ready to be drawn up and administered within seconds. This isn't a suggestion; it's a fundamental safety requirement. If you don't have it, you don't inject.

Period. The consequences of not having it instantly available are simply too dire to contemplate. The moment you suspect a vascular occlusion, stop injecting immediately. Do not inject another drop.

Aspirate any product you can recover as you withdraw the needle or cannula. Then begin the following protocol without delay. Massage the area firmly. Firm and prolonged massage can encourage blood flow and help dislodge any obstruction. Apply heat to the area.

Warmth encourages vasodilatation. Tap the area gently. Tapping over the injection site can dislodge intra-arterial emboli. Assess and reassess the capillary refill time continuously. Apply moderate pressure with a finger to the area for five seconds, then release. The time for the skin to return to its normal colour should be two seconds or less.

If it is greater than three seconds, or if conservative measures are not improving the picture, move immediately to hyaluronidase. Hyaluronidase is

your primary treatment. The ACE Group High-Dose Pulsed Hyaluronidase Protocol recommends repeated administration of relatively high doses of hyaluronidase into the whole area of compromised tissue.

Not just where the filler was injected. Repeated hourly until clinical resolution is observed. It is not necessary to inject directly into the vessel itself. Injecting hyaluronidase into the subcutaneous plane surrounding the area has shown more favourable outcomes. Alongside hyaluronidase, administer aspirin 300mg as a stat dose immediately. Then 75mg daily until the vascular occlusion has resolved.

Aspirin limits platelet aggregation and clot formation. Consider systemic antibiotics. Necrotic tissue is prone to opportunistic anaerobic infection. A note on nitroglycerin paste. This was historically recommended to encourage vasodilatation. Current evidence does not support its use. It can cause a more congested appearance and worsen perfusion.

It is no longer recommended. The most important thing you can do after managing a vascular occlusion is to document everything. Photograph the area. Provide the patient with an emergency contact number. Follow up daily until the situation has completely resolved. Diligent follow-up is the best approach to prevent a complication from turning into a litigious medical malpractice claim.

Biofilm — The Invisible Complication I went to a conference about fifteen years ago. A doctor was presenting on biofilm. He was describing case after case of delayed inflammatory nodules, chronic swelling, and treatment-resistant infections in his patients. I was shocked.

Not because biofilm is rare. Because it was so common in his practice. Then I understood why. The practitioners presenting the most biofilm cases were not from surgical backgrounds. And practitioners who are not from surgical backgrounds have not been taught surgicallevel cleanliness. This is not a criticism.

It is an observation. Surgeons are trained in a very specific, very deliberate way. The rules around contamination are drilled in from day one. Non-surgeons have to learn those rules consciously, because nobody taught them automatically. I have trained hundreds of doctors over the years.

When I ask them to clean the skin at the start of a procedure, the level of skin preparation from non-surgical practitioners is, frankly, not good enough. Even after five cases.

Even after being shown. The habits are not yet ingrained. And it is not just about wiping the skin with antiseptic. It is about everything that happens during the procedure. The needle brushing past the patient's hair. The syringe resting on the corner of the table.

The gloved hand touching the patient's face and then touching the barrel of the syringe.

The uncapped needle sitting in a field that is not clean. All of these are contamination events. All of them are preventable. The Sterile vs. Aseptic Distinction This is the most important concept in this section. Aesthetic injection is not a sterile procedure. It is an aseptic procedure. Sterile means no microorganisms present at all. The inside of an operating theatre during a joint replacement is sterile. The instruments are sterile. The gloves are sterile. The drapes are sterile.

Aseptic means reducing the microbial load to a clinically safe level. The gloves you use in aesthetic practice are not sterile. They are aseptic. The gauze you use is not sterile gauze. It is aseptic.

The skin you are injecting through is not sterile. It never will be. Because you are working in an aseptic environment rather than a sterile one, you must compensate with more deliberate measures. You cannot rely on the environment to protect you. You must rely on your own discipline.

The only thing that is sterile in your treatment room is the material inside the syringe. The outside of the syringe is not sterile when you open it from the package. The needle is sterile until it touches anything. Once it touches the patient's skin, the table, the gauze, the glove, or the air — it is no longer sterile.

