# ESSENTIALS OF COSMETIC DERMATOLOGY IN AESTHETIC MEDICINE

CBAM Aesthetic Mastery Series




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Dr. Reza Akef is a highly experienced and respected cosmetic physician, with over 15 years of experience in teaching and practicing cosmetic medicine. He is currently the Assistant Professor at McMaster University and the Chairman of the Canadian Board of Aesthetic Medicine (CBAM), where he shares his knowledge and expertise with other healthcare professionals to provide the best care for their patients.

Dr. Akef is board certified by the College of Family Physicians of Canada in both Family and Emergency Medicine. Additionally, he is deeply involved in interventional pain practice cooperation for the treatment of Migraines, chronic pain, TMJ, Myofascial pain, and Fibromyalgia. He is a leading cosmetic physician and an internationally recognized specialist in non-surgical cosmetic procedures.

Dr. Akef is dedicated to understanding his clients' concerns and creating personalized treatment plans that meet their needs and goals. He is committed to thoughtfully and strategically creating individualized combinations of leading, safe treatments that exceed client expectations. He is also the founder of Angel Gloss spa in Toronto, an established, leading destination for non-surgical cosmetic procedures for individuals seeking leading physicians with proven experience, the latest technology, and cutting-edge techniques.

Dr. Akef is actively involved in multiple training courses, webinars, and conferences all around the world, including prestigious international conferences such as IMCAS, AMWC, and Dubai Derma. He participates as a speaker at these events and is always looking to stay up to date with the latest developments in the field of cosmetic medicine.

**Mohsen Talani** is the Director of CBAM. He has over 18 years of experience teaching anatomy in Canada and internationally to students and healthcare practitioners in different fields of medicine such as physicians, dentists, nurses, chiropractors, and physiotherapists.

After graduating from medical school at a young age, he became a medical doctor in his home country, Iran, and practiced in the field of emergency medicine. While pursuing his Master's thesis in neuroscience at McGill University, he discovered his passion for teaching. Since then, he has been working in the field of medical science education, with a focus on anatomy.

He strongly believes that with the rapidly growing aesthetic industry, all practitioners should have a profound knowledge of skin basics, fat compartments, muscles, blood vessels, and nerves, in order to provide safe and effective care to clients and prevent or minimize post-treatment complications linked to lack of anatomy knowledge.

He is currently training healthcare practitioners who seek to have a deep knowledge of anatomy. He incorporates fun, innovative, and easy-to-understand methods of teaching in all of his classes, guest lectures, and presentations. Additionally, he supervises the cadaver lab dissections and hands-on practicals.



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# *Preface*

Cosmetic dermatology has evolved from a discipline once concerned narrowly with the management of skin disease into a vast and increasingly sophisticated field that now sits at the intersection of medicine, technology, and the human desire for self-confidence. The modern practitioner is expected not only to understand the structure and function of the skin, but to be conversant in laser physics, regenerative medicine, pharmacology, and the careful art of patient assessment — and to know, in any given clinical encounter, which of these tools is the right one to reach for.

This handbook has been written to serve as a compact and clinically useful reference across the six core domains that make up the foundation of contemporary aesthetic practice: acne, scarring, cellulite and adipose disorders, pigmentation, vascular lesions, and hair loss.

Each Part follows the same logical arc that experienced clinicians follow at the bedside — understanding the underlying biology, accurately classifying what is seen, and selecting a treatment plan suited to the individual patient.

The text is written for a mixed audience: medical aestheticians refining their existing knowledge, registered nurses moving deeper into clinical procedures, and physicians who want a structured, single-volume overview of the field. The depth is set deliberately at a level where every chapter is detailed enough to be clinically useful, but compact enough that the whole work can be read in a focused weekend and returned to whenever a particular condition arrives in the clinic.

What this book is not is a substitute for hands-on training, supervised practice, or the manufacturer-specific guidance attached to any device or product.

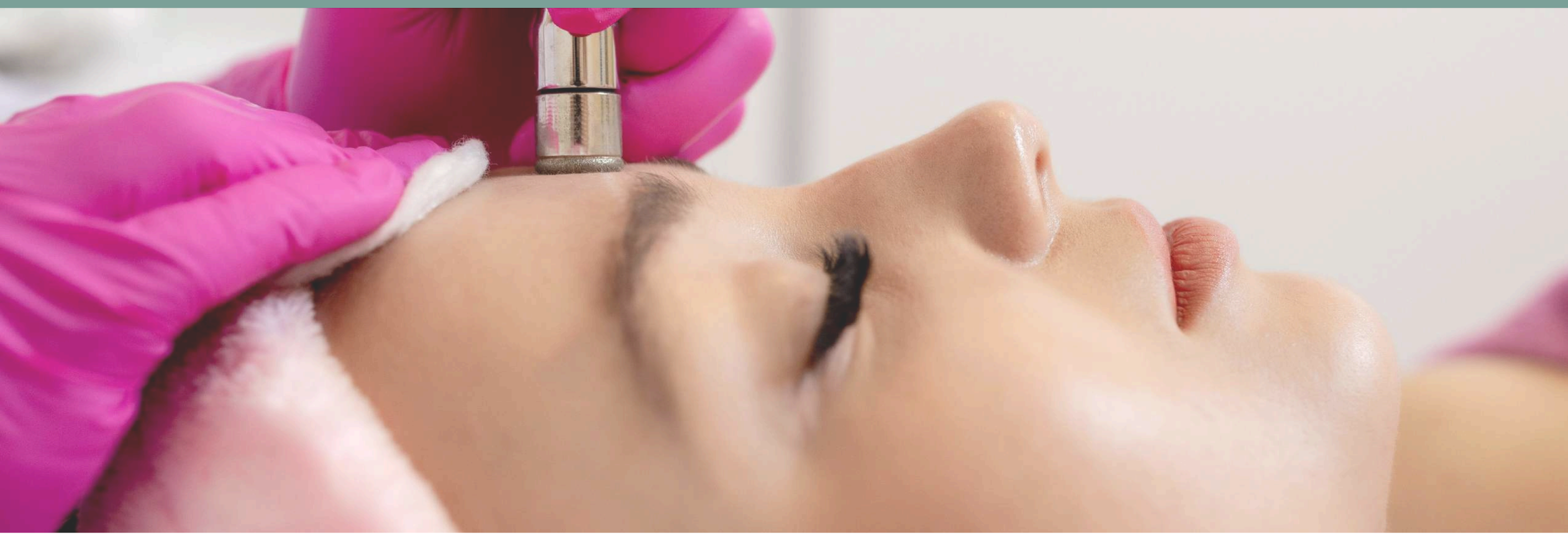
Aesthetic medicine rewards practitioners who couple a strong theoretical foundation with disciplined practical learning. This volume is intended to be the first half of that pairing.

The material here is based on the curriculum of the Canadian Board of Aesthetic Medicine (CBAM) Cosmetic Dermatology Interactive Online Course, restructured and re-presented as a textbook for ease of reference.

The original lessons have been organised into clinical themes rather than weekly modules, so the reader may use the book either sequentially as a structured learning path or selectively as a clinical reference.

It is the author's hope that this handbook will sit on the desks of practitioners as a trusted companion: open often, marked freely, and returned to again and again as the field — and the practitioner — continue to grow.





# *How to Use This Book*

This handbook is organised into six clinical Parts, each devoted to one of the principal domains of cosmetic dermatology practice. Within each Part, the chapters progress from underlying biology and pathophysiology, through clinical assessment, to treatment selection and execution — mirroring the workflow of a real consultation.

For practitioners new to the field, read the book sequentially. Each Part has been written to stand on the shoulders of the one before it, and the cumulative effect is a complete foundation in cosmetic dermatology.

For experienced practitioners, the book is designed for selective use. The Table of Contents and the Appendices have been built so that any single chapter, table, or scale can be retrieved in seconds during clinical hours.

For trainers and supervisors, each chapter closes with a Key Points summary that may be used as the basis for case discussion, oral examination, or self-review.

The following conventions are used throughout:

- **Key Points boxes** at the end of each chapter distill the most clinically important takeaways.
- **Clinical Pearls** highlight practice-tested observations that experienced practitioners learn through experience.
- **Cautions flag** situations in which a treatment choice or technique carries a heightened risk profile.
- **Figure and table** placeholders marked [FIGURE X.Y] and [TABLE X.Y] indicate where diagrams, clinical photographs, or reference tables are to be inserted at the design stage.
- **Cross-references of the form** (see Chapter X) point the reader to related material elsewhere in the book.

- **The appendices** at the end of the book contain forms, scales, and reference tools that have been formatted for practical use — they may be photocopied or adapted for use in the practitioner's own clinic.

**A note on safety:** every clinical recommendation in this book represents general practice as currently taught in the source curriculum. The practitioner is, at all times, responsible for applying their own clinical judgement, deferring to the instructions for use (IFU) of any specific product or device, and complying with the regulatory framework of their own jurisdiction.

# *Part I. ACNE AND ITS MANAGEMENT*



1



# WHY ACNE HAPPENS

Acne is the most common skin condition you will see. It affects about 85% of people between their teens and late twenties, and roughly 1 in 12 is left with permanent scars. For many patients it is not just a skin problem — it damages confidence and mood long after the spots fade.

Acne is a chronic inflammatory disease, and four things have to go wrong together for it to develop.

Once you understand those four things, every treatment in Chapter 3 makes sense — because each one targets one or more of them.

## 1.1 The pilosebaceous unit

Acne always starts in the pilosebaceous unit: the hair follicle, its oil (sebaceous) glands, and a tiny muscle. These open onto the skin through a pore. They cover the whole body except the palms and soles — which is why those areas never get acne.

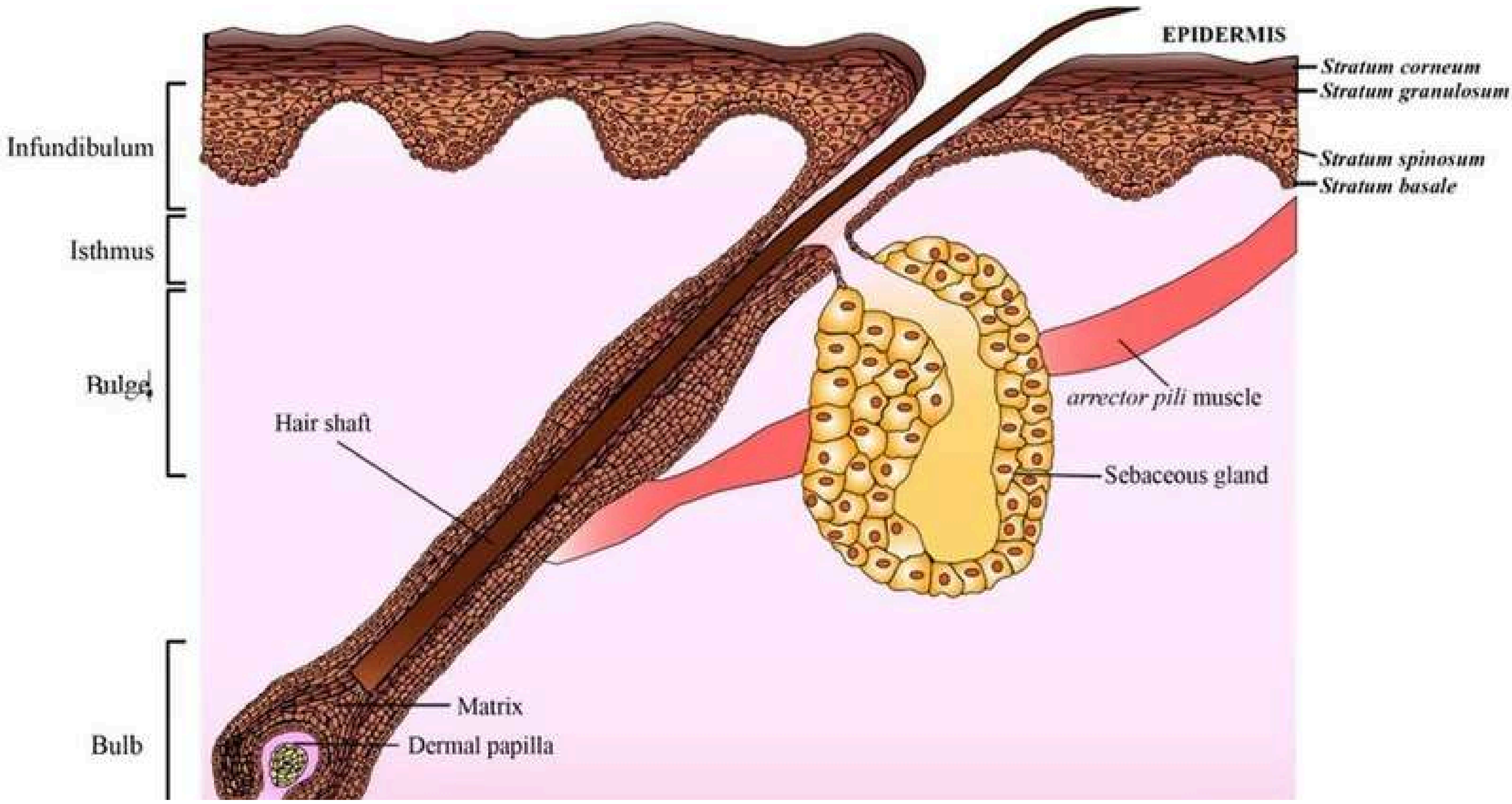



**FIGURE 1.1 — Cross-section of the pilosebaceous unit**

The oil gland makes sebum, which keeps skin and hair from drying out — about 90% of your skin's oil comes from these glands. Oil production is driven by hormones, especially androgens, which is why acne starts at puberty.

Three things about this anatomy explain almost everything that goes wrong:

- The pore can get blocked when the lining cells get sticky and pile up.
- The inside of the follicle — no oxygen, lots of oil — is the perfect home for one bacterium, C. acnes.
- The oil gland isn't passive — it's an active hormone- and inflammation-producing organ.

## 1.2 The four pillars of acne

For acne to develop, four things go wrong at once — and they feed each other, which is why acne is so stubborn and why one treatment is rarely enough.

| # | What goes wrong | What you see | What targets it |
|---|---|---|---|
| 1 | Too much oil — and the wrong kind | Shiny, oily T-zone | Retinoids, isotretinoin, hormonal therapy, low-GI diet |
| 2 | Pore lining gets sticky and clogs | Whiteheads, blackheads, rough texture | Retinoids, salicylic acid, peels |
| 3 | *C. acnes* shifts to a bad strain, forms biofilms | Red, tender spots | Benzoyl peroxide, short antibiotics, light/laser |
| 4 | Immune system overreacts (Th17) | Redness, swelling, dark marks | Anti-inflammatories, dapsone, isotretinoin |

**Table 1.1 — The four pillars at a glance**

## Pillar 1 — too much oil (and the wrong kind)

At puberty, hormones turn up oil production. In acne, two things happen: there's more oil, and its chemical makeup changes (some fats oxidize into inflammatory chemicals). So it isn't just how much oil — it's what kind. That's why controlling oiliness alone often doesn't fix acne.

## Pillar 2 — the pore gets blocked

The lining cells become sticky and form a plug. The first lesion is invisible — a microcomedone — forming weeks before any spot appears. Every acne lesion starts here, which is why treatment must continue even when the skin looks clear.

🔑 **Pearl**

why you keep treating after clearing. Microcomedones form 4–8 weeks before you can see them. Stop the moment skin clears and relapse is guaranteed. Keep a topical retinoid going 2–3 nights a week as maintenance.

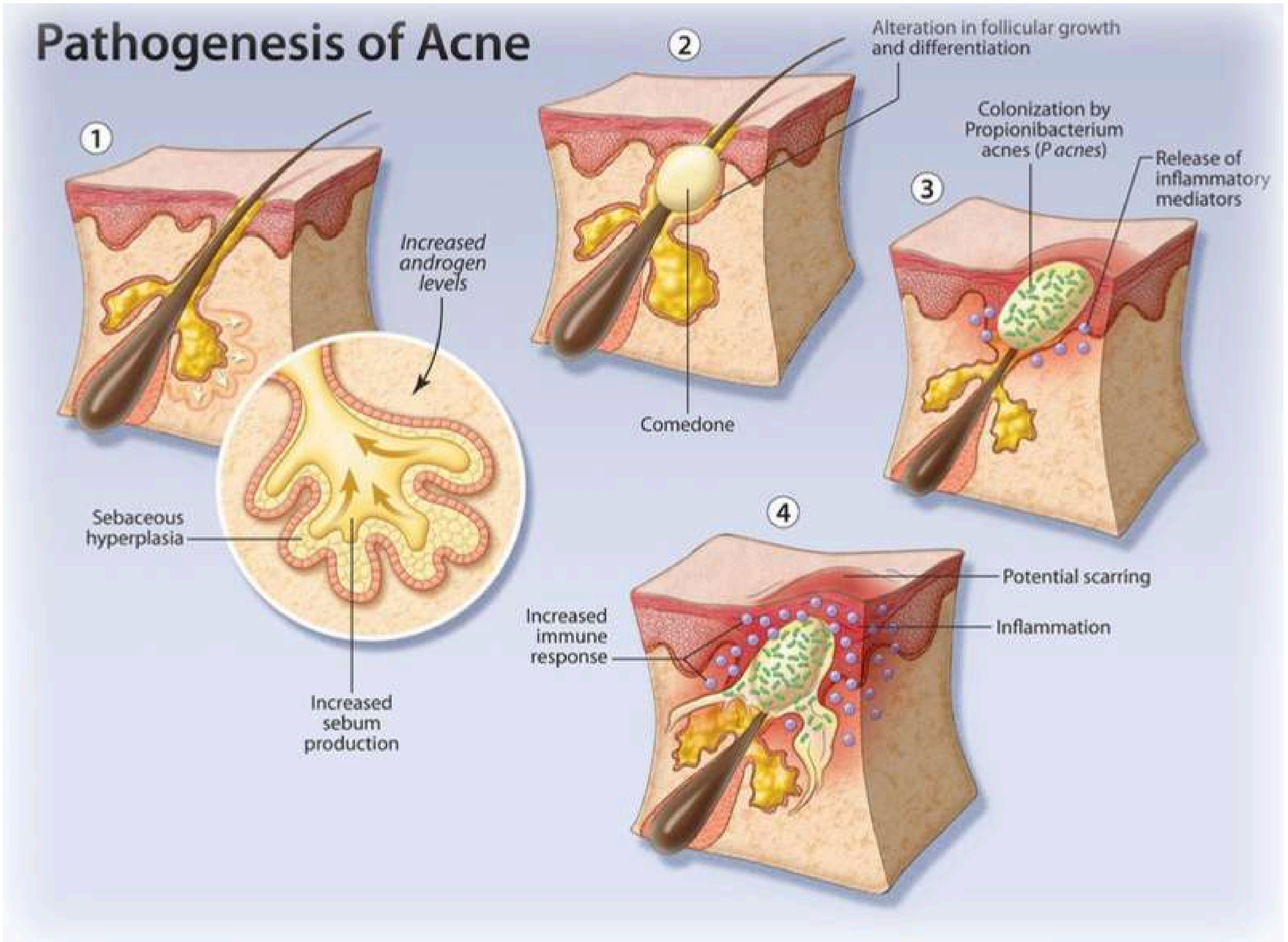
## Pillar 3 — the bacteria shift

C. acnes lives on everyone's skin — even clear faces — making up about 87% of the bacteria in these units. So the problem isn't more bacteria. What changes is: one bad strain takes over, the bacteria form protective biofilms (much harder to kill), and the skin's microbe balance is lost (dysbiosis). This is why long-term antibiotics are discouraged — they worsen the imbalance — and why benzoyl peroxide is the go-to: bacteria can't become resistant to it.

## Pillar 4 — the immune system overreacts

Around the blocked, oily, bacteria-filled follicle, the immune system releases inflammation chemicals — the redness, swelling, and tenderness of each spot. The pattern is called Th17 inflammation, and it explains why anti-inflammatory treatments help even when they aren't killing bacteria. If the follicle bursts, its contents spill into the skin and you get a papule, pustule, or nodule.

How they feed each other: excess oil feeds the bacteria → bacteria trigger inflammation → inflammation makes cells stickier → sticky cells trap more oil. That loop is why combining treatments that hit different pillars (e.g. retinoid + benzoyl peroxide) beats any single one.




## 1.3 Hormones, diet, and leptin

Acne is hormone-driven. Without androgens switching on the oil glands there is no acne — which is why it starts at puberty and tracks with conditions like PCOS.

### Androgens — the original driver

Testosterone and its stronger form DHT enlarge the oil gland and increase oil. Important: most acne patients have normal blood hormone levels — their oil glands are simply unusually sensitive. So routine hormone blood tests are usually pointless, and local anti-androgens (like topical clascoterone) can work without touching body-wide hormones.

### Insulin and IGF-1 — the diet link

These metabolic hormones drive acne: they make the oil gland grow and produce oil, boost androgen action, and trigger inflammation. Acne patients have higher IGF-1, and the higher it is, the worse the acne. Trials show a low-glycemic diet lowers both IGF-1 and acne severity. So “diet matters” is real, not folklore.

| Factor | What it does | What to tell the patient |
|---|---|---|
| High-GI foods (white bread, sugary drinks, sweets) | Spike insulin → IGF-1 → more oil + inflammation | Switch to low-GI: whole grains, vegetables, legumes |
| Dairy (esp. skim milk, whey) | Raises insulin and IGF-1 | Try 6–8 weeks dairy-free if acne is stubborn |
| Whey protein | Strong IGF-1 stimulant | Swap for plant-based protein |
| *PCOS / insulin resistance* | Chronic high insulin → high IGF-1 | Refer for evaluation; consider metformin in selected cases |
| Chocolate, fried/fatty food | Weak or no consistent link | Don’t make patients feel guilty — focus on GI load and dairy |

**Table 1.2 — Diet factors and what to tell patients**

## Leptin — the newest piece

Leptin, a hormone from fat cells, also acts as an inflammation signal in the skin. The oil gland makes its own leptin, which triggers local inflammation, and the same diet pathway (mTORC1) raises it.

