



Healing Water. (40x60 inch acrylic on canvas.)

CHAPTER 1

THE ART OF HEALING

Why the Body Heals

Healing is as old as life itself. Every living thing must be able to heal to survive. As surgeons, it is not our touch that heals, as much as we may like to think it is. That may feel like a blow to the ego, but it is an invitation into a relationship with each patient. It is the body that heals. The cells and the tissue. It is designed to heal. As we study healing and master our craft, we are indeed participating in a much greater thing: life.

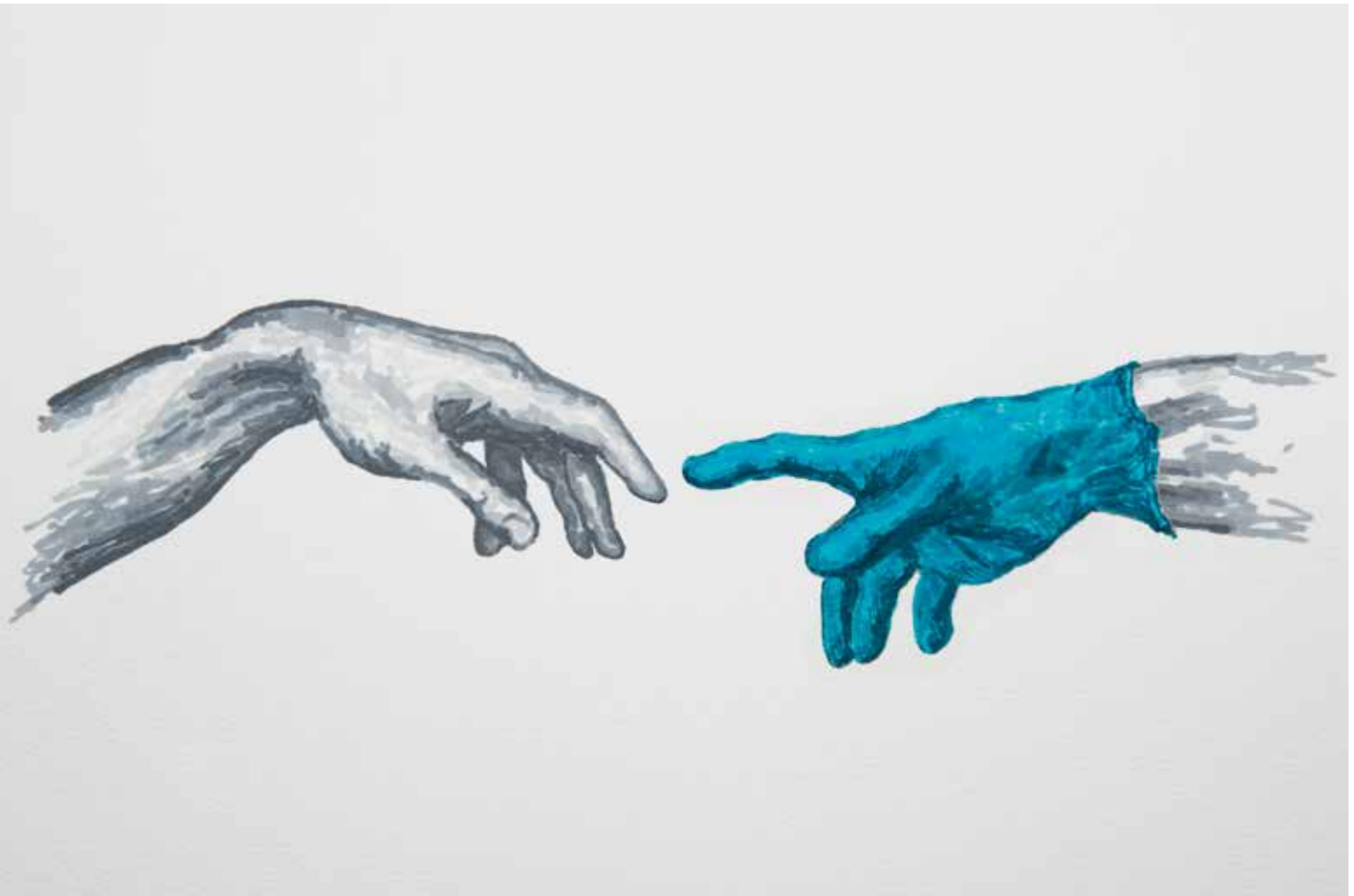


Figure 1-1 (Artwork inspired by Michelangelo.)

In oral surgery residency, we are brought up believing we have to know everything. That we are the end of the line and we just have to figure it out. The fear of being wrong and failure has been instilled in us. I'm not sure if it is our personality or our training that makes us that way, but I am pretty sure it makes surgeons a bit difficult to deal with at times. We like taking the simple and making it complicated. I do the opposite. I work to make the complicated simple and the simple even simpler. I have always been more interested in the questions, curious to learn and discover. Often, the beauty I find is in the depth and complexity, and yet the profound simplicity of life.

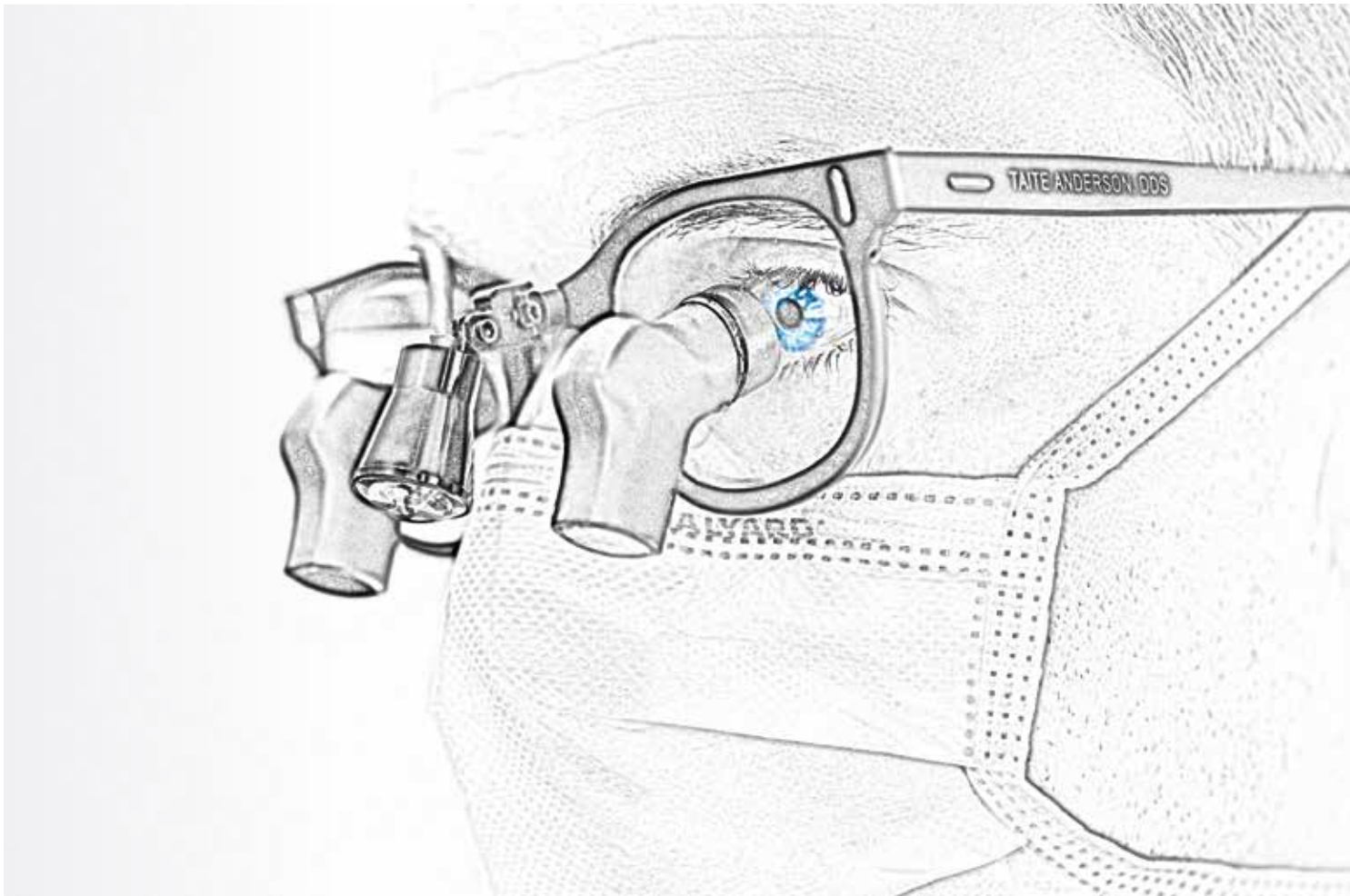


Figure 1-2 Self portrait. I want to share what I look for and what I see in surgery.

In my world as a surgeon, healing always begins with an injury. **We injure to heal.** We cut tissue and remove things that are causing problems—things the body is incapable of fixing on its own.