What Biofilm Actually Is The single biggest cause of biofilm formation is bacterial contamination at the time of injection. Specifically, introducing the patient's own resident skin flora into the deeper tissue layers. When a needle or cannula penetrates the skin, it can catch microscopic traces of bacteria hiding beneath the surface layer and within the pores, and seed them directly onto the filler material.

The bacteria responsible are almost never exotic hospital superbugs. They are the ordinary commensal bacteria that live naturally on human skin and in the oral cavity: Organism Source *Staphylococcus epidermidis* Skin surface *Staphylococcus aureus* Skin, nasal mucosa Notes Most common biofilm pathogen in aesthetic practice More virulent; associated with more aggressive presentations *Cutibacterium acnes* Sebaceous follicles *Streptococcus* species

Oral mucosa Particularly relevant in acneprone skin Especially relevant in lip filler due to proximity to the mouth Once these bacteria are dragged into the subcutaneous or periosteal layers, the dermal filler acts as a foreign body substrate. The filler gel has no blood supply. No active microcirculation.

The body's immune cells — macrophages and neutrophils — cannot effectively patrol or penetrate the centre of the product mass. The bacteria bind to the surface of the filler and rapidly secrete an Extracellular Polymeric Substance — a sticky, slimy shield made of proteins and polysaccharides.

This matrix seals the colony inside, physically blocking antibiotics from reaching it and making the bacteria up to one thousand times more resistant to treatment than free-floating organisms. This is why biofilm is so difficult to treat once it forms. The Dormant Period and Reactivation Once a biofilm forms, it typically enters a dormant, low- metabolic state.

The patient may feel completely fine for months or even years. There is no obvious sign that anything is wrong. Then something wakes it up. The three most common reactivation triggers are: Transient bacteraemia. A dental cleaning, a dental procedure, or a systemic infection sends a wave of unrelated bacteria through the bloodstream.

These bacteria adhere to the existing filler site and reactivate the dormant colony. This is one of the reasons patients should inform their dentist that they have dermal filler before any dental work. Local physical trauma.

Aggressive facial massage, laser treatment, microneedling, or a subsequent filler injection in the same anatomical region physically disturbs the bacterial colony and triggers an inflammatory response. Immune fluctuations.

Viral illnesses or vaccinations that temporarily redirect or hyperactivate the immune system can cause it to suddenly detect and attack the hidden biofilm matrix, producing a delayed inflammatory nodule that appears to have no obvious cause. Clinical Presentation Biofilm typically presents as a delayed inflammatory reaction.

Redness, swelling, and firmness at the injection site appearing weeks to months — sometimes years — after treatment.

The key distinguishing feature is the delay. An immediate inflammatory reaction after filler is usually a technique issue or a hypersensitivity response. A delayed reaction — particularly one that appears after a dental procedure, an illness, or a vaccination — should raise biofilm as the primary differential.

Prevention — The Only Reliable Strategy Because a fully formed biofilm is incredibly difficult to eradicate, prevention is the only reliable strategy. Remove all makeup. Not just from the injection site.

From the entire face. Your gloved fingers move across the face during treatment. Makeup anywhere on the face is a contamination risk. Use chlorhexidine or alcohol-based antiseptic. Apply it properly.

Allow it to dry completely before injecting. Antiseptic that has not dried is still active and can carry bacteria on the surface. Never inject through active lesions. No active acne papules.

No perioral dermatitis. No cold sores. These are bacterial reservoirs sitting directly on the skin surface. Minimise needle passes. Every re- entry of a needle into the skin increases the probability of dragging surface bacteria into the deeper tissue. Plan your injection sequence to minimise the number of entry points.

Hold the syringe correctly. Do not let the barrel rest on the patient's skin. Do not let the needle brush past hair, clothing, or any surface that is not clean. Once the cap is removed, the needle is exposed. Treat it accordingly.

Do not place the syringe down between passes. If you must pause, recap the needle. The moment the tip touches a surface, it is contaminated. **Treatment — When Prevention Has Failed** When biofilm is suspected, the treatment protocol is: 1. Dissolve the filler with hyaluronidase.

The biofilm cannot survive without its substrate. 2. Prescribe a course of antibiotics covering both gram-positive and gram-negative organisms. The standard protocol is clarithromycin combined with metronidazole for four to six weeks. 3.