This helps explain why obesity, metabolic syndrome, and acne tend to cluster together.(Caveat: blood leptin isn’t consistently raised in acne — its action seems mostly local, at the oil gland itself.)

## 1.4 Genetics

Acne runs in families, and the science backs it: twin studies put heritability at about 78–85%. A 2022 study of over 20,000 patients found 46 genetic locations linked to acne — affecting oil-gland size, pore stickiness, immune reactivity, and follicle structure. In other words, each of the four pillars has a genetic component.

You won't do genetic testing, but this should change how you talk to patients:

- Take the blame off them. Acne is overwhelmingly genetic — not a hygiene failure or a punishment for diet. Say this out loud; they need to hear it.
- Set expectations early. A strong family history of severe acne usually means a more stubborn course. Promise effort, not miracles.
- Treat aggressively when family history is severe — including early isotretinoin if first-line fails. Once scars form, they don't go away.

### Key points

- Acne starts in the pilosebaceous unit (follicle + oil glands + tiny muscle) — everywhere but palms and soles.
- Four things go wrong together: too much/wrong oil, a sticky blocked pore, C. acnes shifting to bad strains and biofilms, and Th17 immune overreaction. They feed each other.
- The problem with C. acnes is the strain shift and biofilms, not quantity — which is why long-term antibiotics are out and benzoyl peroxide is in.
- Androgens start it; insulin and IGF-1 (diet) amplify it; leptin adds local inflammation.
- Acne is ~80% genetic. Severe family history = treat aggressively early, and take the blame off the patient.

### 🔑 Pearl

the most useful intake question. "Did your parents or siblings have acne, and how bad was it?" The answer tells you how aggressive to be, how to set expectations, and helps lift the guilt the patient is probably carrying.

2



# LOOKING AT THE PATIENT

Once you know why acne happens, the next job is to look at the patient and accurately describe what you see. This is where most treatment mistakes start. You need three things at the chair: a vocabulary for the lesions, a way to grade severity, and a checklist to rule out look-alikes.

## 2.1 The lesions

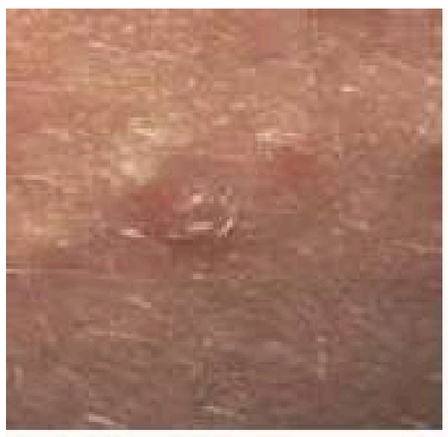
Every lesion starts as an invisible microcomedone, then takes one of two paths:

- Non-inflammatory → stays a comedone (whitehead or blackhead). No redness or pain.
- Inflammatory → papule → pustule → nodule → cyst. Red, sometimes painful, may scar.

EMost patients have both. Which one dominates guides treatment: comedones respond to retinoids and exfoliants; inflammatory acne needs anti-inflammatory and antibacterial agents added in.

### Comedones

- **Closed comedone (whitehead)**: a sealed blocked follicle. Small, smooth, flesh-coloured bump — easier to feel than see. These are where pimples come from, so don't dismiss them.
- **Open comedone (blackhead)**: an open blocked follicle. The dark colour is not dirt — it's oxidized oil and pigment, like a cut apple browning. Telling patients this stops them over-washing (which worsens acne). Open comedones extract easily; closed ones should be opened with a retinoid over weeks, not squeezed.


(a) Whitehead


(b) Blackhead


(c) Papules


(d) Pustules


(e) Nodules


(f) Cysts

## Inflammatory lesions

These are a comedone after C. acnes multiplies inside it and the immune system attacks.

- **Papule**: small, red, solid bump (no pus), under 5 mm. Papules and pustules make up about 90% of inflammatory lesions.
- **Pustule**: a red bump with a visible white/yellow pus head, under 5 mm. The pus is mostly the body's own immune cells, not bacteria — which is why squeezing rarely helps and often makes it worse.

> 🔑 **Pearl**
>
> say this to patients. "That white head is your own immune cells, not infection. Squeezing it pushes inflammation deeper and risks leaving a mark. Leave it — the treatment will clear it in a few days."

- **Nodule**: a large (>5 mm), deep, solid, painful lump. A sign of severe acne; it almost always scars if untreated, and it's the threshold for considering isotretinoin.
- **Cyst**: a deep, fluid- or pus-filled lesion, softer than a nodule. Almost always scars. Multiple nodules and cysts together = nodulocystic acne — the most severe common form and a clear indication for systemic therapy.

## Secondary lesions — the marks left behind

Active acne is only half of what you'll see. The marks left behind are often the bigger cosmetic concern.



| Mark | What it is | Reversible? | First-line |
|---|---|---|---|
| Post-inflammatory erythema (PIE) | Persistent redness where spots were | Yes (weeks–months) | Sun protection; vascular laser if it lingers |
| Post-inflammatory hyperpigmentation (PIH) | Dark marks; more common in deeper skin | Yes, but slo | Sun protection; brighteners (azelaic acid, niacinamide, hydroquinone) |
| Atrophic scars | Lost tissue: ice-pick, boxcar, rolling | No — permanent | Microneedling, MRF, fillers, lasers (Ch 5) |
| *Hypertrophic / keloid scars* | Raised excess scar tissue | No — permanent | Steroid injections, silicone, lasers, surgery |

**Table 2.1 — What's left after the spot heals**

🔑 Pearl

examine systematically. Use bright, angled light and go in order: forehead → nose → cheeks → chin → jawline → neck → chest → upper back. For each area, count comedones, papules, pustules, nodules, marks, and scars. A careless glance loses the patient's trust.

## 2.2 Grading severity

Grading matters because it sets the treatment: mild → topicals; moderate → topicals plus maybe an oral; severe → almost always systemic. Of the many published systems, you need two.

## IGA — your everyday tool

The Investigator's Global Assessment is a fast 5-point scale (0–4) used in most modern trials. Look at the whole face and assign one number.

| Grade | Severity | What you see | Treatment level |
|---|---|---|---|
| 0 | Clear | No lesions; maybe leftover marks | Maintenance only |
| 1 | Almost clear | Rare comedones; ≤1 small inflammatory lesion | Topical retinoid; maintenance |
| 2 | Mild | Some comedones + a few papules/pustules; no nodules | Retinoid + benzoyl peroxide |
| 3 | Moderate | Many comedones; some inflammatory lesions; ≤1 small nodule | Topicals + oral antibiotic OR hormonal therapy |
| 4 | Severe | Many lesions; a few nodules | Consider isotretinoin; refer |

**Table 2.2 — IGA acne severity scale**

## GAGS — for detailed documentation

The Global Acne Grading System is more granular and useful for tracking change over time. It splits the face and trunk into six regions, scores each on its worst lesion, and multiplies by a weight:

- Forehead ×2 · each cheek ×2 · nose ×1 · chin ×1 · chest + upper back ×3

Score per region: 0 = none, 1 = comedones, 2 = papules, 3 = pustules, 4 = nodules. Multiply each by its weight and add.

| Total score | Severity |
|---|---|
| 1–18 | Mild |
| 19–30 | Moderate |
| 31–38 | Severe |
| >38 | Very severe |

🔑 **Pearl**

IGA every visit, GAGS at key moments. IGA is fast enough for every visit. Use GAGS at baseline and before big decisions (like starting isotretinoin) so you have an objective number to compare against later.

🔑 **Pearl**

the one question that changes everything. Ask: "On a scale of 0–10, how much is your skin affecting your daily life?" If they say 7+ while their acne looks mild, you are not treating mild acne — you are treating a patient in distress. Match your urgency to the patient, not the score.

### What both scales miss

Both only count active lesions — not scarring, pigment changes, distribution, or psychological impact. So the grade alone never sets the plan. A patient with "mild" acne who is scarring and distressed may need far more than the number suggests.

## 2.3 Differential diagnosis

Not every red bump is acne — and treating a look-alike as acne fails, often making it worse. The single most useful question is:
"Are there comedones?"

Comedones are the defining feature of true acne. See them, and you're almost certainly looking at acne. Don't, and you should seriously consider something else.

| Condition | Comedones? | How to tell, and why it matters |
|---|---|---|
| Rosacea | No | Central-face redness, flushing, visible vessels; triggered by heat, alcohol, spice. Retinoids and BPO can flare it — different treatment entirely. |
| Perioral dermatitis | No | Small uniform papules around the mouth (sparing the lip border); often from topical steroid use. Stop all topical steroids. |
| Folliculitis (bacterial, Malassezia, Demodex) | No | Uniform papules/pustules. Malassezia favours forehead/chest and itches. Acne treatments don't work — needs antibiotic, antifungal, or ivermectin. |
| *Drug-induced acneiform eruption* | No (usually) | Sudden, uniform lesions all the same stage, tied to a new drug (steroids, lithium, EGFR inhibitors, isoniazid, anticonvulsants). Stop the drug if possible. |

**Table 2.3 — Acne look-alikes**

Also keep in mind: sebaceous hyperplasia (yellow umbilicated papules in older adults), milia (tiny keratin cysts), keratosis pilaris ("chicken skin" on arms/thighs), and hidradenitis suppurativa (deep recurrent nodules in armpits/groin — needs specialist care).

## When to suspect a hormonal cause

Even when it is acne, investigate further (and consider endocrine referral) when you see: adult-onset acne in a woman with no teenage history, pre-menstrual jawline/chin flares, irregular periods, excess facial/body hair,

dark velvety neck patches (acanthosis nigricans), female-pattern hair loss, or resistance to standard treatment. These point toward PCOS or insulin resistance.

### 🔑 Pearl

the chairside algorithm. Look for comedones (yes = acne). Check distribution (central face + flushing = rosacea; around mouth = perioral dermatitis; armpits/groin = HS). Ask: uniform or mixed? Acne is mixed; most look-alikes are uniform. Ask about new medications.

- Pustules contain immune cells, not bacteria. Squeezing spreads inflammation and causes scars.
- Secondary marks (PIE, PIH, scars) are often the bigger concern — address them in every plan.
- Use IGA (0–4) every visit; GAGS for documentation. Both miss the psychological impact — always ask how it affects daily life.
- In differential diagnosis, ask "Are there comedones?" A uniform eruption is almost never acne.

### Key points

- All lesions begin as an invisible microcomedone, then take a non-inflammatory path (comedone) or an inflammatory one (papule → pustule → nodule → cyst).

3



# TREATING ACNE

Everything in the first two chapters leads here. You know why acne happens and how to grade it. Now you choose the plan.

Modern acne treatment follows one rule, set out in the 2024 American Academy of Dermatology (AAD) guidelines: combine treatments that work in different ways. One drug fixes one of the four problems from Chapter 1. Real acne needs you to hit two, three, or all four at once — so you combine from the very first visit.

## 3.1 The plan at a glance

Old acne treatment was a ladder: try one thing, wait, climb to the next. The 2024 guidelines threw that out. Now you start with a combination and match it to severity.

Five rules that guide every prescription:

- Combine treatments. A retinoid + BPO is the default for almost everyone.
- Never give an antibiotic alone. Always pair it with BPO to stop resistance.
- Keep oral antibiotics to 3–4 months, then switch to a topical for maintenance.
- Treat scarring or real distress as severe — even if the spots look mild.
- Plan maintenance from day one. Acne is chronic.

| Severity | First-line | Add if needed | Notes |
|---|---|---|---|
| Mild | Topical retinoid + benzoyl peroxide (BPO) | Topical antibiotic (clindamycin) if inflammatory | Avoid antibiotic alone. Combination products help adherence. |
| Moderate | Retinoid + BPO + topical antibiotic | Oral antibiotic (doxycycline) OR hormonal therapy (women) | Limit oral antibiotics to 3–4 months. Always with BPO. |
| Severe | Consider / refer for oral isotretinoin | Bridge with oral antibiotic + topical combination | Scarring or major distress = treat as severe, whatever the lesion count. |
| *Adult women* | Topical regimen + spironolactone ± combined pill | Isotretinoin if it fails | Hormonal therapy beats oral antibiotics in this group. |

**Table 3.1 —What to prescribe by severity**

> 🔑 **Pearl**
>
> the 8–12 week talk. Tell every patient at the first visit: “This takes 8–12 weeks to really work, and your skin may look worse for the first few weeks. That’s normal. Stopping early is the main reason treatment fails.” This one conversation does more for adherence than any drug.

## 3.2 Creams and gels — the foundation

Topicals are the base of every plan: alone for mild acne, alongside tablets for moderate-to-severe.

### Retinoids — the cornerstone

If you remember one thing: a retinoid belongs in almost every plan. It works on three of the four problems and stops new microcomedones forming — which is why it’s the one drug you keep using for maintenance. How to start: begin low (adapalene 0.1% or tretinoin 0.025%), 2–3 nights a week for two weeks, then nightly if tolerated. Pea-sized amount for the whole face, on dry skin, at night. Always pair with a moisturizer and daily SPF. Warn about “purging” (a flare in weeks 2–4 — that’s it working). Avoid in pregnancy; use azelaic acid instead.

| Drug | Use for | Key practical points |
|---|---|---|
| Retinoids (adapalene, tretinoin, tazarotene, trifarotene) | Every plan — all acne types | Apply at night, pea-sized, on dry skin. Start low, build up. Expect a flare in weeks 2–4. Avoid in pregnancy. |
| Benzoyl peroxide (BPO) | All inflammatory acne | Bacteria can’t resist it. Always add it to any antibiotic. 2.5% works as well as 10%. Bleaches fabric — use white. |
| Topical antibiotic (clindamycin) | Inflammatory acne | Never alone — always with BPO, usually as one combined gel. |
| *Clascoterone 1% cream* | Moderate inflammatory acne (men or women) | First topical anti-androgen. Twice daily. No body-wide hormone effects. |
| *Azelaic acid* 15–20% | Acne with dark marks; pregnancy | Slow (8–12 weeks) but safe in pregnancy and great for darker skin. |

**Table 3.2 — Topical treatments at a glance**

## Benzoyl peroxide — the antibiotic's bodyguard

BPO kills C. acnes, and bacteria can't become resistant to it. The single most important rule in acne prescribing: whenever you give any antibiotic, give BPO with it. 2.5% is as effective as 10% with less irritation. Warn patients it permanently bleaches coloured fabric.

# 3.3 Tablets (systemic treatment)

When creams aren't enough. Three options: oral antibiotics, hormonal therapy (women), and isotretinoin.

## Oral antibiotics

At acne doses these mostly calm inflammation rather than kill bacteria — which is why they help even when C. acnes is resistant.

| Drug | Typical adult dose | Notes |
|---|---|---|
| Doxycycline | 50–100 mg once or twice daily (or 40 mg modified-release) | First choice. Take with food, not dairy. Watch for sun sensitivity and stomach upset. |
| Minocycline | 50–100 mg twice daily (or 1 mg/kg ER once daily) | More side effects: pigmentation, dizziness, rare serious reactions. Second choice. |
| Sarecycline | 60–150 mg once daily (by weight) | Narrow-spectrum, gentler on the gut microbiome. More expensive. |
| *Erythromycin / azithromycin* | Varies | Keep for pregnancy or tetracycline allergy. High resistance. |

**Table 3.3 — Oral antibiotics and doses**

Three rules: always add BPO; stop at 3–4 months; choose doxycycline over minocycline (safer, equally effective).

## Hormonal therapy (women)

Excellent for adult women — especially jawline-and-chin acne that flares before periods or comes with PCOS.

- Spironolactone — 50–100 mg daily (PCOS may need 150–200 mg). The 2024 FASCE trial showed it beat doxycycline for moderate female acne. Routine potassium checks are no longer needed in healthy young women. Avoid in pregnancy.
- Combined oral contraceptives — lower androgens; take 3–6 months for full effect. Avoid with a history of clots, migraine with aura, or smokers over 35.

Used together they work well and are the go-to for adult women who also want contraception.

## Isotretinoin — the only "cure"

The most powerful option, and the only one that can give lasting remission, because it hits all four problems at once.

- Use for: severe acne, scarring, major distress, or acne that keeps failing or relapsing.
- Dose: 0.5–1.0 mg/kg/day with fatty food. Aim for a total of 120–150 mg/kg over about 5–7 months. Low-dose (0.25–0.4 mg/kg/day) is gentler and works, but may relapse more often.
- Side effects (almost everyone): dry lips and skin, a possible flare in weeks 2–6, muscle aches, raised triglycerides and liver enzymes. The old depression link has not been confirmed in modern studies — but ask about mood anyway.
- Monitoring: baseline lipids, liver tests, and pregnancy test; recheck lipids and liver at 1–2 months — if normal, stop routine bloods. Monthly pregnancy tests are mandatory.

**🔑 Pearl**

spot hormonal acne. Adult woman + spots on the jawline and chin + flares before periods + irregular cycles = hormonal acne. Start spironolactone — she'll often need no antibiotic at all.

**⚠ Pregnancy — absolute warning.**

Isotretinoin causes severe birth defects from even a single dose, and miscarriage. There is no safe dose in pregnancy. Two forms of contraception are required during treatment and for one month after stopping. Counsel verbally at every visit — not just paperwork.

## 3.4 Procedures (helpful add-ons)

The 2024 guidelines say the evidence isn't strong enough to recommend procedures for or against. So use them to support medical treatment, not replace it.

- Chemical peels: best evidence for salicylic acid 20–30% (good for oily, inflammatory skin) and glycolic acid 30–70%. A course of 4–6 peels, 2–4 weeks apart. Mandelic acid is safer for darker skin.
- Light and laser: blue light (415 nm) kills C. acnes; red light (633 nm) calms inflammation; combined is better. Photodynamic therapy for stubborn cases; pulsed dye laser for leftover redness.
- Comedone extraction: do it after 4–6 weeks on a retinoid, which softens comedones and makes it easier.

**🔑 Pearl**

when a patient asks for a "facial" or laser. Say: "These help, but the foundation is your daily cream and, if needed, tablets. Procedures speed things up — they don't replace the basics."

## 3.5 Maintenance and the patient conversation

The most skipped step, and the biggest cause of failure. Acne is chronic: stop everything when the skin clears and it comes back within weeks, because microcomedones keep forming underneath.

After clearing, simplify:

- Keep the retinoid 3–5 nights a week — the single most important step.
- Keep BPO 2–3 times a week if the acne was inflammatory.
- Stop oral antibiotics.
- Continue hormonal therapy if that's what worked.
- Keep daily sunscreen and moisturizer.

After isotretinoin, reintroduce a retinoid 1–2 months later if the disease was significant.



| Topic | what to say |
|---|---|
| Timeline | “Expect 8–12 weeks before real improvement. The first few weeks may look worse — that’s normal.” |
| Diet | “Cut back on sugary foods and drinks. If your acne is stubborn, try 6–8 weeks off dairy.” |
| Skincare | “Wash gently twice a day. No harsh scrubs or hot water. Use a fragrance-free moisturizer and SPF 30+ daily.” |
| Picking | “Picking is the number-one cause of scars and dark marks. Hands off.” |
| *Adherence* | “Use it exactly as prescribed. Skipping nights makes it work slower.” |
| *Reassurance* | “Acne is mostly genetic. It’s not from being dirty or eating chocolate. You didn’t cause this.” |

**Table 3.4 — What to tell every patient**

Don’t miss the mental health side. Acne is strongly linked to anxiety and depression, and mild-looking acne can cause severe distress. Ask at every visit: “How is your skin affecting your mood and daily life?” If there’s real distress, treat more firmly and offer support.

## Key points

- Combine treatments from the first visit. Default = retinoid + BPO.
- Never give an antibiotic without BPO. Limit oral antibiotics to 3–4 months.
- Adult women with jawline/chin acne: spironolactone 50–100 mg daily, first-line.
- Isotretinoin 0.5–1.0 mg/kg/day (total 120–150 mg/kg) is the only cure — for severe, scarring, or failed acne. Strict pregnancy prevention.
- Procedures are add-ons, not replacements.
- Maintenance is mandatory: retinoid 3–5 nights a week. The 8–12 week talk is your highest-yield five minutes.

# *Part II. Scars and Their Management*



4



# UNDERSTANDING SCARS

A scar is the body's record of an injury that healed. The skin you're born with is unique tissue; once it's gone, the body can't make an exact copy — so it patches the gap with fibrous tissue. That patch is the scar. Different scars are just different ways the healing went, and knowing which one you're looking at is what lets you treat it well.

## 4.1 How skin heals — and where scars come from

Skin has two layers that matter here: the epidermis (outer, regenerates endlessly) and the dermis (deeper, holds collagen, vessels, and glands). The single most important rule in scar management:

Injury that stays in the epidermis heals without a scar. Injury that reaches the dermis always scars.