What makes the body heal? The cells, you may say. But the cells also cause an uproar, inflaming the tissue and destroying the body in the case of autoimmune disease. So, there must be something more. The cells are somehow guided along on their journey of healing.

Surgery is like Graffiti: Vandalism to some, and art to others. Cells see it as vandalism. They only see trauma. They respond to surgery the same way they respond to any injury. They need to heal. But **I see surgery as art.** It is the beginning of the healing that takes place after the patient leaves the surgery. The body already knows how to heal. It has been doing that since its beginning.

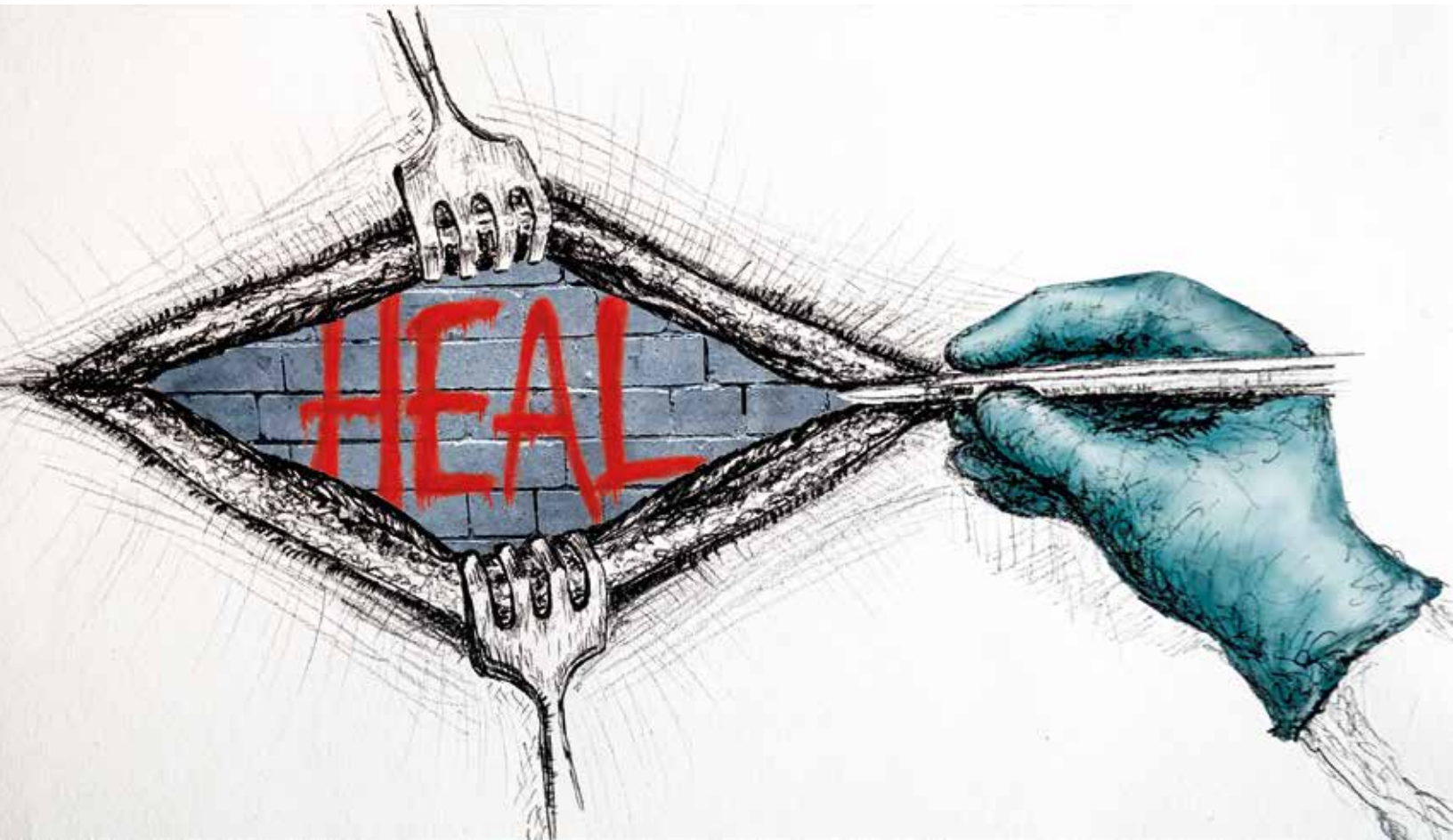


Figure 1-3 (Artwork inspired by Banksy).

The Body Heals: Basic Biology

Let's look at how we can leverage the art of healing in surgery. The most common surgical procedure on the planet is the extraction. Therefore, edentulism must be the most common surgical related defect. It is a problem.



Figure 1-4

Placing an implant after an extraction is our attempt to recreate nature, using our understanding of science to restore beauty and function. We want to make the patient whole again. Implants are the solution to the problem, and they only work when healing is successful.

The goal is not simply getting the tooth out or screwing an implant in place. Holding a tooth in the forceps is not the objective. We want to be kind to the tissue, causing as little trauma as possible, for the cells' sake! If we want to be successful with our implants, we need to pay attention to the details of healing. If we just yank a tooth and jam in a screw, it will fail a lot more often. Failure is really easy. Success is the prize worth pursuing.

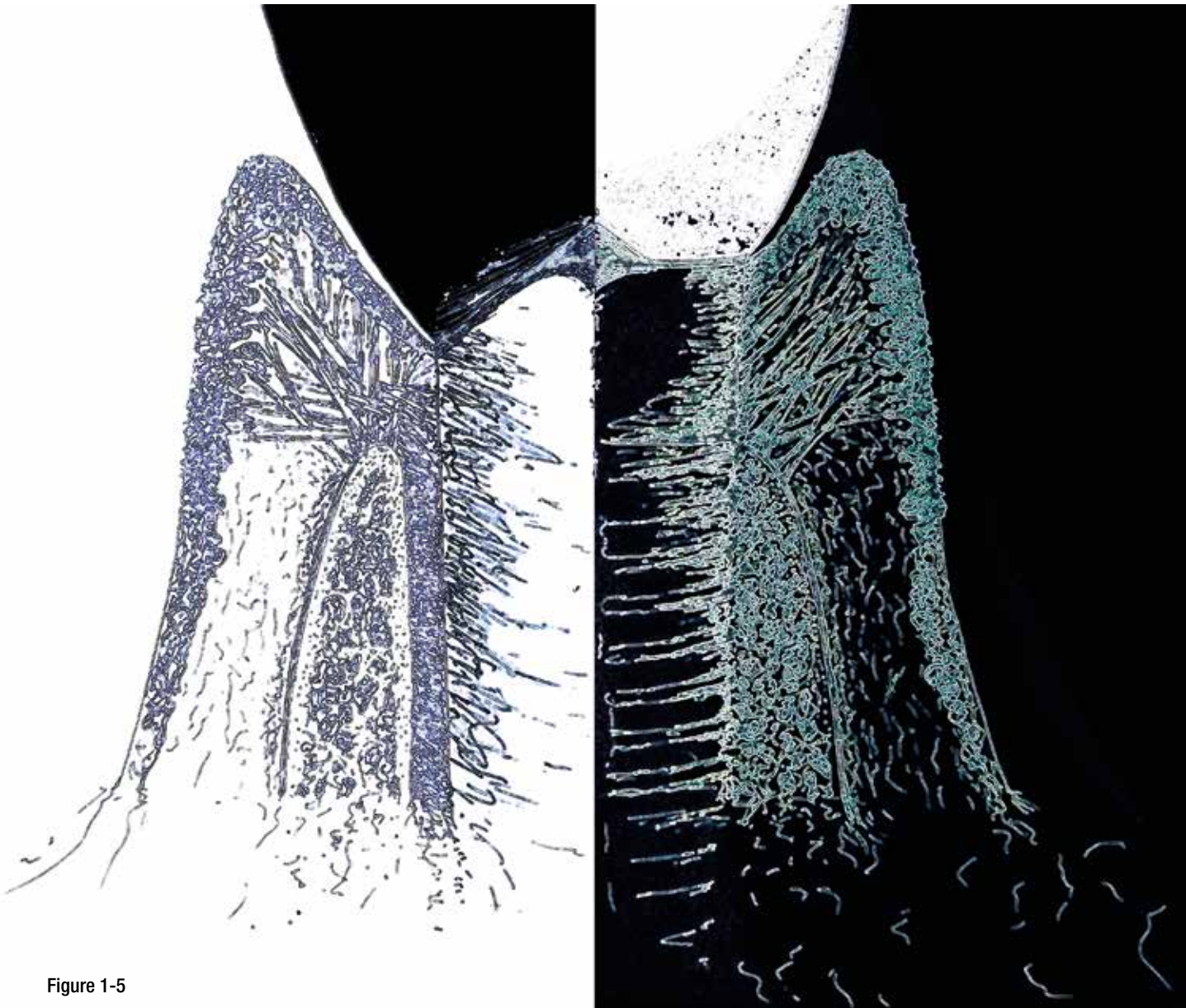


Figure 1-5

Let's look closer at what needs to happen in an immediate implant site for the screw to stay put, to integrate, and, ultimately, to succeed. Here's an extraction site with the hole prepped for an immediate implant. We can see the remnants of the periodontal ligament. If we look closer, we can see individual cells, like platelets.