In some cases, intralesional steroid can reduce the inflammatory component. Do not inject more filler into an area of suspected biofilm. This will worsen the situation significantly. You are not treating a volume deficit. You are treating an infection.

More filler provides more substrate for the bacteria to colonise. “The best biofilm treatment is the one you never need. Clean the skin. Hold the syringe properly. Respect the needle.

These habits take five minutes to learn and a lifetime to maintain.”

Lumps and Bumps — The Most Common Complaint The most common complaint you will receive from patients after filler treatment is not a vascular event. It is lumps. Lumps are the bread-and- butter complication of aesthetic

practice. They are almost entirely preventable.

Fillers are injected as a liquid and remain relatively fluid for the first few days. Over the following two to three weeks, they begin to integrate with the surrounding tissue and firm up. If the filler has been deposited unevenly, it will firm up in the wrong shape.

The result is a visible or palpable lump. The treatment is massage. Firm massage. Not gentle patting. Firm, deliberate pressure applied to the area every day for two to three weeks as the filler settles. Most practitioners do not advise this clearly enough. The result is pocketed lips and uneven cheeks that could have been smoothed out with a week of daily massage.

A note on migration. Patients often worry that massaging will cause the filler to migrate. This is not how migration works. Filler does not move from one compartment to another as a result of massage. Migration occurs because of incorrect injection technique. Placing the product in the wrong plane or the wrong location.

When you see what appears to be migration weeks after treatment, it is almost always the result of where the filler was placed at the time of injection. Not what happened to it afterwards.

If lumps persist after thorough massage over several weeks, a small amount of hyaluronidase injected directly into the lump will dissolve it cleanly. Hyaluronidase is the last resort, not the first response.

Post-Inflammatory Hyperpigmentation — The Skin Complication Post-inflammatory hyperpigmentation, or PIH, is the most common skin complication from aesthetic treatments. Particularly from chemical peels. It occurs when the skin's inflammatory response to a treatment triggers an overproduction of melanin. It results in dark patches that can persist for months. The key to managing PIH is to prevent it where possible.

And to recognise it early when it occurs. Prevention means conditioning the skin before any aggressive treatment. For peels above twenty percent TCA, the skin should be prepared for several weeks beforehand. A regime of hydroquinone, kojic acid, Vitamin C, and retinol. This suppresses the melanocyte activity before you provoke it with the peel. It significantly reduces the risk of PIH.

When PIH occurs, the treatment is a consistent regime of hydroquinone and tretinoin. Applied morning and evening. Combined with a broad-spectrum SPF fifty sunscreen used without exception every day. The

hydroquinone inhibits tyrosinase.

The tretinoin accelerates cell turnover. Results take time — typically six to twelve weeks of consistent use. But the combination is highly effective when used properly. The temptation to repeat the peel to “treat” the PIH is one of the most common mistakes in aesthetic practice.

Do not do it. The skin is already inflamed. Adding another chemical insult will worsen the hyperpigmentation, not improve it. Condition the skin first, resolve the PIH, and then consider whether a further peel is appropriate.

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CHAPTER 45

The Thing No Course Can Give You — On Mentorship

When I was a medical student at Johns Hopkins, I did not learn very much.

I want to be precise about that, because it sounds like false modesty and it is not. I did not acquire a great deal of knowledge there that I could not have acquired elsewhere. What I acquired was a standard.

I was in rooms with people who were the best in the world at what they did, and I watched them. Not on a stage, not in a lecture, not in the carefully edited version of the work that gets presented at conferences — in the ordinary conduct of a day. How they spoke to patients. What they did when something did not go as expected. Which things they took seriously and which things they visibly did not. How long they were prepared to stand and think before doing anything at all.

None of that was taught. It was ambient, and it was the most valuable thing in the building.

A student who has never seen excellence has to construct their idea of “good enough” from their own results, which means good enough is defined by whatever they happen to be capable of at the time. A student who has stood next to somebody genuinely exceptional has an entirely different problem: they now know precisely how far away they are, and they spend the next twenty years closing the distance. The second problem is enormously more useful.

Aesthetic medicine is not different, and in one respect it is worse.

Anything more complicated than safe phlebotomy requires a mentor. That is the position I have arrived at after two decades, and I hold it fairly firmly. A two-day course can teach you where the supratrochlear artery runs and what to do about an occlusion. It cannot teach you what a face looks like when it is

asking for something the patient has not said, and it cannot teach you what to do when the anatomy in front of you is not the anatomy in the diagram — which, since every face is different, is most days.