The epidermis regrows from its base; the dermis can't rebuild its organized matrix, so it lays down a fibrous patch instead. That's why a sunburn heals clean but a deep cut leaves a line — and why inflammatory acne scars while comedones usually don't. Every wound heals through four overlapping phases:

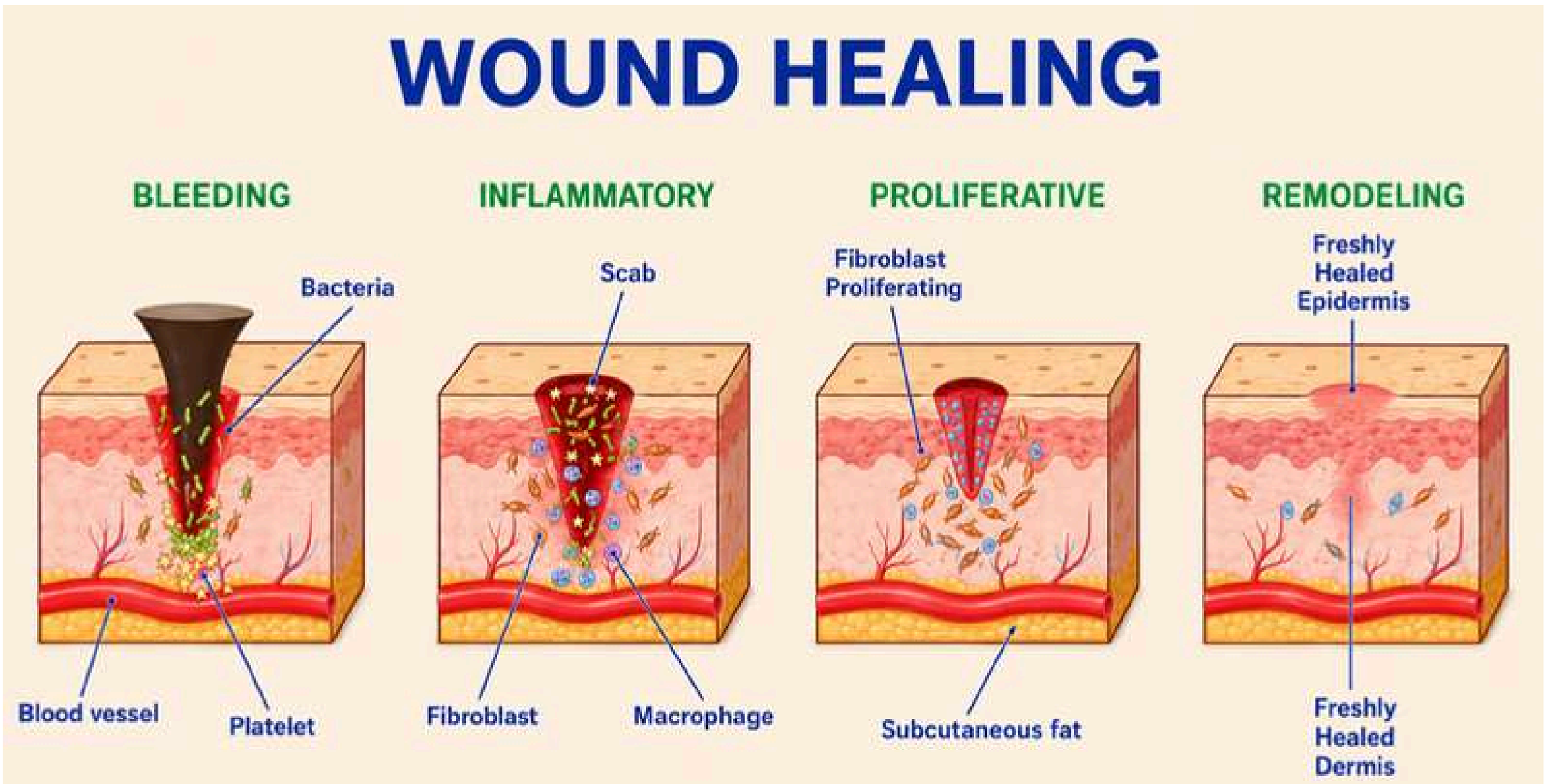



**FIGURE 4.1 wound healing**

| Phase | Timing | What happens |
|---|---|---|
| 1. Hemostasis | Seconds–minutes | Vessels constrict; a clot stops the bleeding |
| 2. Inflammation | Hours–days (peak 2–3 days) | Immune cells clear debris and bacteria |
| 3. Proliferation | Day 3–3 weeks | New vessels form; fibroblasts lay down immature (type III) collagen; the wound closes |
| 4. Remodeling | 3 weeks–2 years | Type III collagen is replaced by stronger type I; the scar fades and softens. Final strength ~80% of normal skin |

**Table 4.1 — The four phases of wound healing**

**🔑 Pearl**

why scars go red, then pale. A new scar is red from all the new blood vessels, which regress over months. Never judge a scar's final look before 12 months — and tell patients that, because the bright-red stage panics them.

Where it goes wrong: the scar type tells you how the four phases were disrupted —

- Too little inflammation/collagen → atrophic (depressed) scar
- Too much → hypertrophic or keloid (raised) scar

What changes the outcome (and most are modifiable — the basis of prevention): age, skin type (Fitzpatrick IV–VI scar and pigment more), family history of keloid, smoking, diabetes; wound depth, location (high-tension areas like chest, shoulders, jawline scar worse), infection; and how the wound was closed and protected.

## 4.2 Traumatic scars

The simplest group: any external injury that reached the dermis — cuts, scrapes, burns, bites, piercings, surgical incisions.

| Subtype | Looks like | Approach |
|---|---|---|
| Linear hypopigmented | Pale, thin line (often surgical) | Often cosmetically acceptable; resurface if needed |
| Widened (atrophic) | Stretched, flat, depressed | Excision and re-closure ± Z-plasty; fillers |
| Hyperpigmented | Darker than surrounding skin | Sun protection; brighteners; gentle laser |
| Contracture | Tight, raised, restricts movement | Refer to plastic surgery; pressure garments |
| Traumatic tattoo | Embedded pigment (e.g. asphalt) | Q-switched laser; early debridement prevents it |

**Table 4.2 — Traumatic scar subtypes**

When you assess one, ask: How old? (scars remodel for up to 2 years — don't treat aggressively in the first 12 months). Where? (tension areas scar worse). Skin type and family history? (both raise hypertrophic risk). Any contracture or functional impact? (that's medical, not cosmetic — refer to plastic surgery).

> 🔑 **Pearl**
>
> why scars go red, then pale. A new scar is red from all the new blood vessels, which regress over months. Never judge a scar's final look before 12 months — and tell patients that, because the bright-red stage panics them.

## 4.3 Hypertrophic and keloid scars

When healing tips toward too much collagen, you get a raised, firm scar. Telling these two apart matters because they behave very differently.



| Feature | Hypertrophic scar | Keloid |
|---|---|---|
| Boundaries | Stays within the original wound | Extends beyond it |
| Onset | Within weeks | Months–years later (sometimes spontaneous) |
| Natural course | Usually flattens over 1–2 years | Persists or grows; doesn't self-resolve |
| Common sites | High-tension areas | Chest, shoulders, upper back, earlobes, jawline |
| Genetics | Any skin type | Strong genetic link; far higher risk in Black patients; family history predicts |
| Treatment response | Generally good | Often poor; recurs after excision |

**Table 4.3 — Hypertrophic scar vs. keloid**

Both are an over-active, prolonged healing response with fibroblasts over-producing collagen, driven mainly by TGF-β (more sustained in keloids). Keloids are now seen as a genetic fibroproliferative disorder, not just a big scar.

Treatment (detail in Chapter 5): first-line is intralesional triamcinolone, often with 5-fluorouracil for keloids; silicone gel/sheets (strongest non-procedural evidence); pressure therapy; pulsed dye laser for redness; Nd:YAG as an adjunct. Excising a keloid alone almost always recurs — always combine with steroid, radiation, or pressure.

**🔑 Pearl**

one question before treating any raised scar. "Does it extend beyond the original injury?" Yes = keloid (treatment is suppression, not cure; recurrence is common). No = hypertrophic (much better outlook).

**🔑 Pearl**

the ear-piercing keloid. After treating an earlobe keloid, the patient must never re-pierce that ear — it will keloid again, larger. Say this explicitly.

## 4.4 Acne scars: types and morphology

The most common — and most varied — scars you'll treat. Inflammatory acne ruptures a follicle into the dermis and destroys tissue.

The repair goes one of two ways: too little collagen → atrophic (depressed) scar; too much → hypertrophic/keloid (less common, usually chest, back, jawline). About 80–90% of acne scars are atrophic, in three subtypes (Jacob classification):

| Subtype | Shape | Looks like | Responds best to |
|---|---|---|---|
| Ice-pick | Narrow (<2 mm), deep, V-shaped | Sharp deep pits, central face | TCA CROSS, punch excision, deep peels. Poor response to laser/microneedling alone |
| Boxcar | Wider (2–4 mm), sharp vertical walls | Chickenpox-like; cheeks, temples | Fractional laser, microneedling, MRF, punch elevation |
| Rolling | Wide (>4 mm), shallow, sloping; tethered by bands | Wavy texture; smooths when stretched | Subcision (releases bands) + filler + RF microneedling |

**Table 4.4 — Atrophic acne scar subtypes**

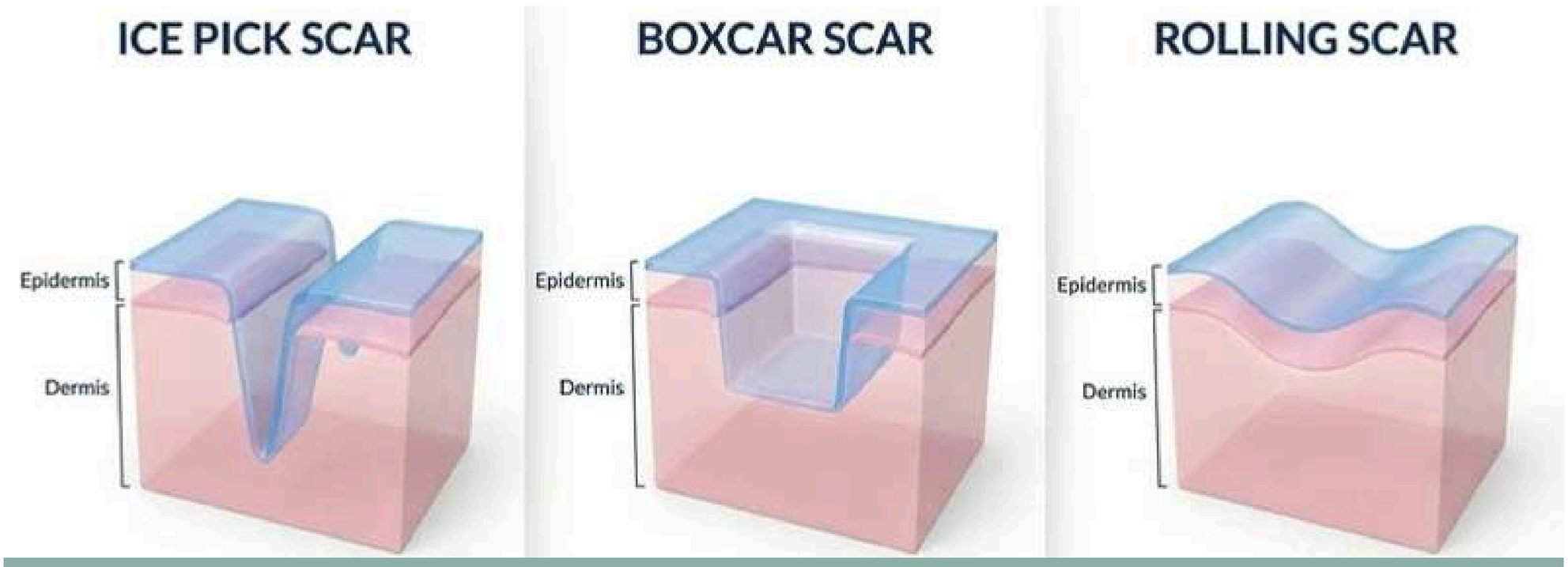



**FIGURE 4.2 — Cross-section of ice-pick, boxcar, and rolling scars**

Mnemonic: Ice-pick = V (narrow & deep); Boxcar = U with vertical walls; Rolling = wavy ground, no sharp edges.

A minority are raised (hypertrophic) — jawline, chest, shoulders, back; more in Fitzpatrick IV–VI or with a keloid history. Treated like other raised scars (steroid, silicone, ± 5-FU).

Why subtype matters: each has a different anatomy, so each needs a different tool. Ice-pick goes deep through a narrow channel — surface lasers miss it (needs TCA CROSS or punch). Boxcar has steep walls and a floor — responds to resurfacing. Rolling is tethered from below — subcision must come first. That's why most patients need a combination, not one modality.

**🔑 Pearl**

diagnose before you treat. Stretch the skin under side-lighting. Scars that smooth out when stretched are rolling (tethered). Scars that stay visible are ice-pick or boxcar. This 30-second test guides the whole plan.

Documenting: the Goodman & Baron scale (grades 1–4) and the ECCA scale are common; in daily practice, standardized photos (front and three-quarter, neutral and stretched) are most useful.

## Key points

- A scar forms whenever injury reaches the dermis; epidermis-only injury heals clean.
- Healing has four phases (hemostasis → inflammation → proliferation → remodeling); maturation takes up to 2 years.
- Hypertrophic scars stay within the wound; keloids extend beyond and are strongly genetic.
- 80–90% of acne scars are atrophic: ice-pick, boxcar, or rolling.
- Subtype determines treatment — one modality won't fix all types in the same patient.
- Most acne-scar patients need combination therapy over 6–18 months.

5



# SCAR TREATMENT MODALITIES

There is no single best scar treatment — the right choice depends on the scar type, the patient's skin type, your equipment, and your training. Four core modalities to know:

## 5.1 Microneedling radiofrequency (MRF) — the modern workhorse

MRF combines two things: tiny needles that create controlled micro-injuries (triggering new collagen) and radiofrequency heat delivered through them (remodeling deeper tissue). Together they do what neither does alone — surface collagen plus deep dermal remodeling, where most scar tissue sits.

Best for: boxcar (good), rolling (good, especially after subcision), ice-pick (modest — combine with other tools).

Insulated vs. non-insulated needles: insulated needles release energy only at the tip, sparing the epidermis — lower risk of dark marks (PIH), so they're the safer default in darker skin. Non-insulated needles heat the whole needle length — stronger surface effect, higher PIH risk.

Practical parameters: needle depth 0.5–3.5 mm (deeper for deeper scars); usually 3–6 sessions, 4–6 weeks apart; downtime 2–5 days of redness and pinpoint scabbing.

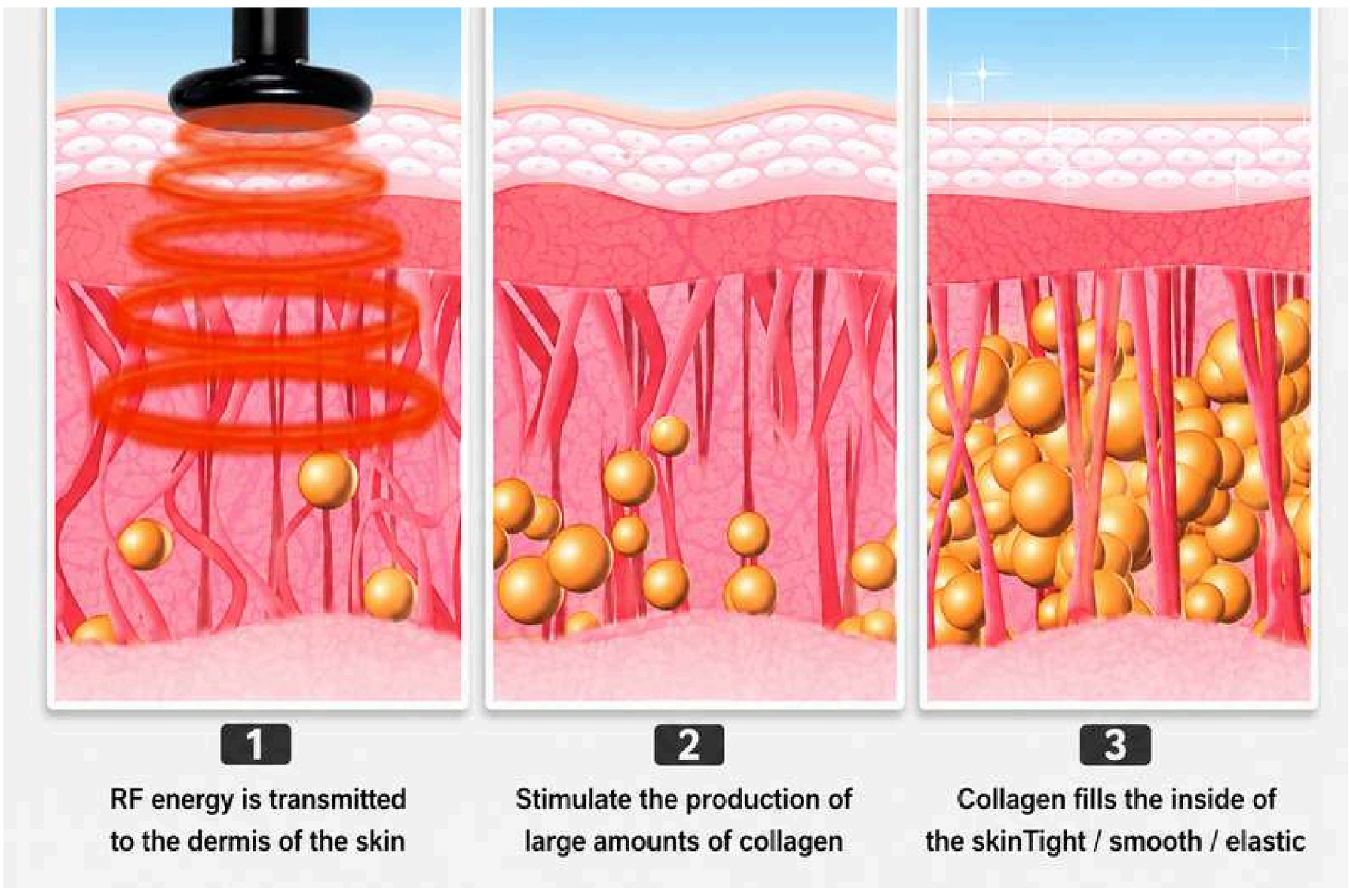



**FIGURE 5.1 — How MRF works**

Contraindications: pregnancy, active infection (treat active acne first), active cold sores (pre-treat if HSV history), implanted electronic devices (relative), keloid-prone (caution, test patch). Recent isotretinoin is no longer an absolute bar, but stay cautious.

> 🔑 **Pearl**
>
> why MRF is replacing $CO_2$ laser. Similar scar improvement to fractional $CO_2$, but less PIH in darker skin, shorter downtime, and safer in skin of colour. For a mixed-Fitzpatrick practice, it's the modern default.

## 5.2 Chemical and laser resurfacing

Resurfacing removes or denatures a controlled depth of skin so the body regrows it. Deeper means a bigger result — but more downtime and more risk.

| Peel | Depth | Use in scars | Notes |
|---|---|---|---|
| Salicylic 20–30% | Superficial | Mild texture + PIH | Penetrates follicles; good for oily skin |
| Glycolic 30–70% | Superficial | Mild scarring + tone | 4–6 sessions; lower strength safer in dark skin |
| Jessner's | Superficial–mid | Scarring + pigment | Well tolerated |
| TCA 20–35% | Medium | Boxcar floors; widespread atrophic | Reaches papillary dermis; PIH risk in dark skin |
| TCA CROSS 50–100% | Focal deep | Ice-pick — procedure of choice | Tiny amount on the scar floor only; "frosting" endpoint; 3–6 sessions |

**Table 5.1 — Chemical peels for scar treatment**

**🔑 Pearl**

TCA CROSS for ice-pick scars. CROSS = “Chemical Reconstruction Of Skin Scars.” A toothpick delivers high-strength TCA (50–100%) into the scar itself, sparing normal skin; healing fills the defect from below. Usually 3–6 sessions, 4–6 weeks apart. The first-line, most evidence-based ice-pick treatment.

| Laser | Wavelength | Type | Downtime | Best for / risk |
|---|---|---|---|---|
| Fractional $CO_2$ | 10,600 nm | Ablative | 1–2 weeks | Deep boxcar, rolling. High PIH risk in Fitz IV–VI |
| Fractional Er:YAG | 2,940 nm | Ablative | 5–7 days | Moderate scarring. Lower PIH than $CO_2$ |
| Non-ablative 1550/1565 nm | Infrared | Non-ablative | 2–3 days | Mild–moderate; skin of colour. Needs several sessions |
| Pulsed dye (PDL) | 585–595 nm | Vascular | None | Post-acne redness; raised scars. Little textural effect |
| Q-switched / pico Nd:YAG | 1064 nm | Pigment | None–1 day | Atrophic scars in dark skin; PIH. Modest texture effect |

**Table 5.2 — Laser resurfacing options**

Match depth to depth: shallow/mild → non-ablative or superficial peels; moderate boxcar → fractional ablative or MRF; deep boxcar floors → focal TCA or punch + resurfacing; ice-pick → TCA CROSS or punch (resurfacing is an adjunct); raised scars → avoid aggressive resurfacing, use PDL/Nd:YAG/steroid.

Don’t stack aggressive resurfacing in one visit. Rough safe intervals: superficial peel → 2 weeks; medium TCA → 4 weeks before laser; fractional laser → 4–6 weeks; ablative $CO_2$ → 8+ weeks.

## 5.3 Injectables and surgical options

### Subcision — essential for rolling scars

A needle or blunt cannula is passed under the scar to cut the fibrous bands tethering it down; the small pocket of blood that forms scaffolds new collagen and lifts the scar. Use an 18G–22G needle or blunt cannula (gentler, less bruising); fan in the subdermal plane; 2–4 weeks apart; average 3–5 sessions before adding surface treatments.

🔑 **Pearl**

subcision comes first. Go straight to MRF or laser on rolling scars without subcision and the bands keep pulling the scars down — looks good on paper, disappointing in the mirror.

### Dermal fillers

Inject under depressed scars to restore volume. Best for pliable rolling and boxcar scars (not ice-pick). HA (common, reversible, lasts 6–18 months); CaHA; PLLA (a biostimulator that grows the patient's own collagen over months). PMMA (Bellafill) is the only filler FDA-approved specifically for acne scars. A bolus under a tethered scar bulges around it, not into it — combine with subcision.