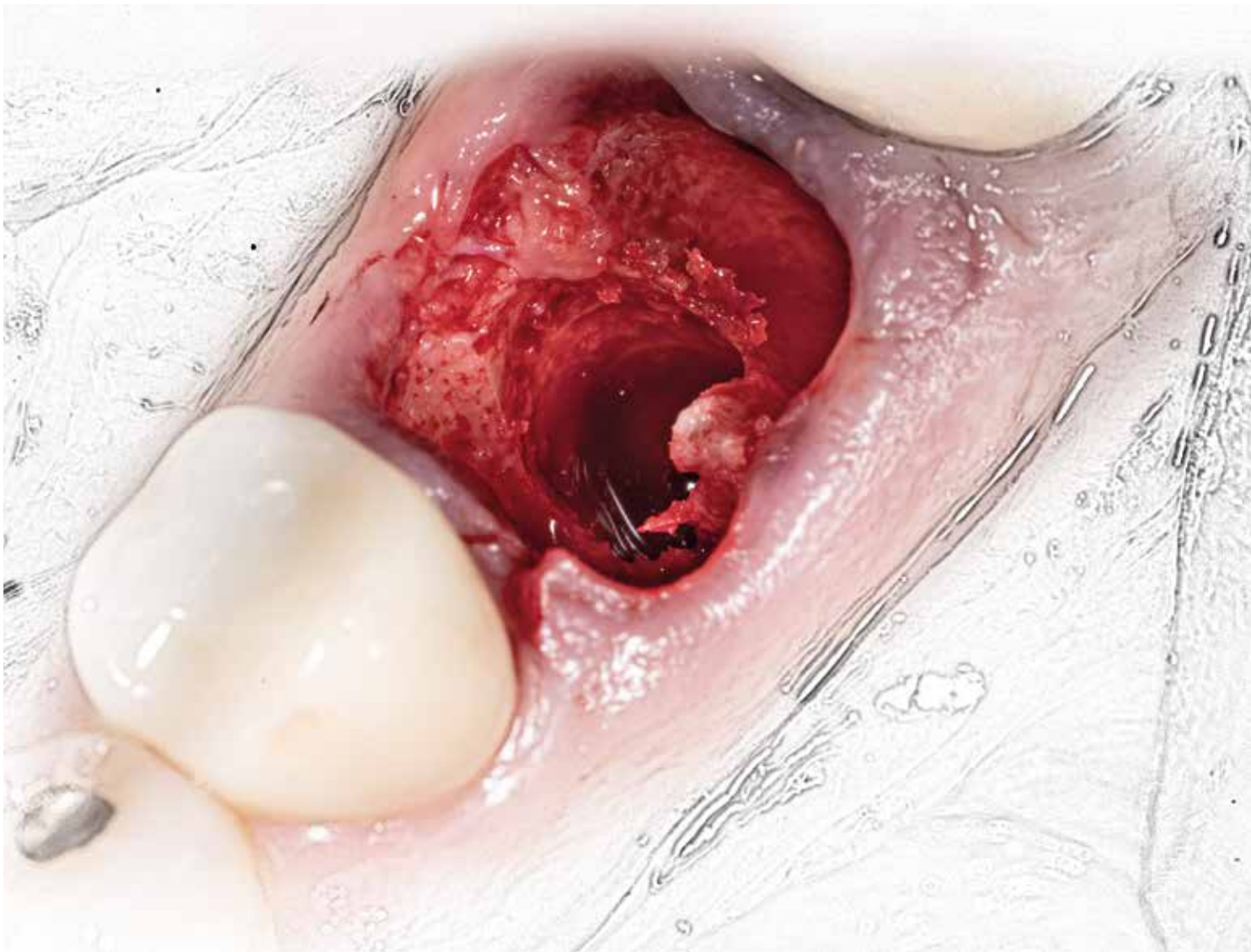


Figure 1-6



Figure 1-7 (Artwork inspired by Paul Cézanne.)

This is how I envision platelets. They usually hang out as simple, round, anucleate cytoplasmic cell bodies, but they are a powerhouse for healing. When activated, platelets grow sticky tentacles and release signaling proteins to stop bleeding and orchestrate healing.

Platelets play a big role in hemostasis, which is why we call them thrombocytes, thrombo for clotting and cyte for cell. But they are so much more than

just a clotting cell. They participate in all phases of wound healing: hemostasis, inflammation, proliferation, maturation. Platelets also have a role in immunity. They are a powerhouse for healing. Filled with sacs of signaling proteins, they are always ready to stimulate cell growth, differentiation, inflammation and repair. When released, the proteins biochemically attach to receptors on nearby cells, telling them precisely what to do. It is almost like they pull biological levers on the cells to make things happen.

**THROMBO
CYTE**

**CLOTTING
CELL**

Figure 1-8

Some of the proteins they release are called growth factors. These regulate and maintain cellular growth. We label them according to their primary roles. For instance, Vascular Endothelial Growth Factor (VEGF) triggers the formation of new blood vessels.

As dentists, we tend to see these acronyms and gloss over. All these growth factors seem too complex and like they don't really matter. But they do. They all work together to heal. They are part of the everyday life of our bodies. They are a part of us. Like Andy Warhol transformed everyday objects into works of art, I want to call attention to how important and beautiful these things are. Appreciate them.



Figure 1-9 (Artwork inspired by Andy Warhol.)

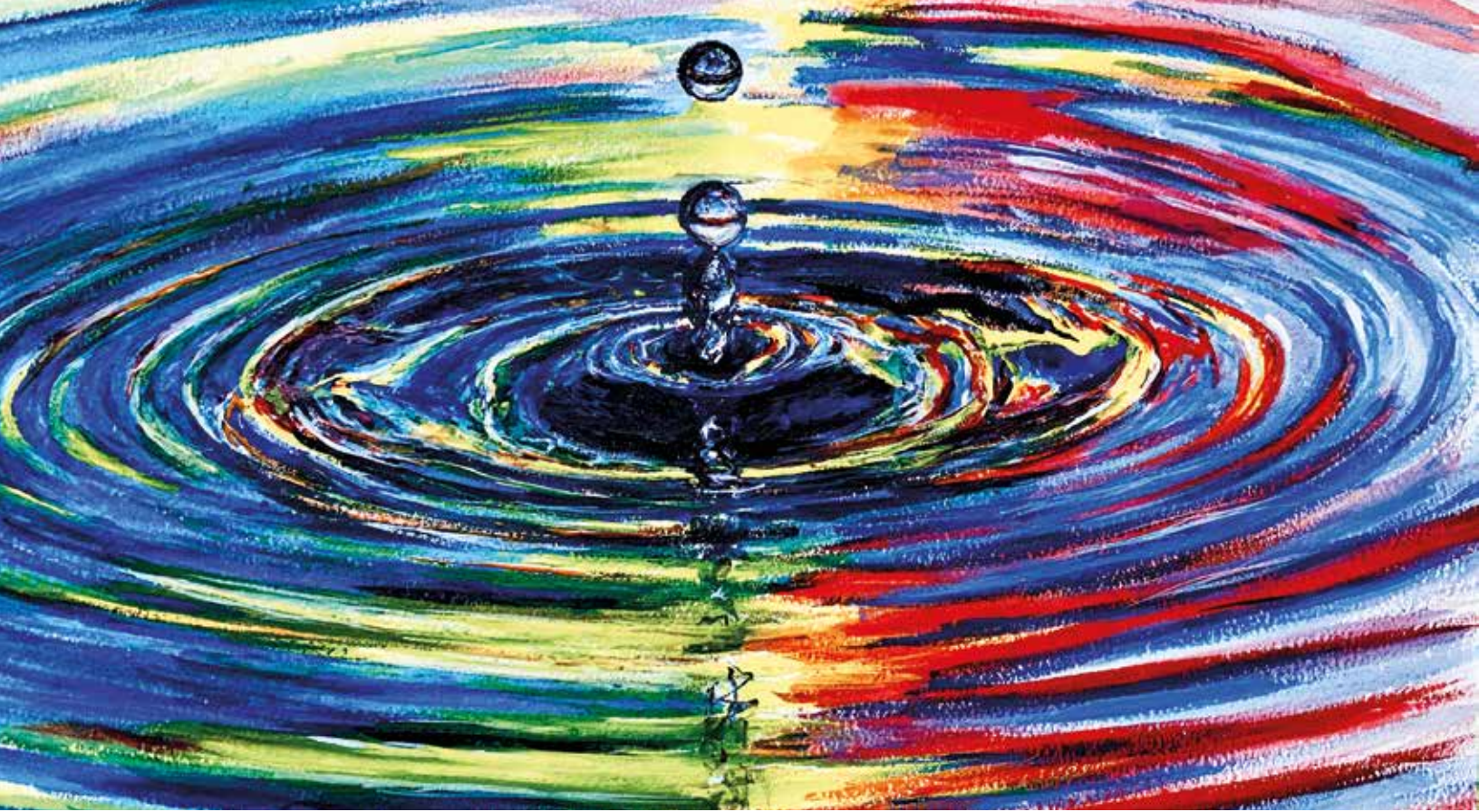


Figure 1-10 When growth factors are released, their signal ripples outward in all directions. Like ripples, growth factors themselves don't physically change much of anything. But they are directly responsible for so very much. The signal is amplified the closer you get to the droplet. Each small droplet impacts everything around it.