What the course cannot show you

Sit in an experienced practitioner's clinic for a fortnight and you will see the parts of this job that have no module attached to them.

You will see consultations — actual ones, not demonstrations. How the question gets asked. What silence is used for. How a patient's stated request is gently separated from their actual complaint without anybody being made to feel foolish. That skill is the whole of Part V of this book and it cannot be conveyed in writing, including by me.

You will see the treatments that carry no price. The five minutes spent looking at a face before touching it. The topping-up of something eighteen months old that nobody is charging for. The consultation that ends with nothing being done, and the practitioner appearing entirely untroubled by that, which teaches a junior more about integrity than any lecture on ethics.

You will see somebody deal with a complication in real time, which is worth more than any number of slides about complications.

And you will see the business, which is the part nobody expects to learn and everybody needs. How the diary is built. Why the room is laid out the way it is. What is said on the telephone before a patient ever arrives. The reasons a practice is busy are largely invisible from the treatment chair, as I said in the previous chapter, and the fastest way to make them visible is to stand in one and pay attention.

Choose upwards, and be inconvenient

Two pieces of practical advice.

Choose the most experienced mentor you can possibly get. Not the most available, not the cheapest, not the one running the course with the best marketing. The gap between a good mentor and an exceptional one is not incremental — you inherit their standard, their instincts, their sense of what is normal, and you will carry it for the rest of your career. A mediocre mentor does not slow you down. They install a ceiling.

Then be prepared to be a nuisance. The hardest part of this is access, because the people worth watching are busy and have no obvious reason to let you into their clinic. Ask anyway.

Ask repeatedly. Offer to be useful. Take the unglamorous slot. I have never regretted saying yes to somebody who asked to observe, and neither have most of the practitioners I respect — the profession is considerably more generous than it looks from outside, and the limiting factor is almost always that nobody asked.

What it is actually worth

I will put a number on it, or as close as I can.

Watching someone excellent, properly, for a sustained period, is worth years. Not weeks. Years — of technical development, of the confidence to do less, of the judgement to decline, and of the commercial decisions that determine whether you still have a practise in a decade.

Every serious mistake in this book cost me something, and a proportion of them were mistakes that somebody in the room could have prevented with a sentence, had there been somebody in the room.

For a long stretch of my early career there was not. I was a lone practitioner learning by doing, and the tuition was paid by my patients rather than by me.

Do not repeat that. Find somebody better than you and stand next to them until you have stolen everything they know.



CHAPTER 46

The Last Lesson

Here is the great paradox of aesthetic medicine: the better you get at this job, the less visible your work becomes.

When you start out, you want everyone to see what you did. You want the impossibly sharp jawlines, the massive lips, the obvious cheeks. You want the applause. You want to walk into a room and have people point to a face and say, *He did that*.

But eventually, if you have any talent at all, you realise that the ultimate goal is to be entirely forgettable. The best compliment a patient can get is not “Who does your filler?” It is “You look incredibly well-rested. Did you just get back from holiday?”

You are not building monuments to your own ego. You are quietly erasing the shadows that life leaves behind.

Technician vs. Artist

A technician is someone who injects exactly what the patient asks for. A patient walks in, points at a line, and asks for 2ml in the lips. The technician nods, opens the box, and delivers 2ml in the lips. It is a transaction. It requires no thought, no spine, and no vision.

An artist is someone who injects what the patient actually needs. The artist says no. The artist explains why. The artist protects the patient from their own body dysmorphia, from the terrifying distortions of Instagram filters, and from the well-meaning but catastrophic advice of their friends.

To be an artist in this industry requires courage. It requires the willingness to let a patient walk out of your clinic angry because you refused to overfill

their face. They will eventually realise you were right. And if they don't, you didn't want them representing your work anyway.

The Obligation

You have an obligation to keep learning. This field moves with terrifying speed. What I taught five years ago, I sometimes contradict today. That is not hypocrisy; that is the scientific method. If you are doing the exact same things in the exact same way you did them in 2020, you are falling behind.

You must read. You must attend conferences. You must look at your own complication rates and have the humility to admit when your technique needs to change. The moment you think you know everything about the face is the moment you become a danger to the public.