### Punch techniques — for individual deep scars:

| Technique | Tool | Indication | Notes |
|---|---|---|---|
| Punch excision | 1.5–3 mm punch | Deep ice-pick / narrow boxcar | Sutured; trades a pit for a fine line scar |
| Punch elevation | Punch matching scar | Boxcar with acceptable floor depth | Floor lifted to skin level; keeps original colour/texture |
| Punch grafting | Punch + donor (post-auricular) | Larger ice-pick | Texture mismatch is the limitation |

**Table 5.3 — Punch techniques**

Follow punch work 6–8 weeks later with resurfacing of the whole area to blend.

### Intralesional steroid — for raised scars

Triamcinolone acetonide (Kenalog) is the workhorse for hypertrophic and keloid scars. Concentration 10–40 mg/mL (start lower in thin skin or small scars); enough volume to blanch the scar (≈0.1–0.5 mL per cm²); every 4–6 weeks, usually 3–6 sessions. Side effects: telangiectasia, skin/fat atrophy, hypopigmentation, rarely Cushingoid effects at high cumulative doses. For keloids, combine with 5-FU (commonly triamcinolone + 5-FU ≈1:9) — more reliable flattening, fewer steroid effects.

🔑 **Pearl**

inject into the scar, not around it. Steroid in the normal skin around a keloid causes atrophy and depigmentation that may be permanent. Stay within the scar tissue; if you can't get it in, go deeper or pre-soften with cryotherapy or a PDL session.

## 5.4 Preventing scars

The best scar treatment is preventing the scar. Two parts:

Acne-scar prevention (your first job): treat moderate-to-severe or inflammatory acne aggressively and early — consider early isotretinoin in high-risk patients; time spent inflamed is scar substrate. Stop the picking — the single most common preventable cause of acne scars; say it at every visit. Photoprotect. Treat PIH/PIE early (easier in the first 3 months).

| Strategy | When to start | Evidence |
|---|---|---|
| Low-tension wound closure | At the wound | Strong |
| Silicone gel or sheets | 2 weeks post-injury, daily for 3–6 months | Strong |
| Sun protection (SPF 30+) | From re-epithelialization onwards | Strong |
| Pressure garments | Day 7–14, for several months (burns) | Strong (burns) |
| Scar massage | 3–4 weeks post-injury, daily | Moderate |

| Strategy | When to start | Evidence |
|---|---|---|
| Onion extract (Mederma) | Daily for 3 months | Weak |
| Vitamin E | — | Insufficient; may worsen scarring |

**Table 5.4— Evidence based post injury scar prevention**

Two take-homes: silicone is the most evidence-based topical — daily for at least 3 months. Vitamin E is not the magic ingredient many think — it may even worsen scarring; stop recommending it.

**🔑 Pearl**

counsel every post-procedure patient. “For the next 3 months your scar decides whether it’ll be good or bad. Three things tip it: silicone gel daily, sunscreen daily, and no picking. Do those and you’ll get the best scar your body can make.”

**Key points**

- MRF is the modern workhorse for atrophic acne scars — like fractional $CO_2$ but safer in skin of colour.
- TCA CROSS is the procedure of choice for ice-pick scars.
- Subcision is mandatory before resurfacing rolling scars.
- Intralesional triamcinolone (10–40 mg/mL) ± 5-FU is first-line for hypertrophic scars and keloids.
- Fillers restore volume in pliable rolling/boxcar scars; PLLA gives longer biostimulation.
- Punch excision/elevation help individual deep scars.
- Silicone gel or sheets daily for ≥3 months is the best-evidence prevention. Vitamin E is not.

6



# PUTTING IT TOGETHER: TREATING THE WHOLE PATIENT

The earlier chapters treated scars as individual technical problems. Real patients aren't like that. They arrive with a mix of scar types across several areas, often with leftover active acne, pigmentation, and real distress. A plan built around one procedure disappoints them. A plan built around the whole face, the whole patient, and the whole timeline can change their life.

## 6.1 Think in three dimensions

Scars were treated for decades as a surface problem — and the same surface treatment disappointed many patients. The reason is anatomical: acne scars are 3D distortions, with parts at different depths that behave differently. The 3D Rejuvenation model frames every patient in three layers, each needing different tools:

| Dimension | What it is | What lives here | Treatments |
|---|---|---|---|
| 1. Surface (epidermis / superficial dermis) | Texture, tone, surface pigment | PIH, PIE, fine scar walls, photodamage | Peels, retinoids, superficial laser, IPL, shallow microneedling |
| 2. Depth (mid-to-deep dermis) | Collagen, vessels, fibrotic bands | Boxcar walls and floors, dermal loss, rolling-scar tethering | Subcision, fractional ablative laser, MRF, biostimulator fillers, focal TCA |
| 3. Volume (fat and bone) | The deep scaffolding | Fat loss after severe acne, deep tethering, deficits casting shadows | Volumizing fillers (HA, CaHA, PLLA, deep); rarely fat grafting |

**Table 6.1 — The 3 dimensions of scar rejuvenation**

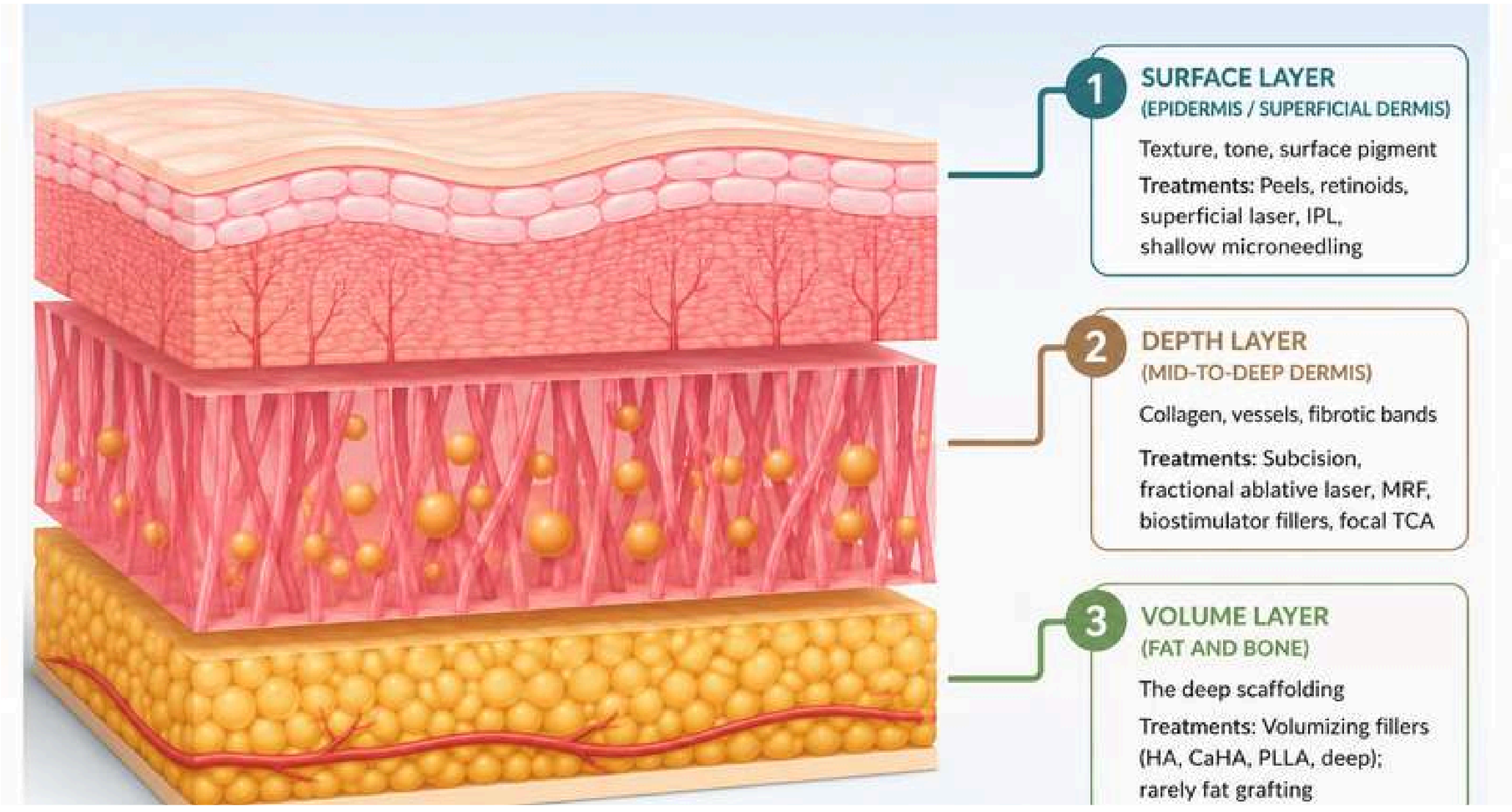



**FIGURE 6.1 — The three-layer 3D Rejuvenation framework**

Why all three: treat only the surface and the rolling scars stay and the cheek still looks hollow; treat only the depth and tethered scars don't fully release without subcision while volume shadows remain. Address all three in sequence — subcision + filler, then MRF/laser, then TCA CROSS for ice-picks, then peels — and you get a transformation no single treatment delivers.

🔑 **Pearl**

diagnose in three dimensions. Examine three times: flat overhead light (surface), side light with the skin stretched (depth/tethering), and head tilted under top light (volume/shadow). Each lighting reveals a different layer.

## 6.2 Combining treatments

Three rules:

- Treat the disease first. Never start scar treatment on active inflammatory acne — new inflammation makes new scars. Stabilize first (may be 3–6 months of topical ± oral therapy).

• Treat deep to superficial: subcision (release tethering) → volume with filler where needed → deep remodeling (MRF, fractional laser, focal TCA) → surface refinement (peels, topicals, gentle laser). Reversing this leaves the real problem untouched.

• Space sessions for recovery. Rough intervals: subcision → MRF/laser 2–4 weeks; MRF or fractional laser between sessions 4–6 weeks; ablative $CO_2$ → next major procedure 8–12 weeks; HA filler → laser over same area 2 weeks.

| Scar profile | Suggested sequence over 6–12 months |
|---|---|
| Mostly rolling + mild boxcar | Subcision ×2 (4 wks apart) → MRF ×3–4 (6 wks) → HA/PLLA filler if volume deficit → maintenance peels every 3 months |
| Mostly ice-pick | TCA CROSS ×4 (4–6 wks) → punch excision of resistant ones → fractional laser/MRF ×2–3 to blend |
| Mixed (ice-pick + boxcar + rolling) | Treat active acne → subcision ×2 (rolling) → TCA CROSS on ice-picks (parallel) → MRF/fractional laser ×4–6 over whole area → filler for residual depth → surface peels |
| Hypertrophic (jawline / chest) | Intralesional triamcinolone ± 5-FU ×3–6 (4 wks) → daily silicone → PDL for redness → avoid resurfacing |
| Atrophic + PIH | Topical brighteners + sun protection 8–12 weeks first → then combination at lower energies |
| Still on isotretinoin | Defer most procedures until 6 months after finishing; topical maintenance only |

**Table 6.2 — Sample combination protocols (individualize)**

**Same-session combinations that work:** subcision + filler; TCA CROSS + microneedling around it; fractional laser + filler in separate areas; intralesional steroid + PDL for hypertrophic scars. Don't stack multiple aggressive resurfacing modalities in one visit.

**Topicals are the backbone between procedures:** a retinoid (skin quality, PIH, priming — pause around procedures), daily SPF 30+ (non-negotiable), brighteners for PIH, and a gentle cleanser and moisturizer. Patients who follow the daily plan get far better cumulative results.

**🔑 Pearl**

the 12-month timeline. Most plans run 6–12 months, sometimes 18. The dramatic online before-and-afters are a year of layered work, not one visit. Set this expectation before the first procedure.

## 6.3 Planning and expectations

Even the best technical plan fails if the patient doesn't understand it or expects the impossible. The consultation is as important as any procedure.



| Step | What you do |
|---|---|
| **1. History** | Active acne, past treatments, family history, isotretinoin, pregnancy/contraception, medications, psychological burden |
| **2. Examination** | All three dimensions, multiple lighting, standardized photos |
| **3. Classification** | Identify the scar subtypes present and the dominant patterns |
| **4. Plan** | A staged combination protocol with sessions, intervals, and adjuncts |
| **5. Expectations** | Realistic estimate (≈50–80%, not 100%); 6–18 month timeline; cost in time and money |
| **6. Consent** | Written, per modality, with specific risks (PIH, prolonged redness, treatment failure, scar worsening) |

**Table 6.3 — The six-part first consultation**

Promise: meaningful improvement (≈30–70% for atrophic scars over a full course), better overall texture and tone, lasting results with sun protection, and a staged plan you'll follow together.

Don't promise: 100% removal, dramatic change in one visit, results in the first session, or equal improvement across all scar types (ice-pick is the most resistant).

**🔑 Pearl**

the before-and-after honesty test. Look at the patient's screenshot together, identify the scar types in it, then ask "Do your scars look like those?" Often they don't — and that conversation, done early, protects everyone.

Psychological dimension: acne scarring is linked with anxiety, depression, social withdrawal, and sometimes suicidal ideation. Screen briefly ("How are these scars affecting your daily life?" / "Do you avoid people or photos?" / "How are you feeling overall?"). If there's real distress, offer or refer for support — but don't refuse treatment because someone is distressed; effective scar treatment is itself one of the most powerful things you can do for that distress.

When to refer: active severe acne → dermatologist; confirmed keloids in high-risk skin → keloid-experienced dermatologist; significant distress or body-dysmorphic features → mental health professional; burn, surgical, or contracture scars → plastic surgery; expectations that can't be aligned with reality → sometimes the ethical choice is to decline.

Follow-up rhythm: 2 weeks after each major procedure (check healing); 4–6 weeks between sessions (assess, plan next); 3 months post-course (photo re-evaluation); 6 and 12 months (long-term, consider maintenance). Side-by-side photos matter — patients adapt to gradual change and forget where they started.

### 🔑 Pearl

the most important sentence in the first consultation. "Your scars will improve a lot, but they won't be gone. What we'll do together is make them much less visible to other people, much less visible to you, and much less of a thing you have to think about every day."

### Key points

- Acne scars are 3-dimensional: surface, depth, and volume. Address all three.
- Sequence runs deep to superficial: subcision → volume → remodeling → surface.
- Never treat scars over active acne.
- Combination therapy over 6–18 months is the standard for moderate-to-severe scarring.
- Set realistic expectations (≈50–80%, not 100%).
- Screen for the psychological burden; refer when needed. Treating the scars is itself powerful for the distress.
- The first consultation is the most important visit. Document with standardized photos at baseline, 3, 6, and 12 months.

# *Part III. Cellulite and Fat Accumulation*



7



# CELLULITE: PHYSIOLOGY AND DIAGNOSIS

Cellulite is the dimpled, “orange-peel” or “mattress” texture seen mostly on the thighs and buttocks. It affects the large majority of women after puberty — by most estimates 80–90% — and is uncommon in men. It isn’t a disease or a danger, but it’s one of the most common cosmetic complaints you’ll meet, and one of the hardest to treat well. The key is understanding that cellulite is a structural problem, not a fat problem.

## 7.1 Cellulite and obesity — not the same thing

The single most important thing to tell patients: cellulite is not the same as being overweight. Slim, fit women get cellulite; losing weight often softens it but rarely removes it. The reason is anatomical. Three things combine to create the dimpled look:

- Fibrous septae — the connective-tissue bands tethering skin to deeper layers. In women they run mostly straight up-and-down (perpendicular), so as the fat layer expands it bulges up between the bands while the bands pull the surface down — creating dimples. In men they criss-cross, bracing the surface; that’s the main reason men rarely get cellulite.
- Fat herniation — fat lobules pushing up toward the skin between the septae.
- Skin laxity — thinner, less elastic skin (from age, hormones, weight change) makes the dimpling more visible.

Hormones (especially estrogen), genetics, and circulation all play a part — which is why cellulite often appears at puberty and can worsen with pregnancy. So while weight management can help, treatment has to address the bands, the fat, and the skin — not just the scale.

> 🔑 **Pearl**
>
> the sentence that resets expectations. “Cellulite isn’t about being overweight — even very slim women have it. It comes from the way the connective bands under the skin are arranged, which is why diet alone usually won’t clear it.” Saying this early stops patients from blaming themselves and chasing weight loss that won’t fix the dimples.

## 7.2 Stages of cellulite

Grading guides both treatment choice and documentation. The classic system is the Nürnberger–Müller scale:

| Grade | What you see |
|---|---|
| 0 | Smooth skin standing and lying; no dimpling even on pinch |
| 1 | Smooth at rest, but dimpling appears on pinching the skin (the “mattress” sign) |
| 2 | Dimpling visible when standing; disappears when lying down |
| 3 | Dimpling visible both standing and lying, with raised nodules and a clearly uneven surface |

**Table 7.1 — Nürnberger-Müller cellulite grades**

Newer validated tools — the Cellulite Severity Scale (CSS) and the dimple-at-rest scales used in modern trials — score several features (number and depth of dimples, raised lesions, laxity, and the Nürnberger grade) to track change over time. For everyday practice, the Nürnberger grade plus standardized photos (relaxed and on pinch, consistent lighting and posture) is enough.

> 🔑 **Pearl**
>
> separate dimples from laxity. Under good light, decide whether you’re mostly seeing discrete dimples (septae-driven — best for subcision-type treatments) or a loose, crepey surface (laxity-driven — better for skin-tightening energy devices). They need different tools.

## 7.3 The physiology of fat accumulation

Localized fat is the other half of this Part — the stubborn pockets that don’t shift with diet and exercise (outer thighs, flanks, lower abdomen, under the chin). To treat them you need to know how fat is stored.

Fat is stored in adipocytes (fat cells) grouped into lobules in the subcutaneous layer. Two points matter clinically:

- Adult fat cells mostly change size, not number. Diet and exercise shrink existing fat cells; they don’t easily remove them. This is why certain pockets are so persistent — and why body-contouring treatments aim to destroy or permanently reduce fat cells rather than just shrink them.

• Fat distribution is largely hormonal and genetic. Where someone stores fat — hips and thighs (“pear”) versus abdomen (“apple”) — is set mostly by sex hormones and genes. Subcutaneous fat (the pinchable kind these treatments target) is different from visceral fat (around the organs), which responds to diet and exercise and matters for health.

The practical takeaway: body-contouring devices target small, diet-resistant subcutaneous pockets in people already near their goal weight. They are not weight-loss tools and won’t touch visceral fat.

> 🔑 **Pearl**
>
> contouring is not weight loss. “These treatments remove or shrink a specific stubborn pocket. They won’t change the number on the scale, and they’re not a substitute for losing weight. The best candidate is already close to their target weight with one or two areas that won’t budge.”

## 7.4 Diagnostic assessment

A good consultation answers four questions:

• What is the dominant problem? Dimples (septae), bulk (fat), or loose skin (laxity)? Often a mix — name the proportions, because each points to a different treatment.

• What grade, and where? Nürnberger grade; map the areas; standardized photos at rest and on pinch.

• Is the patient a good candidate? Near goal weight, stable, realistic, non-smoker, no contraindications. Significant obesity or hopes of weight loss → manage expectations or defer.

• What’s the skin quality? Marked laxity shifts the plan toward tightening and limits what subcision or fat reduction alone can achieve.

Examine standing (most cellulite shows with gravity), in good light, with and without the pinch test, and document with consistent photos.



| Dominant problem | Points toward |
|---|---|
| Discrete dimples (septae) | Subcision-based: manual / precise subcision, acoustic subcision |
| Loose, crepey skin (laxity) | Skin-tightening energy: radiofrequency, microfocused ultrasound |
| Localized fat bulge | Fat reduction: cryolipolysis, deoxycholic acid (submental), laser/RF lipolysis |
| Mixed | Combination, staged over months |

**Table 7.2 — Matching the dominant problem to the treatment direction**

### 🔑 Pearl

photograph everything at baseline. Standing, relaxed and pinched, same angle and light each visit. Cellulite and contour change slowly; side-by-side photos are the only honest measure of progress.

### Key points

- Acne scars are 3-dimensional: surface, depth, and volume. Address all three.
- Sequence runs deep to superficial: subcision → volume → remodeling → surface.
- Never treat scars over active acne.
- Combination therapy over 6–18 months is the standard for moderate-to-severe scarring.
- Set realistic expectations (≈50–80%, not 100%).
- Screen for the psychological burden; refer when needed. Treating the scars is itself powerful for the distress.
- The first consultation is the most important visit. Document with standardized photos at baseline, 3, 6, and 12 months.

8



# TREATING CELLULITE AND LOCALIZED FAT

No treatment "cures" cellulite, and results vary a lot between patients. The honest framework: match the tool to the dominant problem (dimples, fat, or laxity), combine when needed, and set expectations before you start. This chapter runs from the gentlest options to the most invasive.

## 8.1 Lifestyle and topical approaches

Start here, but be honest about the ceiling.

- **Weight and fitness:** staying near a stable goal weight and building muscle (glutes and thighs) improves the look and supports any procedure. It won't erase septae-driven dimples.
- **Diet and hydration**: no specific diet treats cellulite. Healthy eating helps overall — don't oversell it.
- **Topicals**: caffeine creams temporarily firm and dehydrate the surface for a subtle, short-lived effect; retinol may slightly thicken skin over months; most other "cellulite creams" have little solid evidence. Frame them as maintenance polish, not treatment.
- **Massage / mechanical devices (e.g. endermologie):** roller-and-suction massage gives temporary smoothing through fluid shift and circulation; needs ongoing sessions to maintain.

### 🔑 Pearl

the honest topical conversation. "Creams and massage can make the skin look a little firmer for a short time, but they don't fix what's under the surface. If you want real, lasting change in the dimples, we need to treat the bands or the fat directly."