**Vascular Endothelial Growth Factor
VEGF**

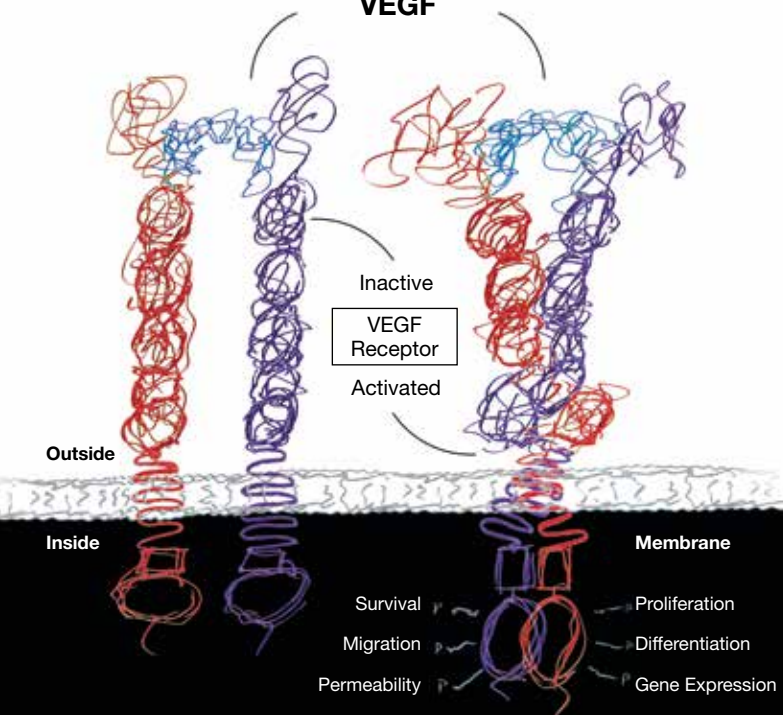


Figure 1-11

When VEGF is released from platelets (and other various sources) it spreads out through the extracellular space. It floats around for a while until it is grabbed by a perfectly matched dimeric protein structure sticking out from cell walls called a VEGF receptor. When this happens, the five outer dimers join and sort of warp around each other. As they contort and snap together, the whole complex twists into a new, more stable shape. This changes the shape inside the cell membrane, exposing an ATP binding site. Thus, a cascade of intracellular events is triggered, impacting gene expression and cell cycle regulation. And this is just one tiny step in the elaborate biochemical chain of events triggered by VEGF. It goes on and on.

Check this out. If you have a blood vessel next to an extraction site with an immediate implant, the platelets in the site release VEGF proteins. This signal causes some of the normal endothelial cells to transform themselves into highly specific tip and stalk cells. They completely change. The transformed tip cells lead the charge and grow antennae that follow the homing beacon toward the VEGF signal. This cell was just like the other endotheli-

al cells before. Right behind it, another endothelial cell transforms into a stalk cell. It becomes a factory producing a new 3D vessel sprouting toward the site. If we want healing next to our implant, we want to crank up the VEGF signal to form new vessels. And angiogenesis is just one of VEGF's many valuable healing effects. It also triggers fibroblasts and keratinocytes to promote collagen formation and healing.

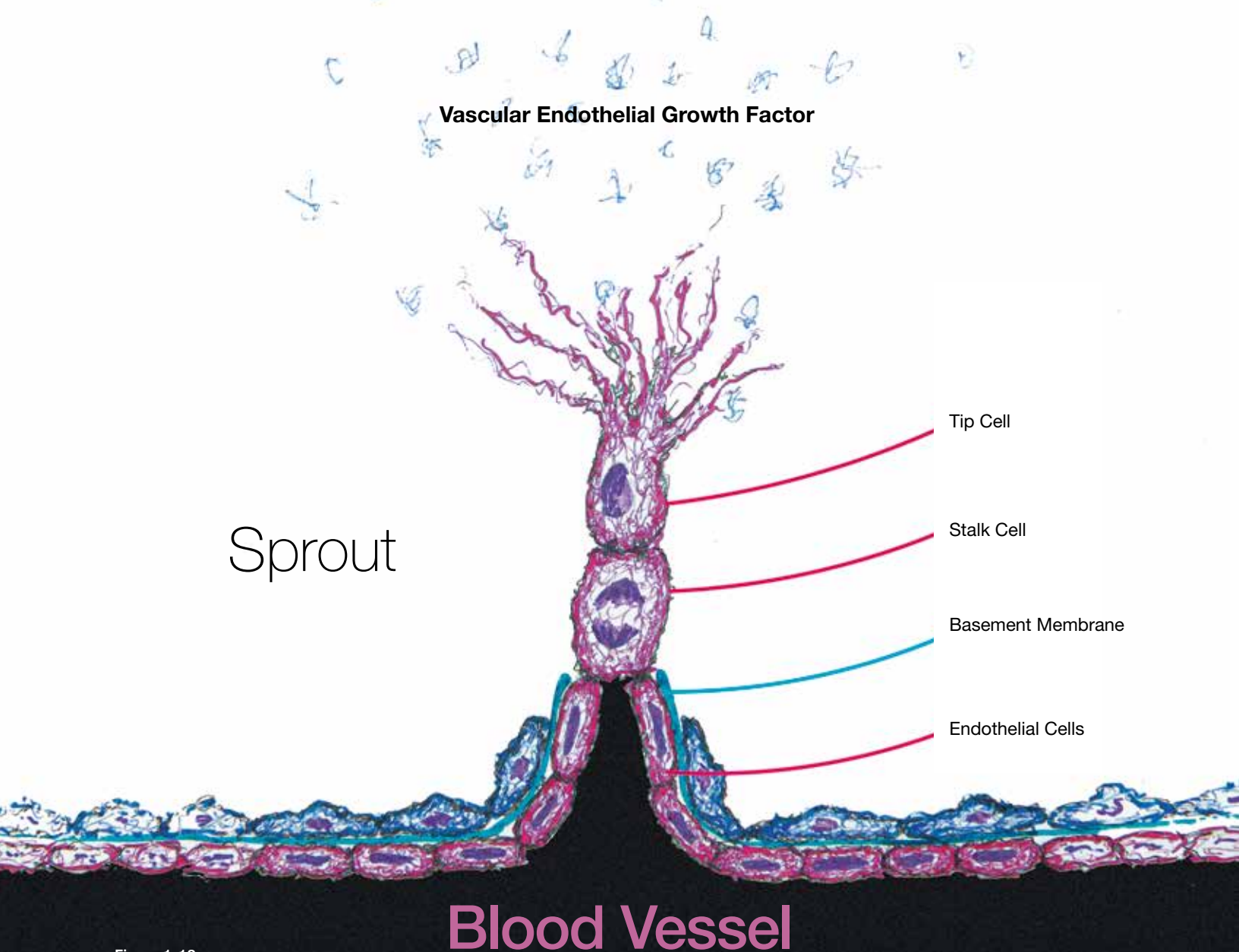


Figure 1-12



Figure 1-13

How do we heal through surgery? We start by viewing healing as the goal. An extracted tooth is not the goal. If getting the tooth out is the goal, we will inherently miss the boat on setting up for healing. During surgery, minimize trauma as much as possible with precise, calculated moves, and then let the body do its thing. It knows what to do. We study it. We learn from it. And we adapt what we do to accom-

modate it. We don't pound it into how we want it to be. (I wish we could. Wouldn't that be nice? "Pound to fit. Paint to match," my father-in-law used to say.)

But that doesn't work in healing. We want to get in, get the job done, and get out. We want to make the body think it was its own idea to heal in a way that meets our needs.



Plasma

Platelet
rich
fibrin

Red
blood
cells

Figure 1-14 (Artwork inspired by Mark Rothko.)

How can we crank up healing right where we want it? In this case, right at the implant-bone interface? One way is by using Platelet Rich Fibrin (PRF). PRF is the concentrated portion of a patient's own blood, turbocharged for healing. It is basically whole blood spun down, which separates the components by density. The red blood cells are the densest and they fall to the bottom. Plasma stays at the top as it is mainly water and electrolytes. And in the middle is the gold. Concentrated platelets, growth factors, white blood cells and fibrin. This is the PRF.

Placing the PRF in the extraction site feeds it a healthy diet of:

- Platelets to stop the bleeding and coordinate the healing process.
- Growth factors slowly released over several weeks.
- White blood cells to defend against bacterial invasion.
- A hearty fibrin mesh to support the tissue in growth.