The Trust

Never forget what is happening in that chair. A human being is trusting you with their face. Their self-image. Their confidence. They are letting you alter the way the world perceives them, and more importantly, the way they perceive themselves when they look in the mirror at 6:00 AM. That is not a transaction. That is a profound responsibility.

I told you at the beginning of this book how I came into this work, and I am not going to tell it again. But there is one part I left out of the preface, because it belongs here.

When you are lying in an ITU bed and a nurse is adjusting your potassium drip and you are genuinely uncertain whether you will be alive in the morning, your entire value system gets stripped down to its foundations. All the noise falls away.

The career anxiety. The competitive nonsense. The obsession with being the best injector in the room. Gone. What remains is very simple: the people you love, the work that actually matters, and the question of whether you have done anything that will outlast you.

I thought about my son. He was young enough that he wouldn't remember this if it went badly. That thought was worse than any of the physical pain.

I thought about the patients I had treated. Not the impressive cases. The ordinary ones. The woman who cried in my consultation room because she hadn't recognised herself in the mirror for three years. The man who came in

quietly, embarrassed, and left looking like himself again. The small, private moments of restoration that nobody posts on Instagram because they are too real and too personal for that.

That is what the work is. Not the dramatic transformations. The quiet ones.

The Aftermath

Recovery from something like that is not linear. The body heals faster than the mind. The marriage didn't survive it — these things rarely do, and I am not going to pretend that wasn't its own kind of grief. The future I had planned, the version of my life I had been building towards, was gone. The surgical career I had trained for was gone. The certainty was gone.

What I had was a practice, a son, and a body that had been rebuilt from the cellular level up. Literally. The stem cells that were transplanted into me were not my original cells. In a very real biological sense, I am a different person than I was before the diagnosis. My immune system was rebuilt from scratch.

I find that useful to think about. Not in a mystical way. In a practical way. If the body can be rebuilt from its foundations, so can everything else.

The practice I built after that was different from the one before. More considered. Less driven by ego. More interested in the long game — in building something that would still be standing in twenty years, that would have taught people who would go on to teach other people, that would have changed the standard of care in this small, strange, underestimated specialty.

This book is part of that.

What I Actually Want You to Take Away

I have given you forty-odd chapters of anatomy, technique, product science, and clinical logic. Most of it is useful. Some of it you will disagree with. Good. The day you stop disagreeing with things is the day you stop thinking.

But if I could reduce everything in this book to a single sentence, it would be this:

Treat the person, not the feature.

The feature — the fold, the hollow, the line — is just the presenting complaint. The person sitting in your chair has a history, an identity, a relationship with their own face that predates your involvement by decades. Your job is not to correct a deviation from an aesthetic ideal.

Your job is to help them look like themselves, but rested. Like themselves, but well. Like themselves, but the version they feel on the inside.

That requires listening. It requires restraint. It requires the courage to say no when no is the right answer. And it requires the humility to keep learning, keep questioning, and keep holding yourself to a standard that has nothing to do with how many syringes you can sell in a day.

The two questions

I ask every patient the same thing, somewhere in the consultation, usually while I am washing my hands and they think we have finished.

When you look in the mirror, do you recognise yourself? Or were you expecting somebody slightly different?

Almost nobody says they recognise themselves. They laugh, or they go quiet, and then they describe a gap — small, persistent, impossible to articulate until somebody asks. That gap is why they are sitting there. Not the fold. Not the line. The line is merely where the gap has become visible enough to point at.

And of my older patients I ask a second question.

When you dream about yourself, how old are you?

Nobody has ever needed time to answer. They smile — all of them smile, and I have never worked out why that should be so consistent — and they give me a number. Thirty-two. Twenty-eight. Forty, the year everything was working. Never their actual age. Not once in twenty years.

So somewhere in each of us there is a version that the mind decided was definitive and has quietly refused to revise. It is the one that appears in dreams. It is the one we expect in the glass every morning, and every morning it is not quite there.

That is the whole of it. That is what fifteen years of asking people why now eventually produced. We are not in the business of making people young, or beautiful, or noticeable. We are in the business of narrowing the distance between the person somebody dreams they are and the person the mirror hands back.

The image runs the show

I want to widen that out, because it is not only true in a clinic. It is, as far as I can tell, true of everybody, about everything.