## 8.2 Energy-based devices

A broad group working by heat, cooling, or shock-wave energy. Most need a series of sessions and give gradual, modest results — better for laxity and mild cellulite than for deep discrete dimples.

| Device | How it works | Best for | Notes |
|---|---|---|---|
| Radiofrequency (mono/bi/multi polar) | Heats dermis/subcutis → collagen tightening | Skin laxity, mild cellulite | Series of 4–8; gradual; good safety record |
| Microfocused ultrasound (HIFU) | Focused heat at set depths → tightening; deeper for fat | Laxity; some fat reduction | Minimal downtime; builds over months |
| Acoustic wave therapy (shockwave) | Pressure waves improve circulation, stimulate collagen | Mild–moderate cellulite | Radial or focused; series of 6–8; modest evidence |
| 1440-nm Nd:YAG minimally invasive laser (e.g. Cellulaze) | Side-firing fiber under the skin cuts septae and heats tissue | Discrete dimples; one-time | Minimally invasive, local anesthesia; longer-lasting than surface devices |
| Cryolipolysis (fat freezing) | Cools fat to selectively destroy fat cells | Localized fat bulges (not cellulite itself) | ~20–25% fat-layer reduction per area over 1–3 months; rare paradoxical fat growth |
| Laser / RF lipolysis (e.g. 1060-nm diode) | Heats and destroys fat cells | Localized fat | Gradual fat-layer reduction |

**Table 8.1 — Energy-based options**

For cellulite specifically, the strongest evidence sits with subcision-type approaches (next section) and, among energy devices, the 1440-nm laser and acoustic wave therapy. Radiofrequency and HIFU shine where laxity is the main issue.

## 8.3 Injectable and minimally invasive options

This is where the best results for true dimples live, because these target the fibrous bands directly.

### Subcision — the core principle

A needle or device is passed under a dimple to cut the septa tethering it down; releasing the band lets the surface lift. Options:

- Manual subcision — an 18G needle or blunt cannula fanned under the dimple, as in scar work. Effective and inexpensive; results depend on operator skill; bruising is common.
- Precise / tissue-stabilized subcision (Cellfina) — a device that stabilizes the skin and cuts septa at a controlled depth; FDA-cleared, with studies showing improvement lasting 2–3 years.
- Targeted manual subcision device (Avéli) — a handheld device that finds and releases individual septa under local anesthetic in a single visit; FDA-cleared and increasingly popular.
- Acoustic subcision / rapid acoustic pulse (Resonic) — non-invasive; rapid sound-pressure pulses shear septa with no needles, no anesthesia, and essentially no downtime; a single session with improvement out to a year in trials.

### Injectables

- Biostimulator fillers (PLLA, CaHA) — injected to stimulate collagen and add support to depressed areas; growing evidence as an adjunct, especially alongside subcision.
- Collagenase (Qwo) — was the first FDA-approved injectable for buttock cellulite (2020), dissolving the collagen septa enzymatically. It was withdrawn from the market in December 2022 over unpredictable, prolonged bruising and discoloration. Worth knowing for context, but no longer available.

### Deoxycholic acid — for localized fat, not cellulite

Deoxycholic acid (Kybella / Belkyra; ATX-101) is a synthetic version of a bile acid that destroys fat cells on contact.

- FDA-approved only for submental fat (under the chin). Use elsewhere is off-label and not established.
- Dosing (submental): area-adjusted 2 mg/cm²; 0.2 mL per injection, spaced 1 cm apart; up to 50 injections (10 mL = 100 mg) per session; up to 6 sessions at least 1 month apart.

• Expect swelling (sometimes marked), bruising, numbness, and pain after each session. The destroyed fat cells don't return, so results are durable if weight stays stable.

**⚠ Caution**

deoxycholic acid and the smile. Deoxycholic acid is FDA-approved for the chin only. The marginal mandibular nerve runs through the area; injecting outside the mapped safe zone can cause a temporary (occasionally longer) asymmetric smile. Off-label body use carries unproven safety. Know the anatomy or don't inject.

**🔑 Pearl**

match the procedure to the problem. Dimples = release the band (subcision, acoustic subcision); fat bulge = reduce the fat (cryolipolysis, deoxycholic acid where appropriate); loose skin = tighten (RF, HIFU). Treating a dimple with a fat device, or a fat bulge with subcision, disappoints everyone.

## 8.4 Patient selection and realistic expectations

**The right patient:** near a stable goal weight, with discrete treatable dimples and/or a defined fat pocket, good general health, no active infection in the area, not pregnant, and — most importantly — realistic.

**The wrong fit:** someone expecting cellulite to vanish, someone counting on it for weight loss, significant laxity expecting smoothness from fat reduction alone, or unstable weight.

**Promise:** visible improvement in dimples and contour, softer texture, and — for subcision and fat reduction — durable results if weight stays stable. **Don't promise:** complete or permanent elimination (cellulite commonly returns in part over years as new septa form), identical results between patients, or change after a single gentle (energy or topical) session.

| Problem | First-choice options | Honest message |
|---|---|---|
| Discrete dimples | Subcision / acoustic subcision; 1440-nm laser | “We can release the bands causing the dimples — expect real, lasting improvement, not perfection.” |
| Localized fat pocket | Cryolipolysis; deoxycholic acid (chin) | “We can shrink this specific pocket. Stay weight-stable and it lasts.” |
| Loose, crepey skin | Radiofrequency; microfocused ultrasound | “We’ll firm the skin gradually over a few months — this is tightening, not fat removal.” |
| Mixed | Staged combination over months | “We’ll tackle the bands, the fat, and the skin in stages — plan on several months.” |

**Table 8.2 — Quick guide: problem → first-choice options → what to say**

### 🔑 Pearl

the expectation sentence for cellulite. “We can make these dimples noticeably less visible, and good treatments can last a couple of years — but cellulite is structural, so some tends to come back over time. The goal is a real, visible improvement you’re happy with, not perfectly smooth skin.”

### Key points

- No treatment cures cellulite; match the tool to the dominant problem — dimples, fat, or laxity — and combine when needed.
- Lifestyle and topicals help overall and give short-lived smoothing, but won’t fix septae-driven dimples.
- Energy devices (RF, HIFU, acoustic wave) give gradual, modest results, best for laxity

and mild cellulite; cryolipolysis reduces localized fat ~20–25% per area.

• Subcision-based options (manual, Cellfina, Avéli, acoustic subcision) give the best, most durable results for true dimples by releasing the bands.

• Deoxycholic acid (Kybella) is FDA-approved for submental fat only: 2 mg/cm², 0.2 mL/injection 1 cm apart, up to 50 injections/session, up to 6 sessions ≥1 month apart. Mind the marginal mandibular nerve.

• Qwo (collagenase), the first injectable approved for buttock cellulite, was withdrawn in 2022.

• The best candidate is near goal weight and realistic. Promise visible improvement, not permanent perfection.



# *Part IV. Pigmentation Disorders*



9



# MELANOCYTE BIOLOGY AND PIGMENTARY DISORDERS

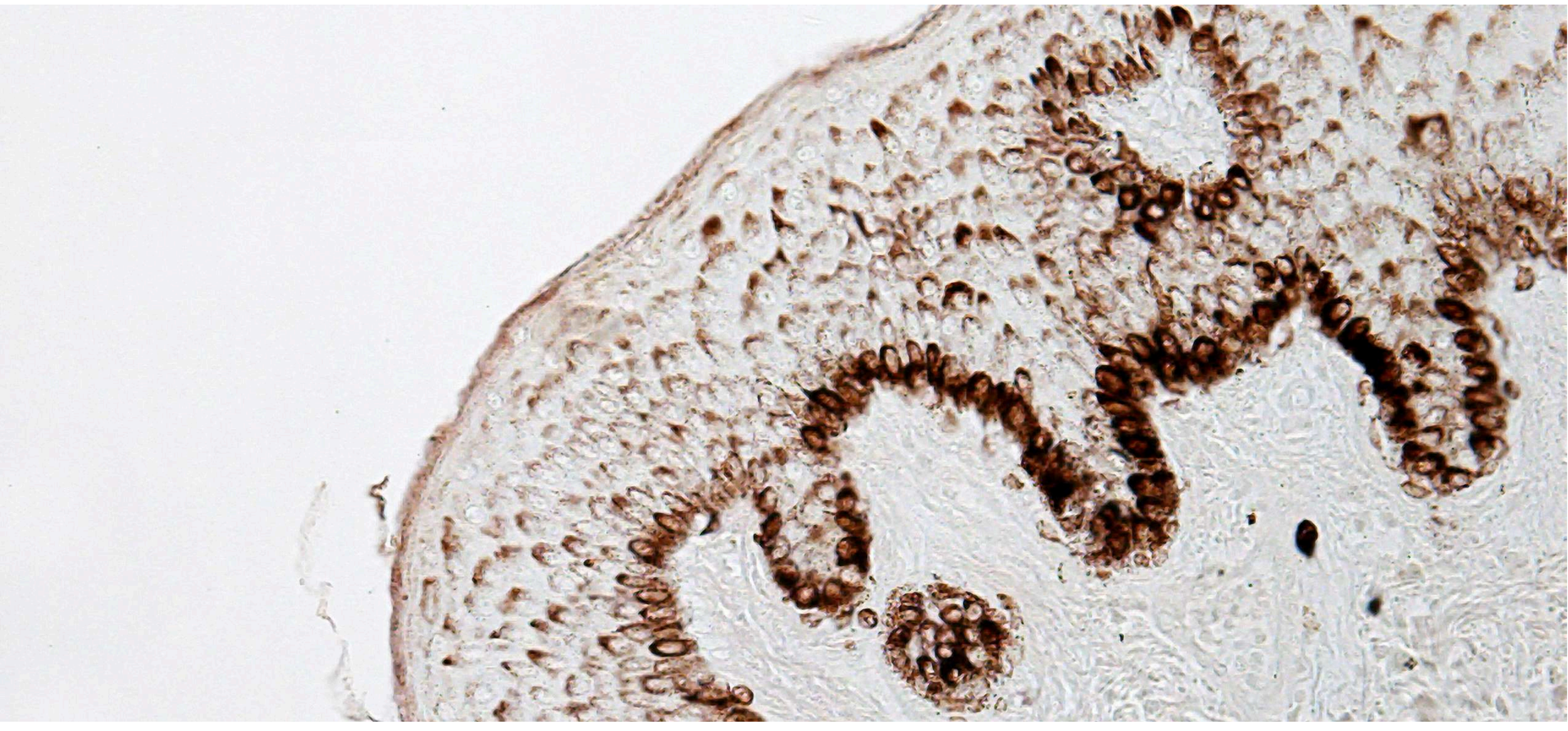
Skin colour comes from one pigment, made by one type of cell. Understanding how that works explains every pigmentary disorder you'll treat — and why some are easy to fix and others frustratingly stubborn.

## 9.1 How melanin is made

Melanocytes are pigment-producing cells in the bottom layer of the epidermis. Each one feeds packets of pigment to about 30–40 surrounding skin cells (together called an "epidermal melanin unit"). Everyone has roughly the same number of melanocytes regardless of skin colour — what differs is how much pigment they make and what type.

The pigment is melanin, made inside organelles called melanosomes through a chain of reactions starting from the amino acid tyrosine. The key (rate-limiting) enzyme is tyrosinase — block it and you reduce pigment, which is exactly how most "brightening" treatments work. There are two kinds of melanin: eumelanin (brown/black, protective) and pheomelanin (red/yellow, less protective).

What drives pigment: UV light is the biggest trigger; hormones (estrogen, MSH), inflammation, and genetics also push melanocytes to make more.



### 🔑 Pearl

why sunscreen is non-negotiable. Every pigmentary treatment you give, from melasma cream to laser, is undone by UV. UV directly switches on the melanocyte. Daily broad-spectrum SPF — ideally tinted with iron oxides for visible-light protection — isn't an add-on, it's the foundation. No sunscreen, no result.

Where the pigment sits matters: epidermal pigment (upper layers) responds well to topicals and gentle treatment — better prognosis; dermal pigment (dropped deeper) is much harder to reach and stubborn; most real cases are mixed. This single fact — how deep the pigment is — predicts how treatable a problem will be, and you can often judge it at the bedside with a Wood's lamp.

## 9.2 Classifying pigmentary disorders

At the simplest level, pigment problems are either too little or too much:

| Category | What it is | Common examples |
|---|---|---|
| Hypopigmentation | Melanocytes lost or not working | Vitiligo, pityriasis alba, post-inflammatory hypopigmentation, tinea versicolor |
| Depigmentation | Complete melanocyte loss | Vitiligo (fully white patches) |
| Hyperpigmentation | Melanocytes overproduce, or pigment is misplaced | Melasma, post-inflammatory hyperpigmentation (PIH), lentigines, freckles |

**Table 9.1 — The pigmentary disorders at a glance**

The chapters that follow take the three you'll see most: vitiligo (Chapter 10), melasma (Chapter 11), and benign pigmented lesions plus tattoo removal (Chapter 12).

### 🔑 Pearl

the first question in any pigment case. "Is there more pigment, or less?" It sounds obvious, but mislabeling hypopigmentation as hyperpigmentation sends the whole plan in the wrong direction. Then ask: how deep, and is inflammation involved?

### Key points

- All skin pigment is melanin, made by melanocytes from tyrosine; tyrosinase is the key enzyme most brighteners target.
- Everyone has similar melanocyte numbers — skin colour reflects pigment amount and type, not cell count.
- UV is the main driver; sun protection is the foundation of every pigmentation treatment.
- Pigment depth (epidermal vs dermal) predicts treatability — epidermal responds well, dermal is stubborn.
- Pigment disorders are either too little (hypo/depigmentation) or too much (hyperpigmentation).

10



# HYPOPIGMENTATION: VITILIGO

Vitiligo is the loss of pigment in patches, caused by the immune system destroying melanocytes. It affects about 0.5–2% of people worldwide across all skin tones, but is far more visible — and more distressing — in darker skin. It's not contagious or dangerous, but the psychological burden is heavy, and that should shape how you treat it.

## 10.1 Clinical presentation

Vitiligo shows as well-defined, milky-white patches, often symmetrical. Two main patterns:

• Non-segmental (common): symmetrical patches on both sides — face, hands, around the eyes and mouth, over joints. Progresses; autoimmune-driven.
• Segmental: confined to one area/one side, usually appears young, progresses for a while then stabilizes. Less linked to autoimmunity.

Clues: the patches are completely white (not just lighter), the skin texture is normal (no scale), and under a Wood's lamp they glow bright white. Hairs within a patch may also turn white (a sign of harder-to-treat disease). Vitiligo often associates with other autoimmune conditions — especially thyroid disease — so checking thyroid function is reasonable.



## 10.2 Differential diagnosis

| Condition | How to tell it apart |
|---|---|
| Pityriasis alba | Pale, faintly scaly patches on children's faces; partial (not total) pigment loss; self-resolving |
| Tinea versicolor | Fine scale, scattered on trunk; fungal (scrapes positive); partial pigment loss; responds to antifungals |
| Post-inflammatory hypopigmentation | Follows a prior rash or injury; partial loss; recovers over time |
| Idiopathic guttate hypomelanosis | Small "confetti" white spots on sun-exposed limbs of older adults |
| Pale, faintly scaly patches on children's faces; partial (not total) pigment loss; self-resolving | Present from early life, stable, doesn't spread |

**Table 10.1 — Vitiligo look-alikes**



The features pointing to vitiligo: complete (not partial) pigment loss, no scale, bright Wood's-lamp accentuation, and a progressive symmetrical pattern.

## 10.3 Treatment approaches

Treatment aims to stop the immune attack and stimulate repigmentation from surviving melanocytes (mostly in hair follicles — which is why hairy areas respond better than fingertips, lips, and bony spots). It's slow: expect months, and say so upfront.

| Treatment | How it works | Notes |
|---|---|---|
| Topical corticosteroids | Suppress the immune attack | First-line for limited disease; short courses; watch for skin thinning — avoid long-term on the face |
| Calcineurin inhibitors (tacrolimus, pimecrolimus) | Immune modulation without steroid thinning | Preferred for face and eyelids; safe for longer use |
| Ruxolitinib cream 1.5% (Opzelura) | Topical JAK inhibitor — blocks the immune signal | First and only FDA-approved repigmentation treatment; nonsegmental vitiligo, ages 12+, twice daily, up to 10% body surface; results may need >24 weeks |
| Narrowband UVB (311 nm) | Stimulates melanocytes, calms immunity | Mainstay for widespread disease; 2–3 sessions/week for months; best repigmentation evidence |
| Excimer laser (308 nm) | Targeted phototherapy | For localized patches; combines well with topicals |
| Melanocyte grafting (surgical) | Transplants pigment cells | For stable, segmental disease unresponsive to medical therapy |
| Depigmentation (e.g. monobenzone) | Removes remaining pigment | Only for very extensive disease where repigmentation isn't realistic — a major, irreversible decision |

**Table 10.2 — Vitiligo treatments**

General principles: face and neck respond best; hands, feet, and lips worst. Combination (e.g. topical + narrowband UVB) beats monotherapy. Sun protection still matters — depigmented skin burns easily, and reducing contrast helps cosmetically.

### 🔑 Pearl

never dismiss the psychological weight. Vitiligo isn't medically dangerous, so it's easy to under-treat — but in darker skin especially it can be socially devastating. Take it seriously, offer treatment, and screen for distress. Camouflage cosmetics and self-tanners (dihydroxyacetone) are legitimate immediate options while medical treatment works slowly.

### ⚠ Caution

set the timeline honestly. Repigmentation takes months and is often incomplete, and some areas (fingertips, lips) may never fully respond. Promise effort and partial-to-good improvement on responsive areas (especially the face), not a cure.

## Key points

- Vitiligo is autoimmune melanocyte loss → completely white patches, normal texture, bright under Wood's lamp.
- Non-segmental (symmetrical, progressive, autoimmune) is common; segmental (one area, stabilizes) is less so.
- Distinguish from pityriasis alba, tinea versicolor, and post-inflammatory hypopigmentation by completeness of pigment loss, scale, and pattern.
- Treatment combines immune suppression (topical steroids, calcineurin inhibitors, ruxolitinib cream) with phototherapy (narrowband UVB).
- Face responds best; hands/feet/lips worst. Combination beats monotherapy; results take months.
- Check thyroid; take the psychological impact seriously; camouflage is a valid immediate aid.

11



# HYPERPIGMENTATION: MELASMA

Melasma is the most common — and most frustrating — hyperpigmentation you'll treat: a chronic, relapsing condition of symmetrical brown patches on the face, mostly in women, strongly tied to sun, hormones, and genetics. The honest framing for every patient: melasma is managed, not cured.

## 11.1 Pathophysiology and patterns

Melasma comes from overactive melanocytes making too much pigment, driven by a mix of triggers:

- UV and visible light — the dominant trigger; even screen and window light matters.
- Hormones — estrogen and progesterone (pregnancy, the pill, HRT).
- Genetics — strong family history; more common in Fitzpatrick III–V.
- Heat and inflammation — including from aggressive treatments, which can backfire.

There's also a vascular component, and melasma is now understood as partly a problem of a weakened skin barrier and photoaging — which is why gentle, anti-inflammatory regimens work better than aggressive ones.

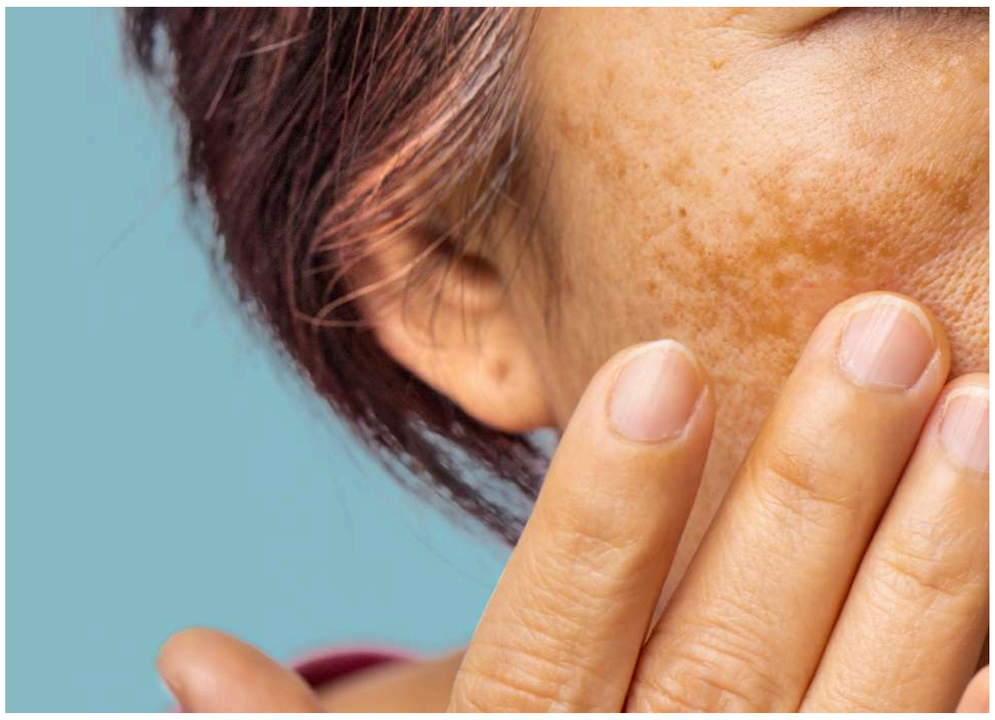
Patterns by location: centrofacial (forehead, cheeks, nose, upper lip, chin — most common), malar (cheeks), mandibular (jawline). By depth (predicts prognosis): epidermal (brown, sharp borders, accentuates under Wood's lamp → treatable); dermal (blue-grey, blurred borders, doesn't accentuate → stubborn); mixed (most cases).



## 11.2 Differential diagnosis

| Condition | How to tell it apart |
|---|---|
| Post-inflammatory hyperpigmentation (PIH) | Follows a specific rash/injury/acne; not necessarily symmetrical; history is the clue |
| Solar lentigines | Discrete, well-defined brown spots on sun-exposed skin; not patchy or symmetrical |
| Exogenous ochronosis | Paradoxical blue-black darkening from prolonged high-strength hydroquinone — a treatment complication to recognize |
| Drug-induced pigmentation | Tied to a medication (e.g. minocycline, amiodarone); different distribution |
| Naevus of Ota / Hori's macules | Blue-grey dermal pigment; need laser, not brighteners |

**Table 11.1 — Melasma look-alikes**



The Wood's lamp is your bedside tool: epidermal pigment gets more obvious under it (good prognosis); dermal pigment doesn't (poor prognosis). This guides both expectations and how aggressive to be.