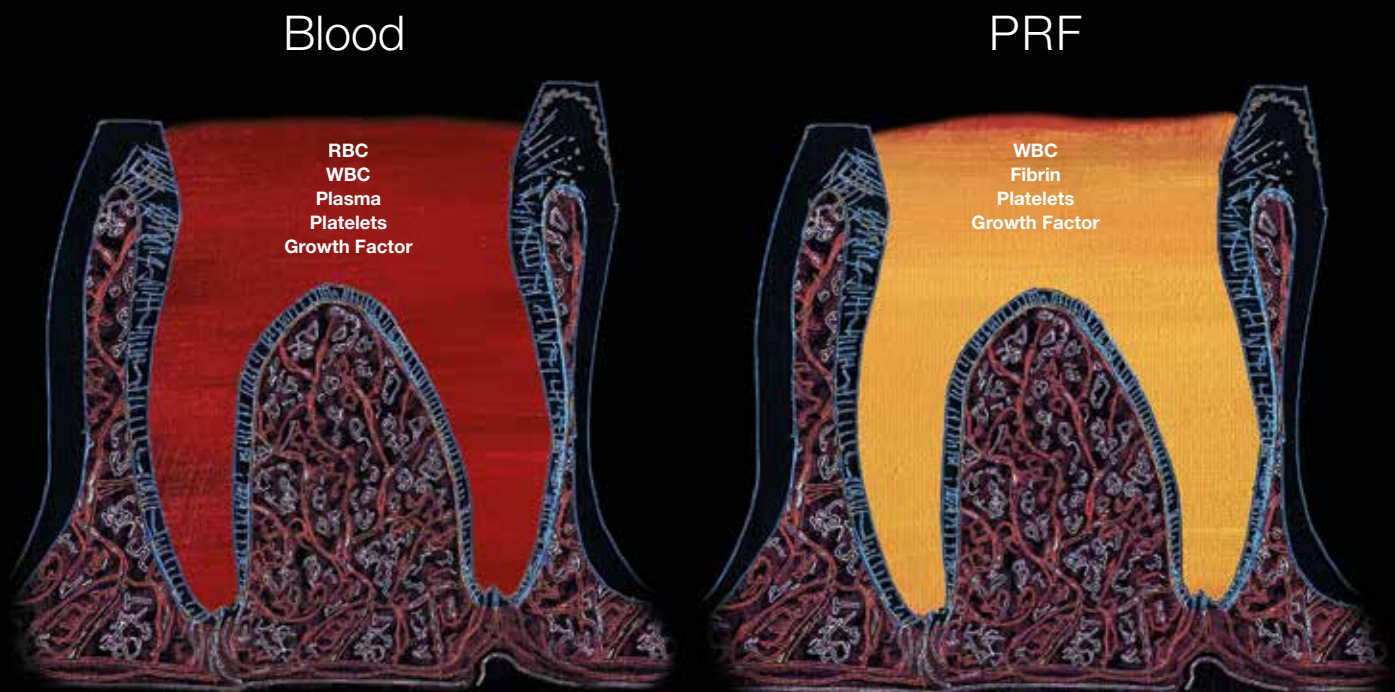


Figure 1-15

The extraction site doesn't need a regular blood clot. The red blood cells and plasma just dilute the healing potential. But it desperately needs platelets, leukocytes, and growth factors. It needs to lay down a framework

for rebuilding, a fibrin mesh of protection. Instead of relying on whole blood, which also contains a bunch of non-healing related components, we spin down the blood and pluck out the good stuff. The gold.

We want the gold next to our implants.

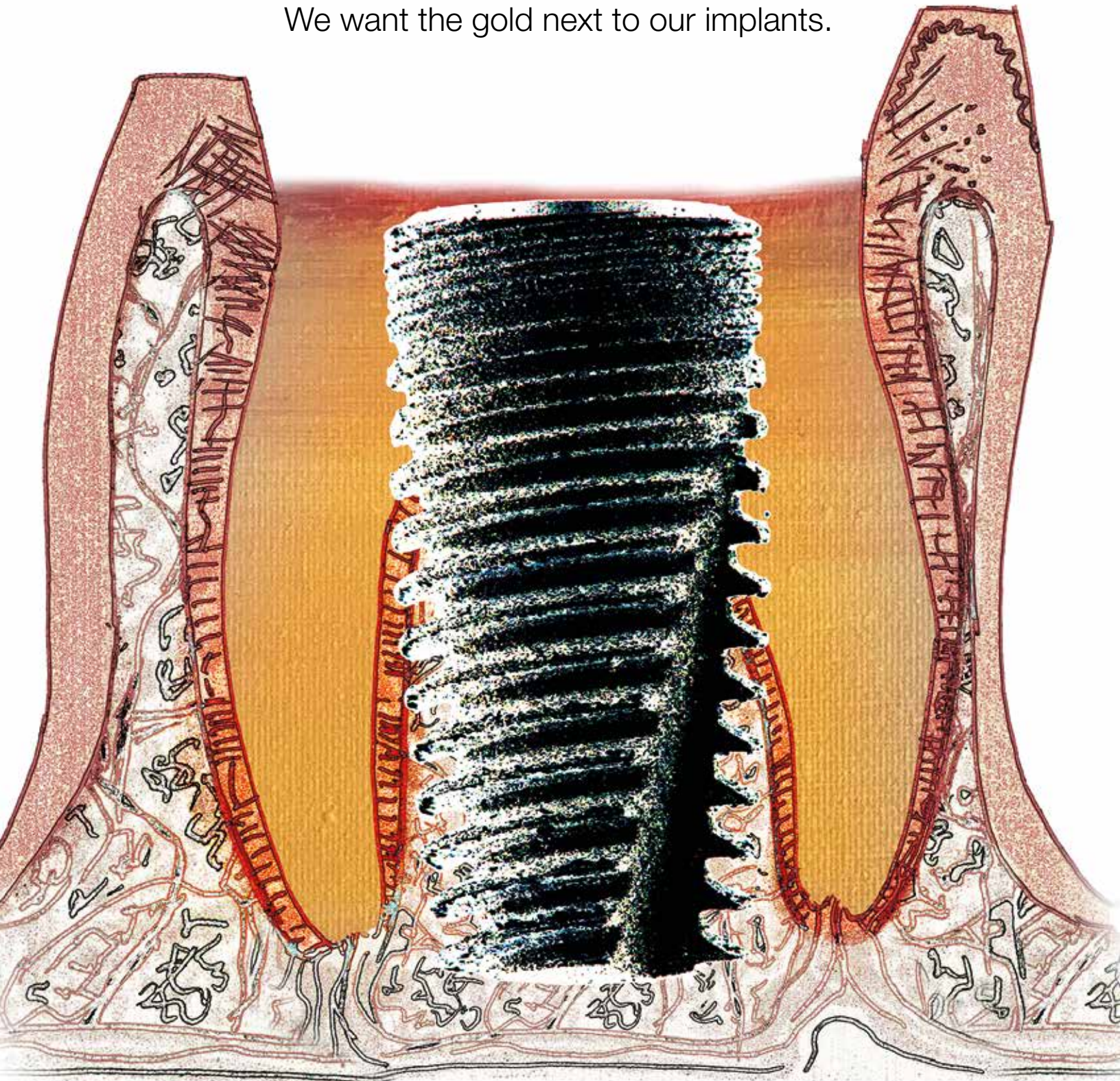


Figure 1-16

What does this actually look like in my practice?
Let's look at an immediate molar implant case.

I routinely mix PRF with allograft to make a sticky bone graft that I place around the implant. I find it efficient to pack the bone when the Ostell peg is in place because it covers the implant and gives me space to work.