People do not act on their circumstances. They act on the image they hold of themselves, and that image is often years out of date and almost impossible to shift.

You see it everywhere once you are looking. The person who loses weight and gains it back, not through weakness but because in their own internal picture they are still the larger version, and the body is quietly obedient to the picture. The friend of mine who never entered a single race in his life but described himself, without irony, as a runner — and so he ran, every day, for decades, because that is what the man in his self-image did.

Change the behaviour without changing the picture and the picture wins, every time, given long enough.

Which brings me to my own.

I smoke. Cigarettes, cigars, whatever is to hand — anyone who follows me online has watched me do it, and a good number of my patients have raised an eyebrow about it across the consultation chair, which is entirely fair. I am a doctor. I came back from cancer twice. I know, in more anatomical detail than most, precisely what I am doing, and I do it anyway.

I have thought about why, and the honest answer is the subject of this chapter. It is not that I do not understand the risk. It is that somewhere in the image I carry of myself, I am a smoker. It is filed under identity rather than habit, next to living slightly too close to the edge and never quite learning another way to live, and identity is the one thing a health warning has never once managed to reach.

I can frighten myself thoroughly and it changes nothing, because fear operates on behaviour and the smoking is anchored a level below that, in the picture.

I am not going to stand at the end of my own book and tell you I have resolved it. I have not. I am telling you it is there, unresolved, because it would be dishonest to spend forty chapters explaining that people are governed by a self-image they cannot see, and then imply that I am somehow exempt.

I am the clearest example in the book. I simply happen to be the one holding the pen.

I say it for a clinical reason as much as a confession. The next time you have a patient who cannot lose the weight, or cannot stop the thing that is hurting them, or keeps returning to a version of themselves that the evidence retired years ago — do not mistake it for a failure of willpower, and do not treat it as

one.

You are not arguing with a decision. You are arguing with a self-portrait, and it was hanging on the wall long before you walked into the room.

The last thing

I want to tell you when I understood that, because it was not in a clinic. After the transplant, my immune system was rebuilt from cells that had not originally been mine. That is not a metaphor; it is what the procedure is. And when my hair grew back it came back black — I had been going grey for years, and what returned was the hair I had at thirty. The universe has a peculiar sense of humour.

I remember standing in a bathroom some weeks after I came home, at the point where you are well enough to be upright and not yet well enough to be anything else, and looking at a man in the mirror who was thinner than me, paler than me, and had my hair from a decade earlier. I did not recognise him. I had been expecting somebody else.

And I could not find my way back to the version I was expecting. There was no procedure for it. I stood there doing the thing I had watched a thousand patients do — searching a reflection for a person who was not in it — and for the first time I was on the other side of the chair.

I was not observing the gap between the dreamed self and the reflected one. I was living inside it, and I could not measure it, and I could not close it.

That is why I ask them. Nearly every patient in this book — the model who looked like herself again, Steven not knowing the man in the shop window, the widow whose reflection had stopped belonging to anyone, the woman who had been sold the wrong treatment four times — has at some point told me, in one form or another, that they do not recognise the person in the mirror, or has described the age they return to in their dreams.

Only Eugene never did, and even Eugene was telling me the same thing by refusing to.

I did not draw that out of them by accident, or because I read it in a paper. I ask because I stood in that bathroom and could not answer the question myself, and I have been asking it of other people ever since, the way you keep pressing on a bruise to see whether it still hurts.

It took me a long time to notice what had happened. I had been handed, in the most literal form available, the sentence I would spend the next twenty years hearing from other people in my chair. They had all been telling me the same thing. I had understood none of it. And then my own face said it to me in a bathroom, and I finally heard it.

That is the last lesson, and it is the only one I would insist on. Not because I read it, or observed it, or reasoned my way to it — but because I went through it, and it is the one thing in this entire book I did not learn from anybody else's face.

The face in your chair belongs to somebody who is carrying a version of themselves that you will never see. You cannot photograph it, you cannot measure it, and they will almost certainly not describe it to you unless you ask. Everything else in this book — the anatomy, the planes, the products, the doses, the twenty years of accumulated caution — exists in service of a single quiet act: closing a little of the distance between the two people they are, without ever making them into a third. Do less. Do it better. And ask them about the mirror.

Then get out of the way and let them look.

End of Aesthetic Talk — First Edition

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