## 11.3 Treatment strategies

Melasma rewards patience and gentleness and punishes aggression (heat and inflammation make it worse). Build the plan in layers.

Foundation (non-negotiable): strict photoprotection — broad-spectrum SPF 50, reapplied, ideally tinted with iron oxides for visible-light protection; this alone improves melasma and prevents relapse. Remove triggers where feasible (the pill/HRT, heat exposure). Without this, nothing else works.

Topical lightening: hydroquinone is the gold-standard tyrosinase inhibitor, prescription strength (commonly 4%), used in courses (≈3–4 months) then rested to avoid ochronosis (regulatory status varies by country — prescription-only or restricted in many regions). The triple-combination cream (hydroquinone + a retinoid + a mild steroid, "modified Kligman's") is the most effective topical, short-term.

Non-hydroquinone options — azelaic acid, kojic acid, niacinamide, vitamin C, topical tranexamic acid, cysteamine — are useful for maintenance and for those who can't use hydroquinone.

Oral: tranexamic acid is the major recent addition. Off-label for melasma; typical dose 250 mg twice daily for about 3 months, often repeated. Screen for clot risk (personal/family history of thrombosis, smoking, the pill) — it's contraindicated where there's thromboembolic risk.

Procedures (with caution): gentle peels (low-strength glycolic, mandelic, or salicylic) as adjuncts; lasers and energy devices only carefully — low-fluence Q-switched/picosecond or non-ablative, in experienced hands. Aggressive lasers commonly cause rebound and PIH, especially in darker skin. When in doubt, don't laser melasma.



| Layer | Options |
|---|---|
| Foundation (always) | Tinted SPF 50 with iron oxides; trigger removal |
| First-line topical | Hydroquinone 4% or triple-combination cream (short courses) |
| Add / maintain | Azelaic acid, vitamin C, niacinamide, cysteamine, topical tranexamic acid |
| For stubborn cases | Oral tranexamic acid 250 mg twice daily (screen for clot risk) |
| Cautious adjuncts | Gentle peels; low-fluence laser only in expert hands |

**Table 11.2 — A layered melasma plan**

### 🔑 Pearl

gentle wins in melasma. The instinct to "blast" pigment with strong lasers or peels backfires — heat and inflammation drive melasma. The best long-term results come from strict sun protection + topical brighteners + (where appropriate) oral tranexamic acid, with procedures kept light. Under-treat rather than over-treat.

### 🔑 Pearl

the expectation conversation. "Melasma is like a tendency, not a one-time stain. We can fade it a lot and keep it faded, but it tries to come back with sun or hormonal changes. Daily sun protection is the treatment that never stops."

### Key points

- Melasma = overactive melanocytes from UV/visible light, hormones, and genetics; chronic and relapsing, mostly in women and darker skin.
- Judge depth with a Wood's lamp: epidermal (accentuates, treatable) vs dermal (doesn't, stubborn).
- Distinguish from PIH, lentigines, ochronosis, and dermal melanocytoses.
- Foundation is strict tinted broad-spectrum SPF + trigger removal — nothing works without it.
- Topicals: hydroquinone/triple-combination in short courses; azelaic acid, vitamin C, cysteamine for maintenance.

- Oral tranexamic acid 250 mg twice daily helps stubborn cases — screen for clot risk.
- Keep procedures gentle; aggressive lasers cause rebound. Manage melasma, don’t promise a cure.

12



# BENIGN PIGMENTED LESIONS AND TATTOO REMOVAL

This chapter covers the common brown spots and growths patients want removed, and the principles of laser tattoo removal. One rule overrides everything here: never treat a pigmented lesion you haven't confidently diagnosed as benign. When in doubt, biopsy or refer — the one thing you must not laser is a melanoma.

## 12.1 Classifying benign pigmented lesions

| Lesion | What it looks like | Notes |
|---|---|---|
| Solar lentigo ("sun/age spot") | Flat, well-defined tan-brown patch on sun-exposed skin | Very common; benign; responds well to laser/peels |
| Ephelides (freckles) | Small tan macules; darken with sun, fade in winter | Benign; genetic |
| Seborrheic keratosis | Waxy, "stuck-on," raised brown/black plaque | Benign; removed by cautery/curettage, not pigment laser |
| Café-au-lait macule | Uniform light-brown patch, present from early life | Usually benign (multiple may signal a syndrome) |
| Melanocytic naevus (mole) | Raised or flat, even brown | Benign but assess for change |
| Dermal melanocytosis (naevus of Ota, Hori's) | Blue-grey patch | Needs Q-switched/pico laser; not topicals |

**Table 12.1 — Common benign pigmented lesions**

The crucial skill is separating these from melanoma. Use the ABCDE rule (Asymmetry, Border irregularity, Colour variation, Diameter >6 mm, Evolving) and the “ugly duckling” sign (a lesion that looks different from the patient’s others).

Any pigmented lesion that is changing, irregular, multicoloured, bleeding, or new in an adult should be seen by a dermatologist before any cosmetic treatment.

## 12.2 Treatment modalities

| Modality | Best for | Notes |
|---|---|---|
| Q-switched lasers (532/694/755/1064 nm) | Lentigines, dermal melanocytoses | Shatter pigment in nanosecond pulses |
| Picosecond lasers | Lentigines, tattoos, melasma (low fluence) | Faster pulses, less heat, often fewer sessions, lower PIH risk |
| IPL (intense pulsed light) | Lentigines, freckles, diffuse sun damage | Good for multiple superficial spots; not a true laser |
| Chemical peels | Superficial epidermal pigment | Adjunct |
| Cryotherapy | Lentigines, seborrheic keratoses | Risk of hypopigmentation, especially in darker skin |
| Curettage / cautery / shave | Raised lesions (seborrheic keratoses) | Physical removal; pigment lasers don’t work on these |

**Table 12.2 — Removing benign pigmented lesions**

The matching principle is the same as everywhere in this book: superficial epidermal pigment responds to gentle surface treatments; deeper dermal pigment needs pigment-specific lasers.

## 12.3 Clinical key points

- Diagnosis before treatment, always. A "spot" the patient wants gone could be a melanoma. If anything about a lesion is atypical, refer before lasering.
- Match the tool to depth and type. Flat brown spots → laser/IPL/peels; raised waxy growths → curettage/cautery; blue-grey dermal pigment → Q-switched/pico laser.
- Mind skin type. Darker skin (Fitzpatrick IV–VI) is far more prone to post-treatment hyper- or hypopigmentation; use conservative settings, test spots, and longer wavelengths.

## 12.4 Pre-treatment precautions and special cases

- Test patch first in darker skin or any uncertainty, and wait to see the response before treating a whole area.
- Sun status. Don't laser recently tanned skin — the background pigment competes for the laser and raises burn/PIH risk.
- Photoprotect before and after every pigment procedure — UV undoes the work and drives PIH.
- Melasma is not a lentigo. Resist treating it like a sun spot with an aggressive laser; it will rebound (Chapter 11).
- Pregnancy. Defer elective pigment laser; avoid hydroquinone and retinoids.

> 🔑 **Pearl**
>
> the lesion you must not laser. If you can't say with confidence a pigmented lesion is benign, don't treat it — refer it. Lasering a melanoma destroys the chance of early diagnosis and can be a fatal mistake. This rule outranks every cosmetic consideration in this chapter.

## 12.5 Tattoo removal

Laser tattoo removal works by photothermolysis: very short, high-power laser pulses shatter the ink into fragments small enough for the immune system to carry away. It's slow, multi-session work.

- Lasers used: Q-switched and picosecond lasers, wavelength matched to ink colour — 1064 nm for black/dark blue, 532 nm for red, 694/755 nm for green and blue. Picosecond lasers generally clear ink in fewer sessions with less collateral damage.

• Sessions: typically 6–12 (often more), spaced 6–8 weeks apart to let the body clear fragments and the skin recover. Amateur tattoos clear faster than professional ones.
• What's hard: black is easiest; green and blue are harder; white, yellow, and flesh tones are very difficult and can paradoxically darken.
• Estimating effort: the Kirby–Desai scale predicts the number of sessions from skin type, ink colour and amount, location, layering, and scarring.

**⚠ Caution**

paradoxical ink darkening. White, flesh-toned, and some red or cosmetic (eyebrow/lip) tattoos can instantly turn black when lasered, due to a chemical change in the pigment. Always test a small area first and warn the patient — this is difficult to reverse.

**🔑 Pearl**

set the session expectation upfront. "This takes many sessions over a year or more, costs more than the tattoo did, and may not clear 100% — especially the coloured parts. We go slowly to protect your skin." Patients who hear this first stay realistic and satisfied.

## Key points

• Never treat a pigmented lesion unless you're confident it's benign — use ABCDE and the ugly-duckling sign; refer anything atypical. Don't laser a possible melanoma.
• Match modality to depth/type: flat brown spots → laser/IPL/peels; raised growths → curettage/cautery; dermal pigment → Q-switched/pico laser.
• Darker skin is prone to PIH/hypopigmentation — test patches, conservative settings, strict sun protection.
• Tattoo removal uses Q-switched/picosecond lasers matched to ink colour; 6–12+ sessions, 6–8 weeks apart; black easiest, colours hardest.
• Beware paradoxical darkening of white/flesh/red inks — always test first.
• Photoprotect before and after every pigment procedure.

# *Part V. Vascular Lesions*



13



# VASCULAR LESIONS: CLASSIFICATION AND DIAGNOSIS

Vascular lesions are marks made of blood vessels — from a baby's strawberry birthmark to the fine red threads on an adult's cheek. They can look alike to the untrained eye but behave very differently, and getting the diagnosis right matters: some resolve on their own, some need urgent specialist care, and only some are yours to treat with a laser. The modern framework (the ISSVA classification) splits them into two big groups: vascular tumours (the cells are actively growing) and vascular malformations (the vessels are wrongly built but not proliferating).

## 13.1 Hemangiomas (the common vascular tumour)

Infantile hemangioma is the most common tumour of infancy, affecting about 4–5% of babies. The story is characteristic: usually not present at birth (or only a faint mark), then a proliferative phase of rapid growth over the first months (the raised red "strawberry"), followed by slow spontaneous involution over years (most are largely gone by age 5–10).

Because most involute on their own, most need no treatment. The minority that do are those threatening function or causing disfigurement — near the eye (blocks vision),



the airway, lip, or nose, or ulcerating lesions.

Treatment when needed:

- Oral propranolol is first-line for problematic hemangiomas needing systemic treatment — typically 2–3 mg/kg/day in divided doses, started and supervised by a pediatric specialist (with cardiac and blood-glucose precautions). It has replaced steroids as the standard.
- Topical timolol 0.5% gel for thin, superficial lesions.
- Pulsed dye laser for residual surface redness/telangiectasia after involution, or for ulcerated lesions.

This is largely pediatric territory. For the aesthetic practitioner, the key skills are recognizing it, knowing it usually self-resolves, and referring problematic ones promptly — not lasering a growing tumour.

> 🔑 **Pearl**
>
> hemangioma vs. malformation, the one-line test. A hemangioma usually isn't there at birth, grows fast, then shrinks. A vascular malformation is there at birth, grows with the child, and never goes away. That single difference in natural history separates the two big groups.

## 13.2 Vascular malformations

These are vessels that formed wrongly before birth. They're present at birth, grow proportionally with the child, and don't involute. They're named by the vessel type:

| Type | Vessel | What it looks like | Notes |
|---|---|---|---|
| Capillary malformation (port-wine stain) | Capillaries | Flat pink-to-red patch, often facial, present at birth; darkens and may thicken with age | The main laser target; lifelong without treatment |
| Venous malformation | Veins | Soft, bluish, compressible swelling that fills when dependent | Often needs sclerotherapy / specialist care |
| Lymphatic malformation | Lymphatics | Clear or blood-tinged vesicles or swelling | Specialist management |
| Arteriovenous malformation (AVM) | Arteries + veins | Warm, pulsatile; high-flow | High-risk; specialist / interventional care |

**Table 13.1 — Vascular malformations**

A key point: a facial port-wine stain over the forehead/upper eyelid (the ophthalmic branch of the trigeminal nerve) can be associated with Sturge–Weber syndrome (eye and brain involvement). These infants need ophthalmology and neurology assessment, not just cosmetic care.

## 13.3 Telangiectasia

Telangiectasias are permanently dilated small surface vessels — the fine red lines on adult faces and legs. Unlike malformations, they're acquired. Common patterns:

- Facial telangiectasia — sun damage, rosacea, aging, topical steroid overuse.
- Spider angioma (spider naevus) — a central red dot with radiating legs; common in pregnancy and liver disease.
- Cherry angiomas — small bright-red papules, very common with age; benign.
- Leg telangiectasia ("spider veins") — often linked to underlying venous insufficiency.
- Poikiloderma of Civatte — reddish-brown mottling on the sides of the neck from chronic sun exposure.

These are the bread-and-butter of aesthetic vascular laser work — mostly benign and treatable — but always consider the underlying cause (rosacea, sun, venous disease, or, for a sudden crop of spider naevi, liver disease).

## 13.4 Differential diagnosis

A practical approach to any red lesion:

- Present at birth or acquired? Birth → malformation (or absent-then-grew → hemangioma). Acquired in an adult → telangiectasia or angioma.
- Does it blanch? Press with a glass slide (diascopy). Vascular lesions blanch (empty and refill); a fixed red/purple that doesn't blanch suggests blood outside the vessels (purpura) — a different problem.
- Flat or raised? Flat patch at birth → port-wine stain; raised growing lump in infancy → hemangioma; soft bluish compressible → venous malformation.
- Any red flags? Pulsatile/warm (AVM — refer); forehead port-wine stain (Sturge–Weber — refer); sudden crop of spider naevi (check liver).

**🔑 Pearl**

always blanch a red lesion. A glass-slide press tells you instantly whether you're seeing blood inside vessels (blanches — vascular, laser-treatable) or blood that's leaked out (doesn't blanch — purpura/bruise, not a laser target). It's the fastest, cheapest diagnostic move you have.

## Key points

- The ISSVA framework splits vascular lesions into tumours (growing cells, e.g. hemangioma) and malformations (mis-built vessels, present at birth).
- Infantile hemangiomas appear after birth, grow, then mostly self-resolve — treat only problematic ones; oral propranolol (2–3 mg/kg/day, specialist-supervised) is first-line.
- Vascular malformations are present at birth and never involute; the port-wine stain (capillary) is the main laser target; AVMs and a forehead port-wine stain need referral.
- Telangiectasias are acquired dilated surface vessels (rosacea, sun, aging, venous disease) — the mainstay of aesthetic vascular laser work.
- Diascopy (glass-slide blanch test) distinguishes vascular lesions from purpura.

14

# LASER AND LIGHT-BASED TREATMENT

Vascular lasers work on one beautifully simple principle, and understanding it is the difference between clearing a vessel and burning the skin around it. This chapter covers that principle, the device options, the role of the Nd:YAG, and how to choose patients and settings safely.

## 14.1 The principle: selective photothermolysis

Selective photothermolysis is the idea that you can destroy a target in the skin without harming its surroundings if you get three things right:

• Wavelength — choose light the target absorbs more than the surrounding tissue. For vessels the target is oxyhemoglobin, which absorbs strongly around 418, 542, and 577 nm. Vascular lasers are built around these peaks.

• Pulse duration — match the pulse to the vessel's size. The pulse should be shorter than the time the vessel takes to cool (its thermal relaxation time), so heat stays inside it. Bigger vessels need longer pulses; tiny vessels need short ones.

• Fluence (energy) — enough to heat and coagulate the vessel, not so much that you damage skin.

Get these right and the vessel heats, coagulates, and clears while the surrounding skin is spared. The competing chromophore to respect is melanin — which is why darker skin needs longer wavelengths and more caution.



### 🔑 Pearl

why melanin changes everything. Hemoglobin is the target, but melanin also absorbs these wavelengths. In darker skin, surface pigment competes for the energy, raising the risk of burns and pigment change. The darker the skin, the longer the wavelength (toward Nd:YAG 1064 nm), the lower the fluence, and the more cooling you use.



## 14.2 Laser and light options

| Device | Wavelength | Best for | Notes |
|---|---|---|---|
| Pulsed dye laser (PDL) | 585–595 nm | Port-wine stains, facial telangiectasia, rosacea redness, hemangiomas | Gold standard for superficial vascular lesions; classic endpoint is transient purpura |
| KTP | 532 nm | Fine superficial facial vessels, cherry angiomas | Well absorbed by hemoglobin; higher melanin risk — caution in darker skin |
| IPL (intense pulsed light) | Broad 500–1200 nm | Diffuse facial redness, rosacea, poikiloderma | Not a laser; versatile for background redness; operator-dependent |
| Nd:YAG | 1064 nm | Deeper/larger vessels; leg veins; darker skin | Penetrates deepest; safest in dark skin; needs care — see 14.3 |

**Table 14.1 — Vascular laser and light devices**

Match by vessel depth and size and by skin type: small superficial red vessels → PDL or KTP; diffuse redness → IPL or PDL; deeper/larger blue vessels and leg veins → Nd:YAG; darker skin → Nd:YAG with conservative settings. Most lesions need a series — often 2–4 sessions for telangiectasia, more for port-wine stains, spaced about 4–8 weeks apart. Port-wine stains in particular often need many sessions and rarely clear completely; set that expectation early.

## 14.3 Nd:YAG for vascular lesions

The long-pulsed 1064 nm Nd:YAG fills the gaps the PDL can't reach:



- Deeper and larger vessels — its longer wavelength penetrates further into the dermis, reaching vessels a PDL can't.
- Leg veins — the workhorse laser for small-to-medium leg telangiectasia (often alongside sclerotherapy, which remains first-line for many leg veins).
- Darker skin — because 1064 nm is absorbed less by melanin, it's the safest vascular wavelength in Fitzpatrick IV–VI.

The trade-offs: it's more painful, and because it carries more energy deeper, the margin for error is smaller — overtreatment risks burns, scarring, and pigment change. It rewards conservative settings, good cooling, and experience.

> 🔑 **Pearl**
>
> Nd:YAG is power and risk in the same beam. It reaches what nothing else can and is safest in dark skin, but it's the least forgiving vascular laser. Start low, cool well, watch the endpoint, and don't chase complete clearance in a single pass.

## 14.4 Patient selection, endpoints, and safety

Good candidates: a clearly vascular (blanching) lesion, realistic expectations, no recent tan, no photosensitizing factors, and — for anything you're unsure about — a

confirmed benign diagnosis first.

Endpoints (what tells you it's working):

• PDL: transient purpura (a grey-purple bruise) is the classic sign the vessel coagulated; newer sub-purpuric settings aim for transient darkening / vessel disappearance without bruising.

• KTP / Nd:YAG: the vessel darkens, spasms, or disappears. For leg veins, look for vessel contraction without skin whitening or blistering.

• Stop before: skin greying/whitening, blistering, or pinpoint bleeding — these mean you've gone too far.

Safety essentials: eye protection for everyone in the room (special care around the orbit); epidermal cooling (contact or air), especially with KTP and Nd:YAG; a test spot in darker skin or any uncertainty; treat untanned skin only; photoprotection before and after to reduce PIH; and honest multi-session expectations.

| Lesion | First choice | Notes |
|---|---|---|
| Facial telangiectasia / rosacea redness | PDL or KTP; IPL for diffuse redness | 2–4 sessions; sub-purpuric settings reduce downtime |
| Port-wine stain | PDL | Many sessions; start early in life; rarely complete clearance |
| Cherry angioma | KTP or PDL | Usually clears in 1–2 sessions |
| Leg telangiectasia / spider veins | Sclerotherapy (first-line) ± Nd:YAG | Rule out underlying venous insufficiency |
| Deeper/larger vessels, or darker skin | Nd:YAG 1064 nm | Conservative settings, strong cooling |

**Table 14.2 — Matching lesion to laser**

### ⚠ Caution

diagnose before you fire. Only treat lesions you've confirmed are benign vascular lesions. A pulsatile lesion may be an AVM (refer, don't laser); a forehead port-wine stain may signal Sturge–Weber (needs eye/brain assessment); a non-blanching purple lesion isn't a laser target at all. The laser is the last step, after the diagnosis is secure.

### 🔑 Pearl

the port-wine stain conversation. "We can lighten this significantly over a series of treatments, and starting young gives the best results — but it often won't disappear completely, and it can slowly come back over years, so occasional maintenance is normal." Honest framing from the first visit prevents disappointment later.

## Key points

- Selective photothermolysis = right wavelength (hemoglobin peaks ~418/542/577 nm) + right pulse duration (matched to vessel size) + right fluence → destroys the vessel, spares the skin.
- Melanin competes for the light, so darker skin needs longer wavelengths (Nd:YAG 1064 nm), lower fluence, and more cooling.
- PDL (585–595 nm) is the gold standard for superficial vascular lesions; KTP for fine vessels; IPL for diffuse redness; Nd:YAG for deeper/larger vessels and darker skin.
- Nd:YAG reaches deepest and is safest in dark skin but is the least forgiving — start conservative.
- Endpoints: PDL purpura (or sub-purpuric darkening); vessel spasm/disappearance for KTP/Nd:YAG. Stop before whitening, blistering, or bleeding.
- Safety: eye protection, cooling, test spots in dark skin, untanned skin only, photoprotection, realistic multi-session expectations.
- Always confirm a benign vascular diagnosis first; refer AVMs and possible Sturge–Weber.