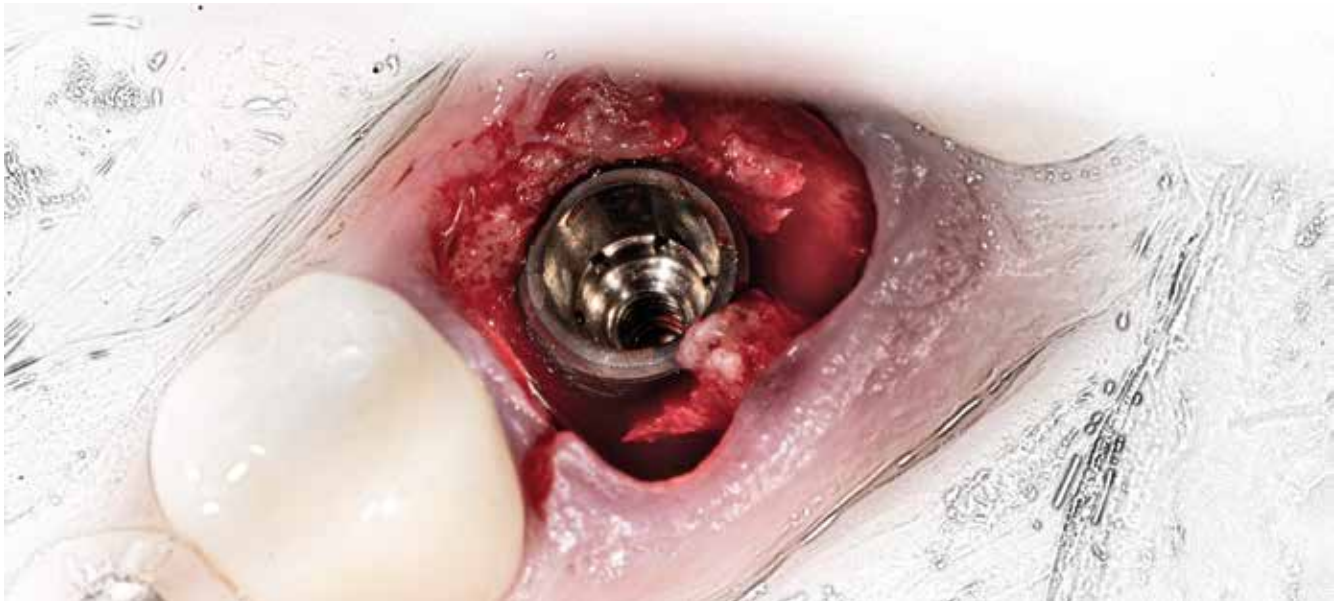


Figure 1-17

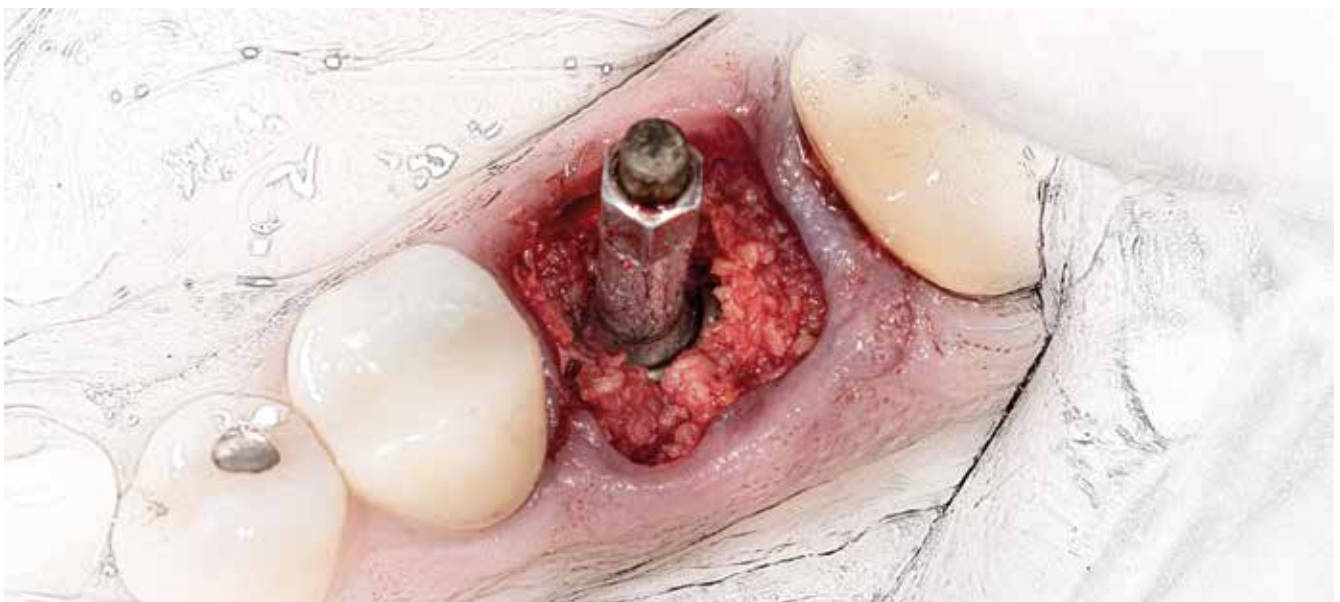


Figure 1-18

Then I place a flared healing abutment.

Before suturing, I tuck a PRF membrane around the abutment. Each of these steps is aimed directly at the healing process. At osseointegration. At making the patient whole.

Implant companies know the key to osseointegration is in the implant's outermost atomic layer. It needs to play well with the bone. It needs to adapt to the process of healing at the cellular and even atomic level. The surface must respect VEGF and promote angiogenesis, among other things. Many unique surface treatments have been studied and applied to implants, all claiming some benefit in short or long-term stability.

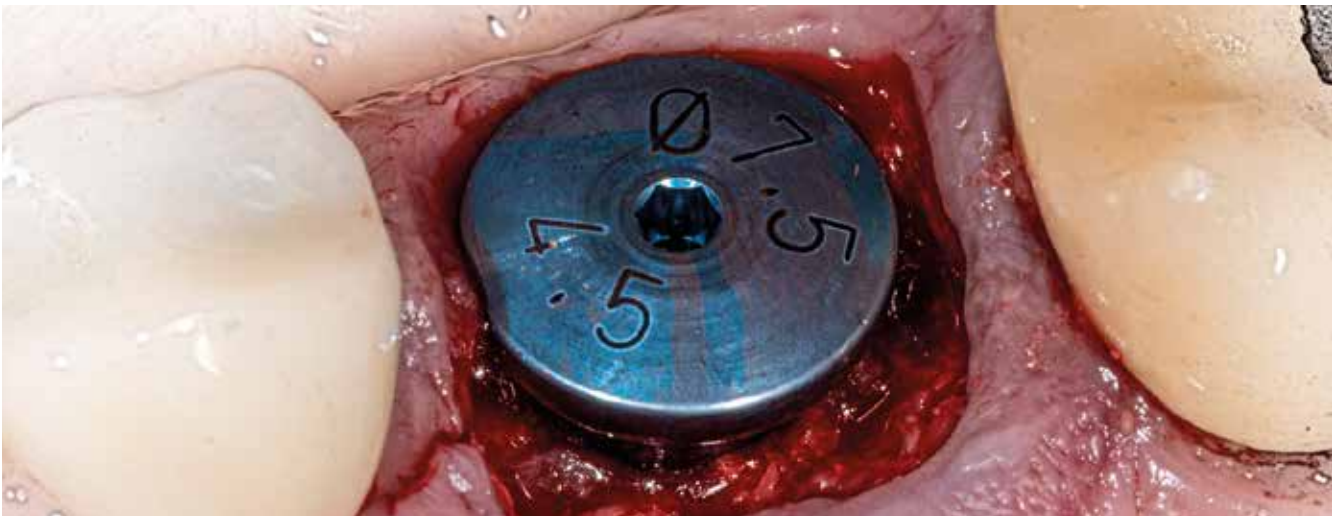


Figure 1-19

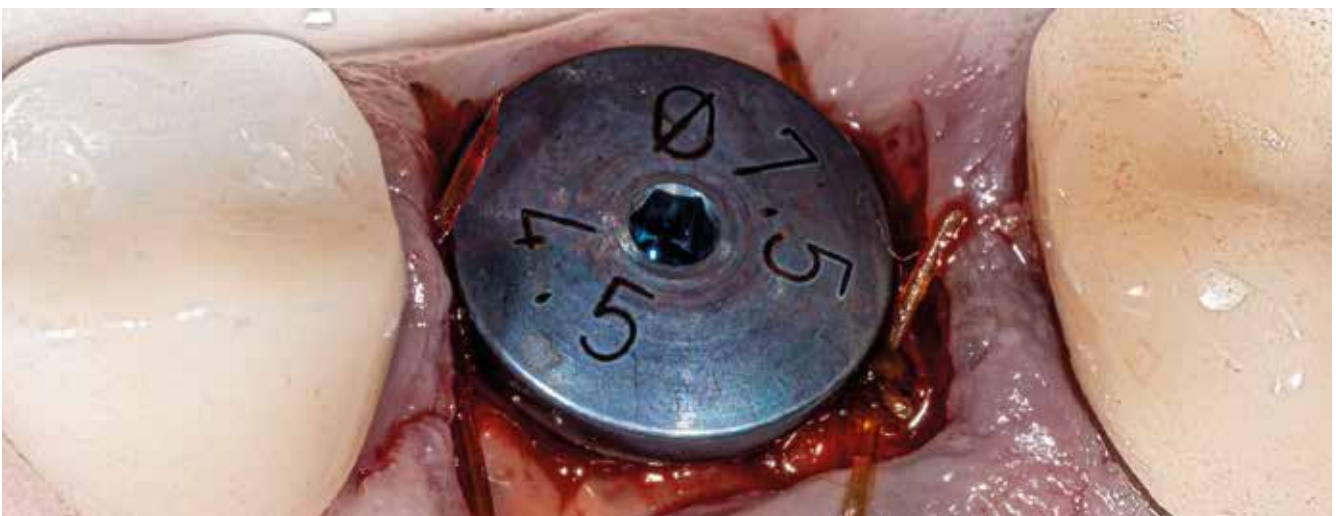


Figure 1-20



We owe many thanks to the pioneer in osseointegration. Per-Ingvar (P-I) Brånemark has left quite a legacy. He looked closely at the world around him, asking questions, looking for understanding. He discovered and developed many of the foundational pieces, the bedrock of implant dentistry. I think he too found beauty in the details, and in putting the pieces together.



Figure 1-21 Portrait of the late Per-Ingvar (P-I) Brånemark, a visionary who explored details and brilliantly wove them together to create the art of implantology. (Artwork inspired by Chuck Close.)

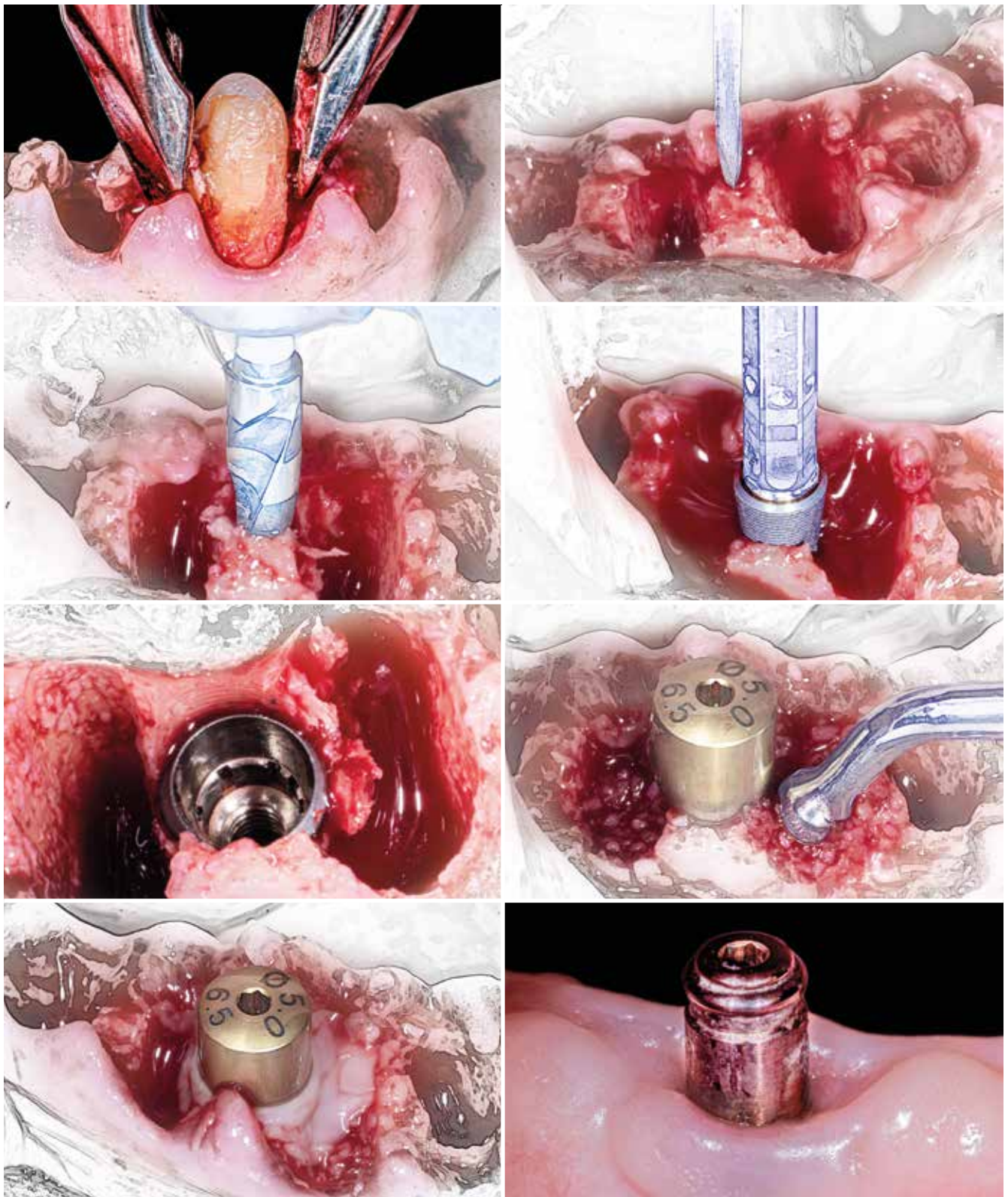


Figure 1-22

And these are just a few small components of the larger healing masterpiece.

When you are figuring out when, where and how to place an implant, it is not just about the screw, the angle or position. It's really not about you... It is very much about the age-old healing process at the most microscopic level of the patient's mouth.

When you think about the art of oral surgery, remember healing is the most fragile and important element. It is the essence of what we do. It's not the screw. It's not the integrated treatment plan. It is in the healing. Break it down even more. Pay keen attention to the details and leverage them and you are certain to have greater success. I want you, and really your patients, to succeed.

Minimize Trauma, Maximize Perfusion

The body is physiologically programmed to succeed at sustaining life. Think about all the different ways it protects and restores itself. The more we understand about this, the better we can serve our patients and achieve the surgical outcomes we all desire.

The brain is the first line of defense, long before the immune system. Be smart.

"If you're gonna be stupid, you gotta be tough."

Grandpa Bill

Let's think for a moment about what it takes for soft tissue to heal. When tissue is incised, cells are

disrupted and destroyed. The blood supply replenishing life in the cells is compromised. It is almost like a microscopic battle scene. The recovery process afterward is dependent on three things: the extent of the damage, the return of the blood supply, and the stamina of the surviving cells.

At its most basic level, all vital tissue requires perfusion. Severe lack of perfusion, more precisely lack of oxygen, is not compatible with tissue repair. This is impacted by macroscopic mechanical injury as well as the post injury inflammatory response.

Maintaining oxygenation is a primary mechanism to optimize tissue healing. We can see how our surgery will affect recovery by observing tissue integrity and color. When blunt injury causes a change in color to blue or white, there is a major disruption in blood supply. (This is different from blanching the tissue under pressure, in which the color immediately returns as the intact vessels refill with blood.) A lack of perfusion pushes the tissue to a hypoxic state, increasing and amplifying tissue necrosis¹.

The power of healing flows through our blood. Through it, the basic elements of healing are transported throughout our body. Some of the resources and signaling pathways are present locally where cells are damaged or being repaired, but the majority of healing is dependent on blood supply. Reperfusion of the injured tissue is therefore a critical step in healing. Where there is ischemia, the capillary network needs to be reestablished. At the direction of angiogenic cytokines, like VEGF, access holes are bored into the existing blood vessels. The sprouting capillaries migrate into the ischemic tissue and organize a new capillary network, restoring oxygenation, and replenishing nutrients to the tissue.

Thankfully, the tissue in most oral surgery procedures already has adequate blood supply before surgery, with the exception of some less common pathologies like medication or radiation induced osteonecrosis. If we protect the tissue, we optimize healing potential.

Surgery is a continuum of reversible and irreversible cellular injury. Remember, the moment a scalpel touches the tissue, there is injury. Set the injury up for healing.

The Continuum of Healing

The immensely complex process of wound healing is frequently divided into three or four overlapping and successive phases: hemostasis, inflammation, proliferation, maturation. I encourage you to think about how each of these steps is impacted during surgery. This awareness can help guide and improve your surgical technique and, ultimately, the patient's outcome.

Optimize hemostasis

With the commencement of the clotting cascade at the first injury to blood vessels, an elaborate series of events takes place.

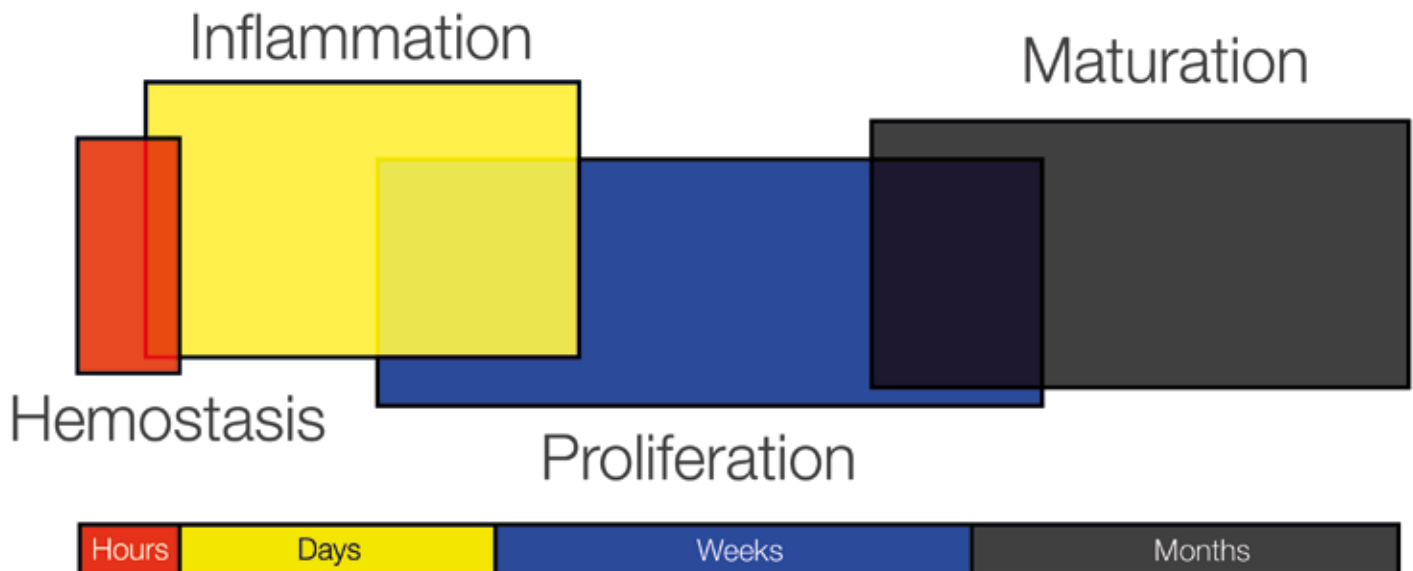


Figure 1-23 Successive phases of healing overlap and interact to keep the process moving.