# *Part VI. Hair Loss*

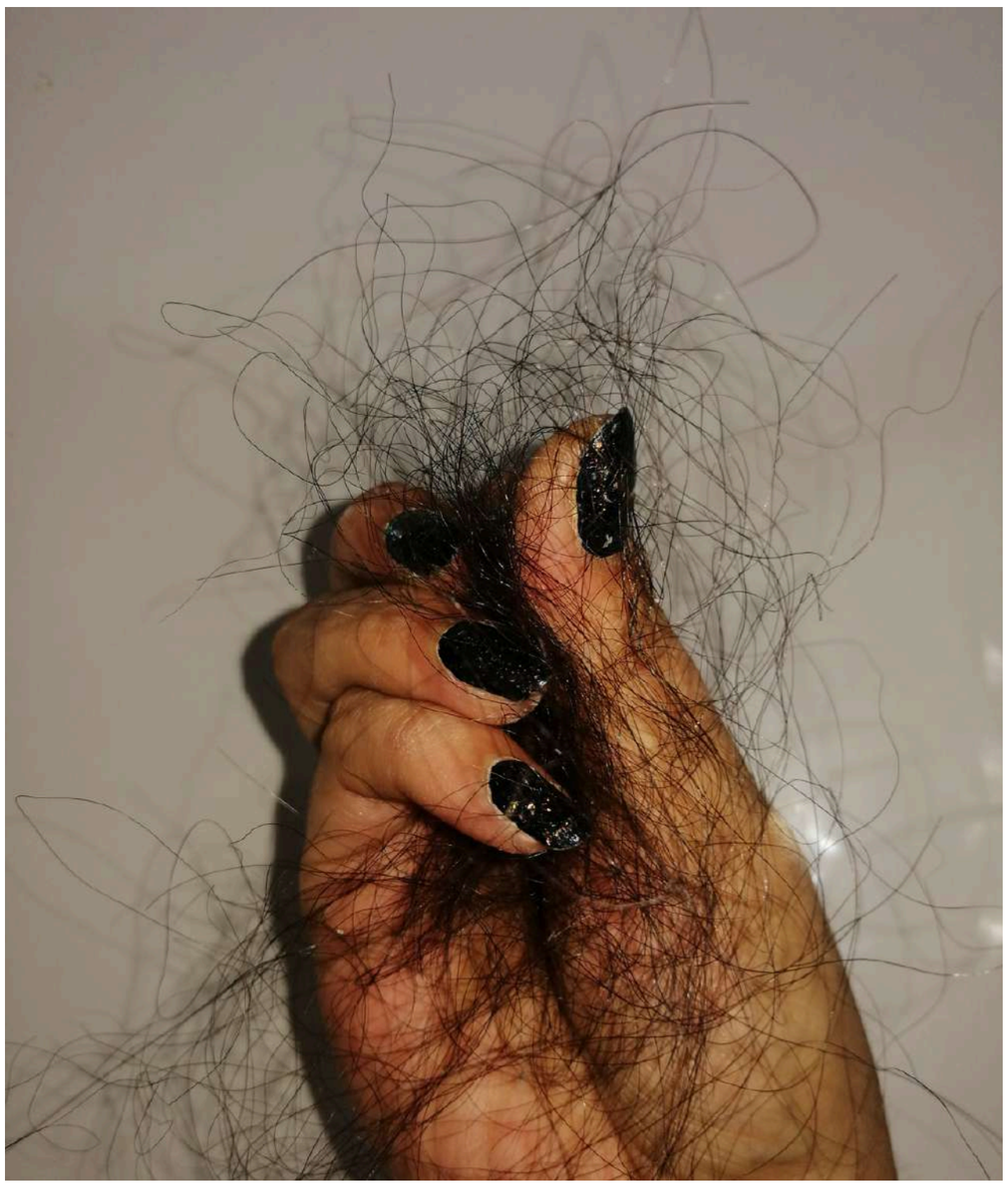


15



# HAIR LOSS: CLASSIFICATION AND PATHOPHYSIOLOGY

Hair loss is one of the most common — and most emotionally charged — complaints in aesthetic practice. The first job is always the same: work out what kind of hair loss it is, because the treatments are completely different and some causes are reversible while others aren't. This chapter covers how hair grows, the main categories of loss, and what drives the commonest type.



## 15.1 Hair follicle anatomy and the growth cycle

Each hair grows from a follicle, and every follicle cycles continuously through three phases:

| Phase | What happens | Duration | Share of scalp hairs |
|---|---|---|---|
| Anagen | Active growth | 2–7 years | ~85–90% |
| Catagen | Brief transition; growth stops | ~2–3 weeks | ~1% |
| Telogen | Resting, then the hair sheds | ~3 months | ~10–15% |

**Table 15.1 — The hair growth cycle**

Because the phases are unsynchronized, we shed ~50–100 hairs a day normally. Two facts drive most of hair-loss medicine:

- Treatments work by keeping follicles in (or pushing them back into) anagen. That's why they take months to show — you're waiting for new growth cycles.
- The key distinction is scarring vs. non-scarring loss. In non-scarring loss the follicle survives and regrowth is possible; in scarring (cicatricial) loss the follicle is destroyed and replaced by scar — regrowth is impossible, so the goal is to stop progression.

> 🔑 **Pearl**
>
> look for the follicular openings. In non-scarring loss you can still see the tiny pore openings on the scalp (the follicle is alive). In scarring loss those openings are gone — smooth, shiny scalp. Scarring alopecia is a "treat now to save what's left" emergency; refer to a dermatologist. Don't waste months on minoxidil while follicles are being destroyed.

## 15.2 Classification of hair loss

| Type | Pattern | Scarring? | Key features |
|---|---|---|---|
| Androgenetic alopecia (AGA) | Men — temples/crown; women — diffuse crown, frontline preserved | No | Most common; genetic + hormonal; gradual |
| Telogen effluvium | Diffuse, all over | No | Excess shed 2–3 months after a trigger (illness, childbirth, stress, crash diet); usually recovers |
| Alopecia areata | Round, well-defined bald patches | No | Autoimmune; sudden; "exclamation-mark" hairs; can regrow |
| Traction alopecia | Along hairlines / where hair is pulled | Early no → late yes | From tight styles; reversible early, scarring if chronic |
| Scarring (cicatricial) alopecias | Variable; loss of follicular openings | Yes | e.g. lichen planopilaris, frontal fibrosing alopecia, CCCA; urgent dermatology |

**Table 15.2 — Main types of hair loss**

The two you'll see most are androgenetic alopecia (the focus of the treatment chapters) and telogen effluvium (usually self-limiting — find the trigger and reassure).

## 15.3 Pathophysiology of androgenetic alopecia

Androgenetic alopecia (male- and female-pattern hair loss) is driven by genetics plus the hormone DHT:

- In genetically susceptible follicles, dihydrotestosterone (DHT) — made from testosterone by 5α-reductase — binds the follicle and triggers miniaturization: with each cycle the follicle shrinks, anagen shortens, and hairs become finer, shorter, and lighter until they barely grow.
- This is why the two most effective drug classes target exactly these steps: minoxidil prolongs anagen and enlarges follicles; 5α-reductase inhibitors (finasteride, dutasteride) cut DHT production.
- The pattern differs by sex by where the sensitive follicles sit: men lose at the temples and crown (Norwood–Hamilton scale); women thin diffusely over the crown while usually keeping the frontal hairline (Ludwig scale).

The occipital (back of the scalp) follicles are genetically DHT-resistant — which is why they're never lost in pattern baldness, and why they're the donor source for transplants.

### 🔑 Pearl

rule out the mimics before treating "pattern" loss. A woman with sudden diffuse shedding may have telogen effluvium (look for a trigger 2–3 months earlier), iron deficiency, or thyroid disease — not AGA. Basic bloods (ferritin, thyroid, and a hormonal screen if there are signs of excess androgens) save patients from being treated for the wrong thing.

### Key points

- Hair cycles through anagen (growth, years), catagen (brief), and telogen (rest/shed); ~85–90% are in anagen and we shed 50–100/day normally.
- The crucial split is scarring vs. non-scarring: non-scarring can regrow; scarring destroys follicles permanently and needs urgent dermatology. Check whether follicular openings remain.
- Common types: androgenetic alopecia, telogen effluvium, alopecia areata, traction, and scarring alopecias.
- AGA = genetic susceptibility + DHT (via 5α-reductase) causing follicle miniaturization; men temples/crown, women diffuse crown.
- Occipital follicles are DHT-resistant — preserved in baldness and used as transplant donor hair.
- Work up diffuse loss (ferritin, thyroid, hormones) before assuming pattern hair loss.

16



# MEDICAL AND LIGHT-BASED THERAPIES

Medical treatment is the foundation of hair-loss management — and the honest headline is that it slows loss and partially regrows hair, but only while you keep using it. Stop, and the benefit fades over the following months. Set that expectation first; everything else follows from it.

## 16.1 Minoxidil and the medical options

Minoxidil

Minoxidil is the first-line, FDA-approved topical for both men and women. It prolongs the active growth phase and enlarges miniaturized follicles.

• Topical: 2% and 5% (5% foam or solution is standard). Apply to a dry scalp; expect an early increase in shedding in the first weeks (old hairs pushed out before new growth — a sign it's working). Results take 3–6 months; continue indefinitely.

• Low-dose oral minoxidil (LDOM): increasingly used off-label when topical is impractical or ineffective — roughly 0.25–5 mg/day (often 1.25–2.5 mg in men, 0.25–1 mg in women), titrated. Main side effect is unwanted hair elsewhere (hypertrichosis); cardiovascular effects are uncommon at low doses but warrant a check.



5α-reductase inhibitors (mainly men)

These cut DHT, the hormone driving miniaturization.

• Finasteride 1 mg/day — FDA-approved for male AGA; blocks type II 5α-reductase and lowers scalp/serum DHT by roughly two-thirds. Effective at holding and partially regrowing hair. Possible side effects: reduced libido, erectile dysfunction, lowered ejaculate volume — usually reversible on stopping; counsel about persistent symptoms and mood.

• Dutasteride 0.5 mg/day — blocks both type I and II 5α-reductase, so suppresses DHT more; approved for AGA in some countries (e.g. Japan, South Korea), off-label elsewhere; often used when finasteride is insufficient.

Hormonal options for women

For female-pattern loss, especially with signs of excess androgens (PCOS), anti-androgens like spironolactone (and, in some regions, cyproterone acetate) are used — the same hormonal logic as the acne chapter. Topical finasteride is an emerging lower-systemic-exposure option.

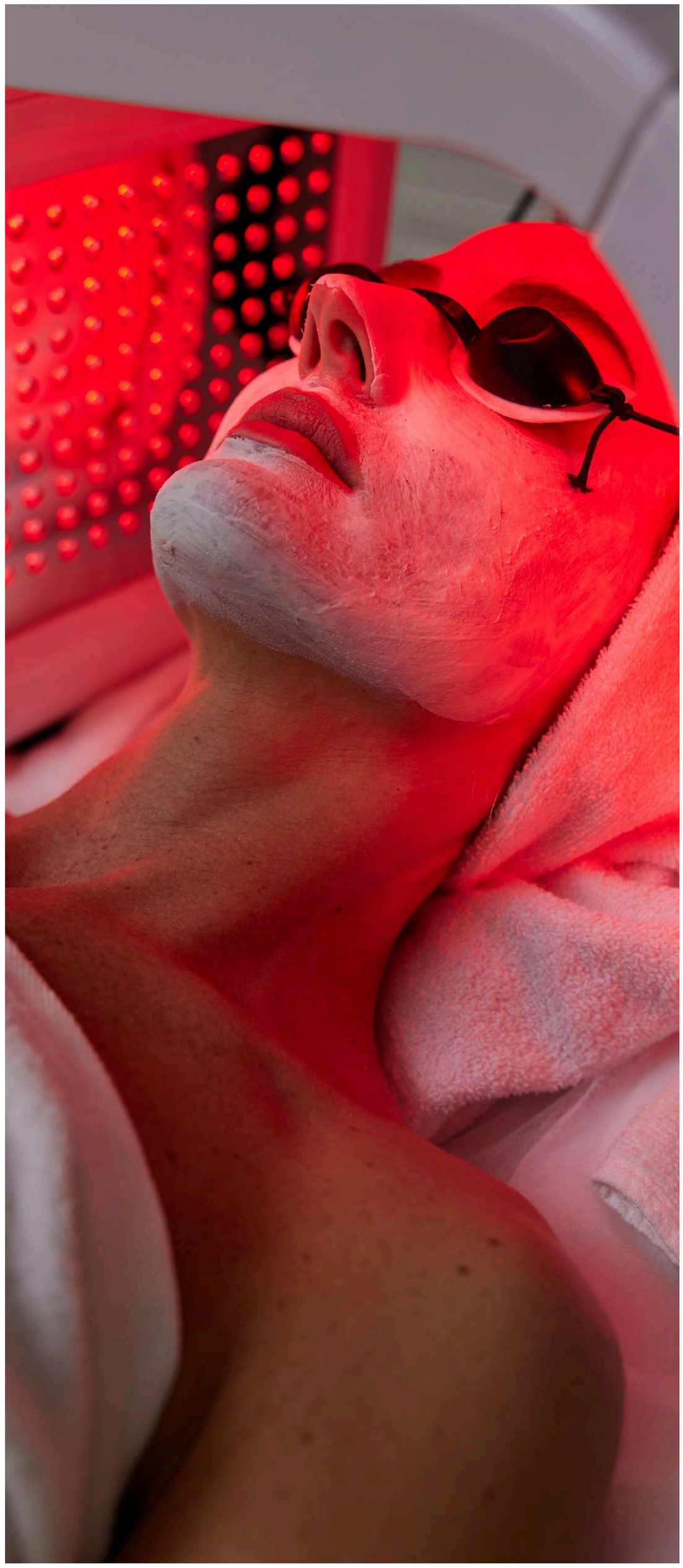
| Drug | Typical use | FDA-approved for AGA? | Notes |
|---|---|---|---|
| Topical minoxidil 2–5% | Men & women, first-line | Yes | Daily; results in 3–6 months; continue indefinitely; early shed is normal |
| Low-dose oral minoxidil | Off-label alternative | No | ~0.25–5 mg/day; hypertrichosis; check cardiovascular status |
| Finasteride 1 mg/day | Men, first-line oral | Yes | Lowers DHT ~⅔; sexual side effects possible; teratogenic |
| Dutasteride 0.5 mg/day | Men, when finasteride insufficient | No (approved in some countries) | Stronger DHT suppression; long half-life; teratogenic |
| Spironolactone | Women (esp. hyperandrogenic) | No | Anti-androgen; avoid in pregnancy |

**Table 16.1 — Medical therapies for androgenetic alopecia**

## 16.2 Light therapy

Low-level laser therapy (LLLT) — also called red-light or photobiomodulation therapy — uses red light (around 650–670 nm) to stimulate follicles, probably by boosting cellular energy and prolonging anagen.

- Delivered by FDA-cleared devices: combs, caps, and helmets for home use.
- Evidence: modest but real improvement in density, best as an adjunct to minoxidil and finasteride rather than a standalone.
- Use: typically several sessions per week over months; needs ongoing use to maintain.
- Safety: very safe, painless, no significant downtime — its main appeal.



🔑 Pearl

combination beats monotherapy, and adherence beats everything. The best medical results come from stacking mechanisms — e.g. topical minoxidil + finasteride (men) ± LLLT. But none of it works if the patient stops. Spend the consultation making sure they understand this is a long-term commitment, not a course they finish.

**⚠ Caution**

finasteride/dutasteride and pregnancy. These DHT-blockers can harm a developing male fetus. They are for men (or carefully counseled, contracepting women under specialist care); women who could become pregnant should not take them or even handle broken tablets.

**Key points**

- Medical treatment slows loss and partially regrows hair only while continued; stopping reverses the gains. Counsel this first.
- Topical minoxidil (2–5%) is first-line and FDA-approved for men and women; results take 3–6 months; an early shed is normal.
- Low-dose oral minoxidil (~0.25–5 mg/day) is an off-label alternative; main side effect is hypertrichosis.
- Finasteride 1 mg/day (FDA-approved, men) lowers DHT ~two-thirds; dutasteride 0.5 mg/day is stronger and off-label in most countries. Both are teratogenic.
- Women with hyperandrogenic pattern loss may use anti-androgens (spironolactone).
- Low-level laser therapy is a safe, modest adjunct, best combined with drugs. Combination therapy and long-term adherence drive results.

17

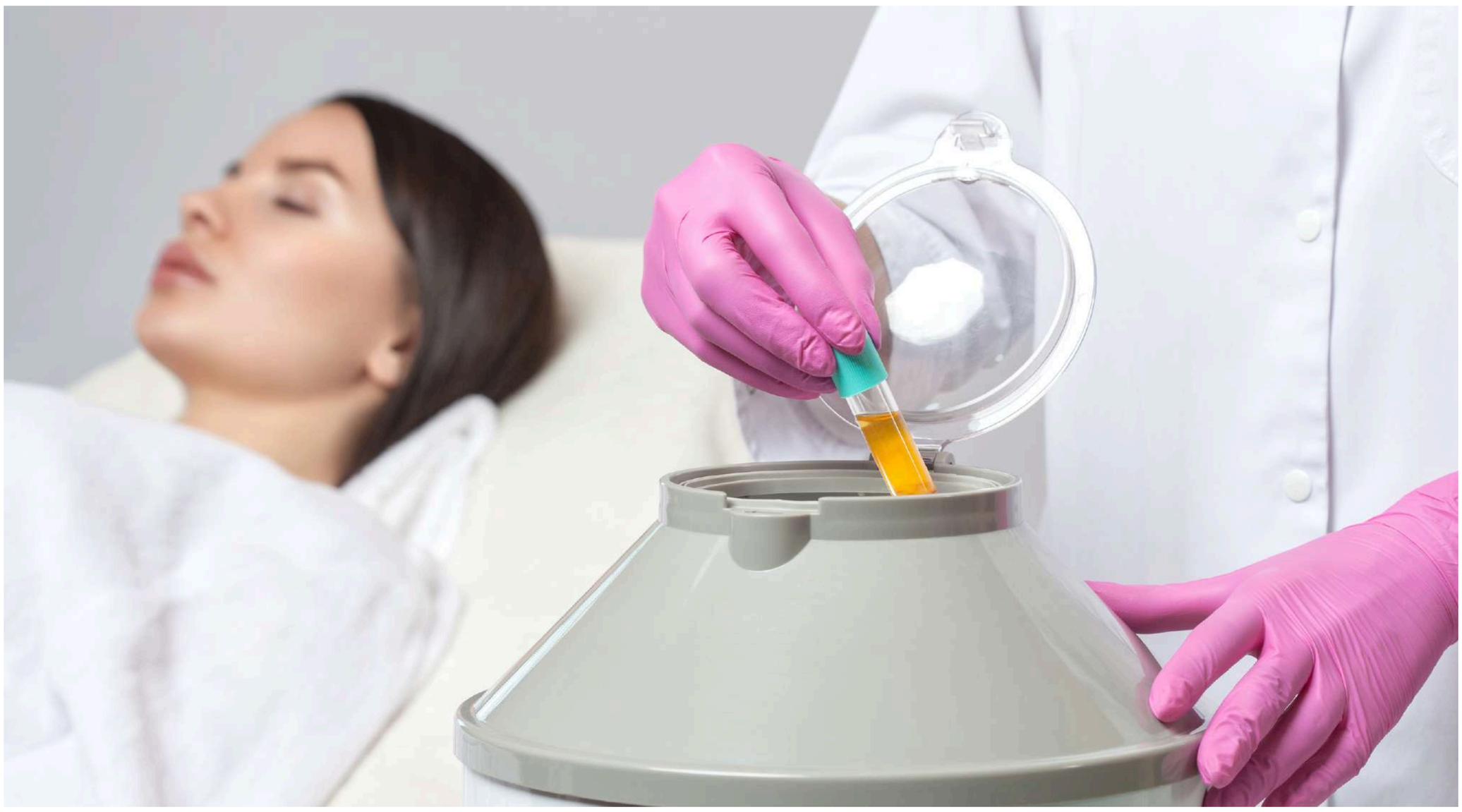


# REGENERATIVE AND PROCEDURAL THÉRAPIES

Beyond drugs and light sit the in-clinic regenerative procedures — using the body's own healing signals to wake up follicles. The two you'll use most are platelet-rich plasma and microneedling, often together and alongside medical therapy.

## 17.1 Platelet-rich plasma (PRP)

PRP uses a concentrate of the patient's own platelets, which are packed with growth factors (PDGF, VEGF, TGF-β, IGF-1 and others) that stimulate follicles, improve their blood supply, and prolong the growth phase.
How it's done: draw the patient's blood, spin it in a centrifuge to concentrate the platelets, then inject the platelet-rich layer into the scalp at the level of the follicles. A typical protocol: a session every month for 3–4 months, then maintenance every 3–6 months. There is no single standardized protocol — preparation methods and schedules vary between systems and studies.
Evidence and expectations: PRP has reasonable evidence for improving density in androgenetic alopecia, best as an adjunct to minoxidil/finasteride rather than a replacement. It works better the more viable (miniaturized but alive) follicles remain — so



earlier is better, and it won't regrow hair from bald, scarred scalp.

Practical points: minimal downtime (soreness, brief redness); because it's autologous, allergy is essentially a non-issue; avoid in active scalp infection or certain blood disorders.

> 🔑 **Pearl**
>
> PRP is an amplifier, not a substitute. It works best on a scalp that still has living, miniaturized follicles and alongside medical therapy. Set the expectation as "thicker, healthier hair and slower loss," not "new hair where it's been bald for years."

## 17.2 Microneedling for hair restoration

Microneedling — the same controlled-injury principle from the scar chapters — creates tiny channels in the scalp that trigger wound-healing growth factors and Wnt/β-catenin signalling (a key hair-growth pathway), and also help topical drugs penetrate.

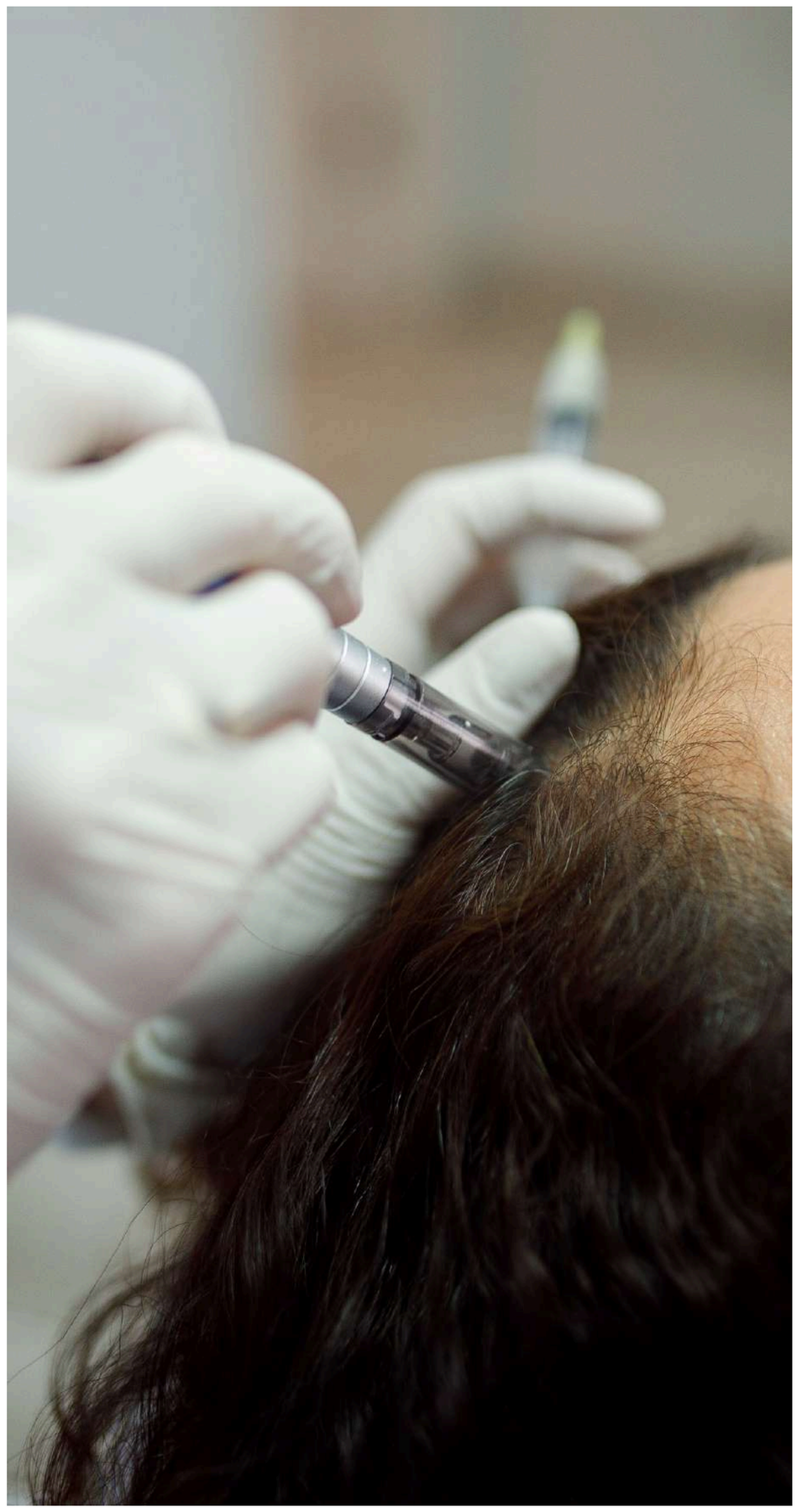
- Technique: roller or pen, typically around 0.5–1.5 mm depth on the scalp, every 1–4 weeks.
- Biggest value is in combination: microneedling + topical minoxidil consistently outperforms minoxidil alone in trials, and it pairs naturally with PRP in the same session.
- Safety: minor; transient redness; avoid over active infection or scarring alopecia.

> 🔑 **Pearl**
>
> stack the regeneratives. The strongest in-clinic results come from combining — e.g. microneedling + PRP in one visit, on a patient already using topical minoxidil (± finasteride). Each works through a different pathway; together they add up.



## Key points

- PRP delivers the patient's own concentrated growth factors into the scalp; typical schedule monthly ×3–4 then maintenance every 3–6 months (no single standardized protocol).
- PRP works best as an adjunct to medical therapy and on scalps that still have living, miniaturized follicles — not on bald, scarred areas.
- Microneedling (~0.5–1.5 mm) triggers growth-factor pathways and boosts drug penetration; microneedling + minoxidil beats minoxidil alone.
- Both are minimal-downtime, low-risk; avoid over active infection or scarring alopecia.
- Combine the regeneratives with each other and with drugs for the best results.



18



# HAIR TRANSPLANTATION

Transplantation is the only treatment that puts hair where there is none — by moving the patient's own DHT-resistant follicles from the back of the scalp to the thinning areas. It's the most definitive option, but it's surgery, it's not for everyone, and its success depends heavily on selecting the right patient and continuing medical therapy afterward.

## 18.1 Patient selection

The whole operation rests on the donor-recipient principle: follicles from the occipital "safe zone" are genetically resistant to DHT, so when moved to a bald area they keep their resistance and continue to grow for life. Good candidates have:

- Stable or medically-controlled loss — transplanting into actively-shedding hair chases a moving target; stabilize with medication first.
- A good donor area — enough density at the back to harvest without thinning it.
- Realistic expectations — a transplant redistributes existing hair; it doesn't add to the total, so very advanced baldness with a poor donor can't be made truly dense.
- Pattern (not scarring or autoimmune) loss — and the right age (operating too young, before the pattern declares itself, risks an unnatural look as native hair recedes around the grafts).



**🔑 Pearl**

medication first, surgery second. A transplant doesn't stop the underlying process. If the patient isn't on minoxidil/finasteride, their native (untransplanted) hair keeps thinning around the grafts, leaving an odd, patchy result in a few years. Stabilize medically before and continue after — this is the single biggest determinant of a natural long-term outcome.

## 18.2 Techniques: FUT vs. FUE

Both methods move individual follicular units (natural groupings of 1–4 hairs); they differ only in how the donor hair is harvested.

| Feature | FUT (strip) | FUE (follicular unit excision) |
|---|---|---|
| Harvest | A strip of scalp is removed; units dissected under microscope | Individual units extracted one by one with a small punch |
| Donor scar | A single linear scar (hidden by hair) | Tiny scattered dot scars (less visible; allows shorter haircuts) |
| Yield | Efficient for many grafts; often best graft quality | More labour-intensive; spares a linear scar |
| Recovery | Slightly longer; sutures/staples to remove | Quicker; no linear closure |
| Best for | Large sessions; when short haircuts aren't a priority | Those wanting very short hair; smaller / scar-conscious cases |

**Table 18.1 — FUT vs. FUE**

## 18.3 Post-procedure care

First days: grafts are fragile. Avoid touching, rubbing, or knocking them; sleep semi-upright; handle gently per the surgeon's protocol.

- Washing: gentle, as instructed, usually from a few days in; no scrubbing.
- Scabbing/redness: normal for 1–2 weeks; the transplanted hairs then shed (expected "shock loss") before regrowing.
- The timeline that matters: transplanted hairs fall out within weeks, regrow from around 3–4 months, give meaningful density by 6–9 months, and reach the final result at about 12–18 months.
- Keep up medical therapy (minoxidil/finasteride) to protect the surrounding native hair, and follow sun and activity restrictions early.

**🔑 Pearl**

warn about the shed and the wait. "The new hairs will fall out in the first few weeks — that's expected and normal. They start regrowing around 3–4 months, and you'll see the real result at about a year." Patients who aren't told this are convinced the transplant failed at week 3.

### Key points

- Transplantation moves DHT-resistant occipital follicles to bald areas, where they keep growing for life — the only way to create hair where there is none.
- Select carefully: stable/controlled loss, a good donor area, realistic expectations, pattern (not scarring/autoimmune) loss, appropriate age.
- FUT (strip, single linear scar) and FUE (individual extraction, dot scars) differ only in harvesting; both relocate follicular units into angle/density-matched recipient sites.
- A transplant redistributes hair — it doesn't increase the total; very advanced loss with a poor donor can't be made dense.
- Medical therapy before and after is essential — otherwise native hair thins around the grafts.
- Expect transplanted hair to shed then regrow from 3–4 months, with the final result at 12–18 months.

# Appendices

## Appendix A. Glossary of Terms

Plain-language definitions of the terms used throughout this book.

| Term | Definition |
|---|---|
| 5α-reductase | The enzyme that converts testosterone into the more potent DHT; the target of finasteride and dutasteride. |
| Adipocyte | A fat cell. In adults these mainly change size, not number. |
| Anagen | The active growth phase of the hair cycle (years); ~85–90% of scalp hairs. |
| Androgen | Male-type hormone (e.g. testosterone, DHT) that drives oil production and follicle miniaturization. |
| Atrophic scar | A depressed scar from too little collagen during healing (ice-pick, boxcar, rolling). |
| Boxcar scar | A wider atrophic scar with sharp, vertical walls. |
| Catagen | The brief transition phase of the hair cycle. |
| Chromophore | The target in the skin that absorbs laser light (e.g. hemoglobin, melanin, water). |
| Cicatricial (scarring) alopecia | Hair loss in which follicles are permanently destroyed and replaced by scar. |
| Clascoterone | A topical anti-androgen for acne, acting at the sebaceous gland without body-wide hormone effects. |
| Comedo (comedone) | A blocked follicle — closed (whitehead) or open (blackhead); the basic acne lesion. |
| Cryolipolysis | Controlled cooling that selectively destroys fat cells to reduce a localized fat pocket. |

| Term | Definition |
| --- | --- |
| Cutibacterium acnes | The skin bacterium (formerly P. acnes) involved in inflammatory acne. |
| Deoxycholic acid | Depigmentation |
| Depigmentation | Complete loss of pigment (e.g. white vitiligo patches). |
| DHT (dihydrotestosterone) | A potent androgen that miniaturizes susceptible hair follicles and drives sebum. |
| Diascopy | Pressing a glass slide on a lesion to see if it blanches (vascular) or not (purpura). |
| Fitzpatrick skin type | A I–VI scale of skin colour and sun response; predicts pigment-related risk (see Appendix B). |
| Follicular unit | A natural grouping of 1–4 hairs; the unit moved in transplantation. |
| FUE / FUT | Follicular Unit Excision (individual extraction) and Follicular Unit Transplantation (strip harvest). |
| GAGS | Global Acne Grading System — a detailed, weighted severity score. |
| Hemangioma | A benign vascular tumour of infancy that grows then usually self-resolves. |
| Hydroquinone | A tyrosinase-inhibiting skin-lightening agent used in melasma (prescription strength). |
| Hyperpigmentation | Excess pigment (e.g. melasma, PIH, lentigines). |
| Hypertrophic scar | A raised scar that stays within the original wound boundaries. |
| Hypopigmentation | Reduced (not complete) loss of pigment. |
| Ice-pick scar | A narrow, deep, V-shaped atrophic scar. |

| Term | Definition |
| --- | --- |
| IGA | Investigator's Global Assessment — a fast 0–4 acne severity scale. |
| IPL | Intense pulsed light — broadband light (not a true laser) for redness and superficial pigment. |
| Isotretinoin | Oral retinoid; the most powerful acne treatment and the only one that can cure. Teratogenic. |
| Keloid | A raised scar that extends beyond the original wound; strongly genetic; recurs. |
| Melanin / melanocyte / melanosome | Skin pigment / the cell that makes it / the organelle it's made in. |
| Melasma | Chronic, relapsing symmetrical facial hyperpigmentation driven by UV, hormones, and genetics. |
| Microcomedone | The earliest, invisible acne lesion; forms weeks before a visible spot. |
| Minoxidil | A hair-growth drug that prolongs anagen; topical (FDA-approved) or low-dose oral (off-label). |
| MRF | Microneedling radiofrequency — needles plus RF heat to remodel scars and tighten skin. |
| Nd:YAG | A 1064 nm laser; reaches deep vessels and is safest for darker skin. |
| PDL | Pulsed dye laser (585–595 nm); gold standard for superficial vascular lesions. |
| Photothermolysis (selective) | Destroying a target with light by matching wavelength, pulse duration, and energy. |
| PIE / PIH | Post-inflammatory erythema (redness) / hyperpigmentation (dark marks) after inflammation. |
| Pilosebaceous unit | The follicle + sebaceous (oil) gland + tiny muscle where acne begins. |
| Port-wine stain | A capillary malformation; a flat red patch present from birth. |

| Term | Definition |
|---|---|
| PRP | Platelet-rich plasma — the patient's own concentrated growth factors, injected to stimulate follicles. |
| Rolling scar | A wide, shallow atrophic scar tethered from below by fibrous bands. |
| Sebum / sebaceous gland | Skin oil / the gland that makes it. |
| Subcision | Cutting the fibrous bands that tether a scar or cellulite dimple so the surface can lift. |
| TCA CROSS | Focal high-strength trichloroacetic acid applied into ice-pick scars. |
| Telangiectasia | Permanently dilated small surface blood vessels. |
| Telogen / telogen effluvium | The resting/shevdding hair phase / diffuse shedding after a trigger. |
| Th17 inflammation | The immune pattern central to inflammatory acne. |
| Thermal relaxation time | How long a target takes to cool; the pulse must be shorter to confine heat. |
| Tranexamic acid | An anti-fibrinolytic used (off-label) orally or topically for melasma. |
| Triamcinolone | An injectable corticosteroid for hypertrophic and keloid scars. |
| Tyrosinase | The rate-limiting enzyme of melanin production; the target of most skin-lightening agents. |
| Vitiligo | Autoimmune destruction of melanocytes causing white patches. |

# Appendices

## Appendix B. Fitzpatrick Skin Type Chart

The Fitzpatrick scale classifies skin by its response to sun. It's one of the most useful tools in cosmetic dermatology: types IV–VI carry a higher risk of post-inflammatory hyperpigmentation (PIH) and scarring, and need more conservative laser, peel, and energy settings.

| Type | Typical appearance | Sun response | Clinical relevance |
|---|---|---|---|
| I | Very pale skin, often red/blond hair, freckles | Always burns, never tans | Lowest PIH risk; highest skin-cancer/photoaging risk |
| II | Fair skin, light eyes | Burns easily, tans minimally | Low PIH risk |
| III | Medium / olive-tinged | Sometimes burns, gradually tans | Moderate PIH risk — start to take care with energy devices |
| IV | Olive / light brown | Burns minimally, tans easily | Higher PIH risk; conservative settings, test spots |
| V | Brown | Rarely burns, tans darkly | High PIH risk; longer wavelengths, low fluence, strong cooling |
| VI | Deeply pigmented dark brown to black | Never burns, always tans | Highest PIH/dyschromia risk; greatest caution; Nd:YAG preferred for vascular |

How to use it: Record the Fitzpatrick type at the first visit. For types IV–VI: favour longer-wavelength lasers (e.g. Nd:YAG), lower energies, generous cooling, mandatory test patches, and aggressive photoprotection before and after any pigment or energy procedure.

# Appendices

## Appendix C. Patient Consent Form Templates

⚠ Not legal advice — adapt before use. These are generic templates to illustrate structure only. Consent requirements vary by country, state, and profession, and consent forms are legal documents. Before using any of these, have them reviewed by your own legal advisor, regulator, and insurer, and tailor them to the specific device or product (and its instructions for use) you are using.

### C.1 General cosmetic procedure consent (template)

Suitable as a base for laser/light, chemical peel, microneedling, RF, injectable, and similar procedures — add procedure-specific risks.

Patient name: _____ Date of birth: _____ Date: _____

Procedure: _____ Area(s) treated: _____

**I confirm that:**

- The nature, purpose, and expected benefits of the procedure have been explained to me, including that results vary between individuals and that more than one session may be required.
- I understand the alternatives, including no treatment, and the likely outcome of each.
- The common and serious risks have been explained, which may include (as applicable): redness, swelling, bruising, discomfort, blistering, infection, temporary or permanent changes in skin colour (lighter or darker), scarring, incomplete results, and the need for maintenance.
- I have disclosed my full medical history, including medications, allergies, pregnancy or breastfeeding status, recent sun exposure/tanning, history of cold sores, keloid tendency, and any prior cosmetic treatments.
- I have had the opportunity to ask questions, and they have been answered to my satisfaction.
- I understand the aftercare instructions and agree to follow them, including sun protection.

• I consent to clinical photographs being taken for my medical record (and, separately, for education/marketing only if I initial here: ____).

Patient signature: _____ Practitioner signature: _____

## C.2 Procedure-specific risk inserts

Add the relevant block to the general form above.

**Isotretinoin (oral)**

Severe, guaranteed birth defects if taken in pregnancy; mandatory contraception and pregnancy testing; dryness; mood changes; blood-test monitoring. (Follow your jurisdiction's pregnancy-prevention programme.)

**Laser / energy device**

Burns, blistering, pigment change (especially in darker skin), scarring, eye injury (eye protection required), incomplete clearance, need for multiple sessions; no recent tan; possible paradoxical darkening of certain tattoo inks.

**Chemical peel**

Stinging, redness, peeling, pigment change, infection, scarring (deeper peels); strict sun avoidance afterward.

**Injectable (filler / deoxycholic acid / PRP / steroid)**

Bruising, swelling, lumps, infection, asymmetry; for deoxycholic acid near the jaw, temporary nerve-related asymmetric smile; for fillers, rare vascular occlusion. PRP uses your own blood (drawn on the day).

# Appendices

## Appendix D. Pre- & Post-Treatment Patient Handouts

For patients. These are plain-language handouts you can copy and adapt. Always defer to the specific instructions for the device/product used and your clinic's protocol.

### D.1 Before your treatment (general)

- Avoid sun and sunbeds for at least 2 weeks before — do not arrive with a tan.
- Stop self-tanner at least 1 week before.
- Tell us about any cold-sore history, recent medications (including isotretinoin), pregnancy, or tendency to thick/raised scars.
- Avoid retinoids/exfoliating acids for a few days before, unless told otherwise.
- Arrive with clean skin, no make-up, where possible.

### D.2 After laser / energy or microneedling

- Expect redness, warmth, and mild swelling for a few days; pinpoint scabbing can occur.
- Keep the area clean; use gentle, fragrance-free cleanser and moisturizer.
- Apply broad-spectrum SPF 30+ every day — this is the most important step.
- Do not pick or scratch; let any scabs fall off naturally.
- Avoid heavy exercise, saunas, and hot showers for 24–48 hours.
- Contact us if you notice spreading redness, pus, or severe pain (possible infection).

### D.3 After a chemical peel

- Redness then flaking/peeling over several days is normal — do not pull the skin.
- Moisturize often and use SPF 30+ daily; avoid sun completely while peeling.
- Pause retinoids and exfoliating acids until fully healed.

### D.4 After an injectable (filler / PRP / deoxycholic acid)

- Bruising and swelling are common and settle over days (longer for deoxycholic acid).
- Avoid heavy exercise and alcohol for 24 hours.

• Use a cold pack for comfort; take paracetamol if needed (avoid aspirin/NSAIDs unless advised).
• Report severe pain, skin colour change, or vision changes immediately.

**D.5 Hair treatment (minoxidil / transplant)**

• Minoxidil: an early increase in shedding in the first weeks is normal and means it's starting to work; results take 3–6 months and only continue while you keep using it.
• After a transplant: the new hairs shed within weeks, regrow from 3–4 months, with the final result around a year — be patient.

# Appendices

## Appendix E. Quick-Reference: Procedures by Indication

**A rapid lookup of first-line options and adjuncts. Always individualize, confirm the diagnosis, and refer to the relevant chapter for detail and doses.**

| Indication | First-line | Adjuncts / notes |
|---|---|---|
| Acne (mild) | Topical retinoid + benzoyl peroxide | Add topical antibiotic (with BPO) if inflammatory |
| Acne (moderate) | Topical combo + oral antibiotic (3–4 mo) OR hormonal (women) | Always pair antibiotics with BPO |
| Acne (severe/scarring) | Oral isotretinoin | Strict pregnancy prevention |
| Ice-pick scars | TCA CROSS / punch excision | Resurfacing only as adjunct |
| Boxcar scars | Fractional laser or MRF | Punch elevation if deep floor |
| Rolling scars | Subcision first | Then MRF/laser ± filler |
| Hypertrophic / keloid scar | Intralesional triamcinolone ± 5-FU | Silicone; PDL for redness; don't excise keloid alone |
| Cellulite (dimples) | Subcision / acoustic subcision | 1440-nm laser; biostimulator filler |
| Cellulite (laxity) | Radiofrequency / microfocused ultrasound | Series of sessions |

# Appendices

| Indication | First-line | Adjuncts / notes |
|---|---|---|
| Localized fat pocket | Cryolipolysis; deoxycholic acid (chin only) | Near goal weight; not weight loss |
| Vitiligo | Topical steroid / calcineurin inhibitor; narrowband UVB | Ruxolitinib cream; combination best |
| Melasma | Tinted SPF + hydroquinone/triple-combo | Oral tranexamic acid; gentle procedures only |
| Solar lentigines | Q-switched/pico laser or IPL; peels | Confirm benign first |
| Tattoo | Q-switched / picosecond laser (match ink colour) | 6–12+ sessions; test for paradoxical darkening |
| Facial telangiectasia / rosacea redness | PDL or KTP; IPL for diffuse redness | 2–4 sessions |
| Port-wine stain | Pulsed dye laser | Many sessions; start young; rarely 100% |
| Leg spider veins | Sclerotherapy (first-line) ± Nd:YAG | Rule out venous insufficiency |
| Androgenetic alopecia | Topical minoxidil ± finasteride (men) | LLLT, PRP, microneedling; transplant if stable |
| Telogen effluvium | Find & treat the trigger; reassure | Usually self-resolves |

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# CBAM Aesthstic Mastery Series




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