Think about how to help the body achieve hemostasis from the moment you pick up an instrument.

- Minimize tissue trauma. Sometimes you can see larger blood vessels in the surgical field. If you are able, try to protect them. Minimizing scope of dissection can also reduce bleeding and promote hemostasis. Clean incisions and dissection will optimize the site for hemostasis, especially compared to a site with torn and jagged tissue.
- Patient derived biomaterials, such as platelet rich fibrin, have tremendous potential to promote hemostasis. They provide an incredible fibrin network and supply increased concentrations of cells and factors to the injury site, stacking the deck in the patient's favor.

Reduce inflammation

Less trauma = less inflammation = better healing. Inflammation is a two-edged sword. It is an asset in directing cellular activity and we need it to heal. If we tried to bypass this step completely, we would significantly impair healing. We need to respect inflammation, but we can also reduce its liability.

- Minimize tissue trauma.
- Inflammation is part of the body's response to protect and heal itself in the face of an invasion or injury. Minimize the injury and potential for invasion and you minimize the inflammation.
- Oxidative stresses of anoxic tissue. When tissue is starved of oxygen, even temporarily, this stresses the tissue and increases inflammation. Protect perfusion during and after surgery.
- Consider the patient as a whole. There may be a role in considering dietary and environmental inflammatory exposures. A systemically inflamed patient will likely have more localized inflammation.

Leverage proliferation

There is a direct relationship between tissue trauma and the need for proliferation. More trauma requires more proliferation.

- Minimize the need for proliferation.
 - Macerated tissue and traumatized bone require much more proliferation from the adjacent cells to restore the site to its previous condition. Careful techniques in soft tissue and hard tissue management will decrease the resources needed to heal.
- Maximize opportunities for proliferation.
 - The osteoprogenitor cells in the periodontal ligament have tremendous potential to promote proliferation of bone. Any time we are able to preserve the periodontal ligament in the socket during an extraction, we maximize the tissue's assets.
 - Maintain scaffold and structure for bone formation in a socket. A two walled defect is set up for a different recovery than a five walled defect. When the buccal plate, in particular, is protected and maintained, the opportunity for proliferation in the site is optimized.

Orchestrate maturation

Influence the maturing tissue's properties. The final wound healing phase is maturation. This is the real goal of what we do. If we keep this in mind each step of the way, we will impact the outcome much more profoundly. What type of mature tissue do we want? What will best serve the patient?

- Quantity of tissue: Consider what adequate volume of soft or hard tissue looks like. A simple example is a single tooth extraction. If the restorative plan consists of a three-unit fixed bridge restoration, it may not seem very important to preserve

the hard and soft tissue. We know resorption will occur anyway. But the contours of the remaining tissue will influence food impaction around the bridge and the amount of bone support around the abutment teeth. If the plan is implant replacement, it is easy to see the importance of adequate ridge width and height. And not just adequate bone for a titanium screw, but sufficient tissue contours to optimize the emergence profile and implant loading. But if the plan is just to leave the space empty, then does it matter much how the tissue matures? Surely a broader, more stable ridge will serve the patient better even if there is no replacement. It is indeed an asset, especially when compared to a narrow, short ridge.

- Quality of tissue. Not only is volume of mature tissue important, but the quality and characteristics are also a key part of the equation. This is tremendously important for both soft and hard tissue. If given the choice to place an implant into type four soft bone or type one dense bone, the dense bone will win every time. We want the most stable outcome. Stable keratinized gingiva versus mobile non-keratinized mucosa? Pick gingiva every time. When considering the art of healing, we need to think about short- and long-term aspects. Maximize the long-term assets and think about setting up the best tissue quality.
- When closing an extraction site, if we coronally advance a flap and obtain primary closure, the best-case scenario for our keratinized gingiva is the final position of the advanced flap. This immediately reduces the potential amount by the width of the socket. Compare that to a repositioned flap with an entire socket site that can heal over with keratinized gingiva. This simple change increases the amount of keratinized gingiva by at least five or more millimeters.
- Consider the esthetics of tissue architecture. Especially in the esthetic zone, the volume of tissue is only the beginning. If there is thick, boggy, non-keratinized gingiva showing at high smile, there's significant room for improvement. If the same volume of tissue is pink and symmetric, it may be wildly successful. Managing not only the quantity but also the potential quality of the tissue is a high calling in the art of oral surgery.

Stress and Healing

We must consider the patient as a whole when we discuss healing. The patients who want to heal and believe they are healing will most certainly heal better than those who are anxious and stressed about it. Stress directly impacts healing. The cortisol release related to stress has a profound impact on healing. While this hasn't been studied at great length yet that I know of, I believe this may be a critical, and often overlooked, aspect of healing.

There was an interesting study that hit home for me. During dental school finals, the study subjects showed a 30% slower recovery from a palatal wound than during their summer break². Dental school finals are no joke, as we all know. But compare that level of stress with the stress some patients are carrying: ill family members, domestic instability, job loss. There are many things in life more stressful than dental school finals. An awareness of how stress impacts healing doesn't decrease the stress, but it could impact and guide how you discuss things with patients or how quickly you proceed to the next stages of their planned treatment.

The Continuum of Healing Simplified

There is a distinct start to healing. It begins the moment of injury. At some point, the tissue is done healing. The transition between phases of healing is not so clearly demarcated. It flows from one to the next. To keep it simple during surgery, I like to think

of the immediate, early and late phases of healing. How can I have a positive impact in each of these phases? Similarly, I want to pay attention to what might have a negative impact on immediate, early and late healing.

Healing is truly incredible. It is complex and difficult to grasp. That's why I like to think about it in those three phases.

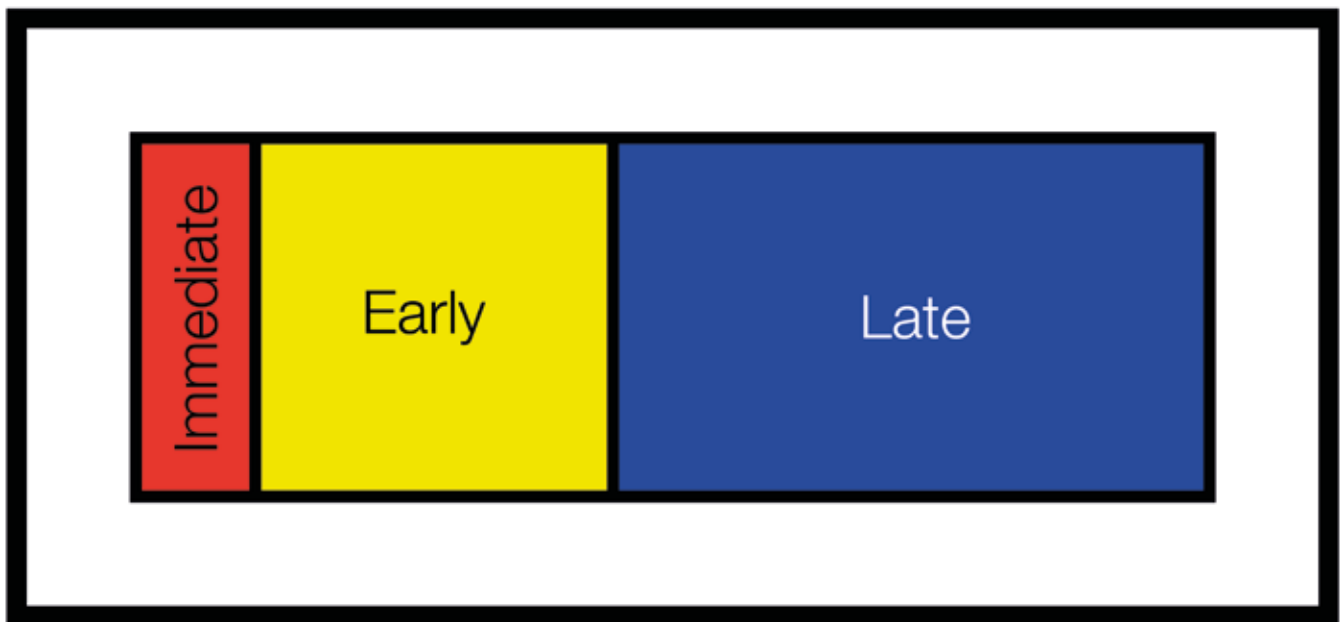


Figure 1-24 During surgery, I like to simplify my thoughts to the immediate, early and late healing phases. What can I do to help cells heal in these three phases?

References

1. Arias JI, Aller MA, Arias J. Surgical inflammation: a pathophysiological rainbow. *J Transl Med.* 2009 Mar 23;7:19.
2. Marucha PT, Kiecolt-Glaser JK, Favagehi M. Mucosal wound healing is impaired by examination stress. *Psychosom Med.* 1998;60(3):362-365.